
بسم الله الرحمن الرحيم

Acute Kidney Injury

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Epidemiology

INCIDENCE

- 1-5% of all patients
- 10-23 % in the ICU

Epidemiology

MORTALITY

- 20-70% Overall
- 79% for patients requiring RRT (ICU)

AKI- definition

- **An abrupt fall in GFR over a period of minutes to days with rapid rise in nitrogenous waste products in blood.**

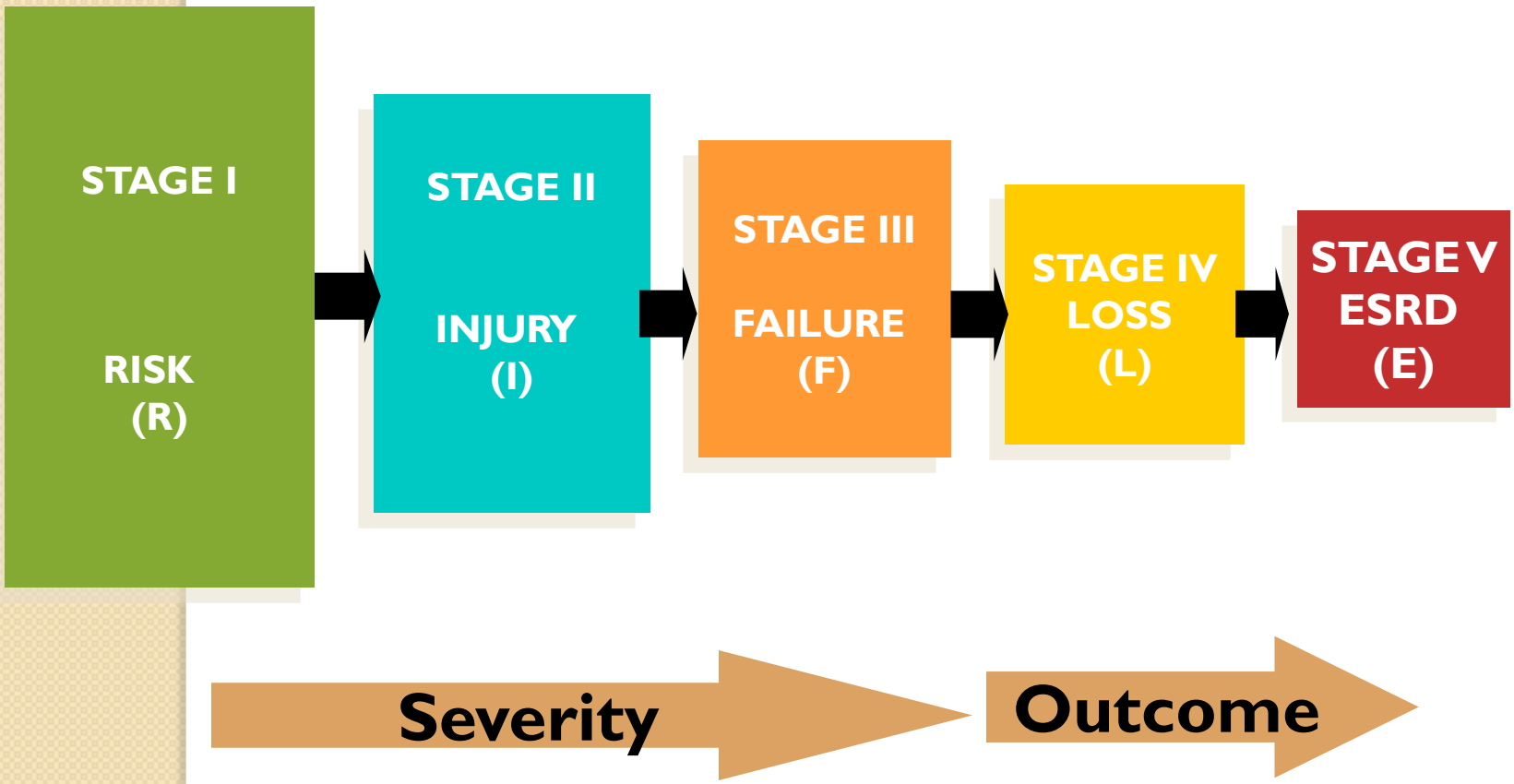
(Rate of production of metabolic waste exceeds the rate of renal excretion)

Definition

AKI is defined as any of the following:

- **Increase in S Creatinine by ≥0.3 mg/dl (≥26.5 µmol/l) within 48 hours;**
  or
- **Increase in S Creatinine to ≥1.5 times baseline, which is known or presumed to have occurred within the prior 7 days;**
  or
- **Urine volume <0.5 ml/kg/h for 6 hours.**

Acute Kidney Injury Network (AKIN- 2005)



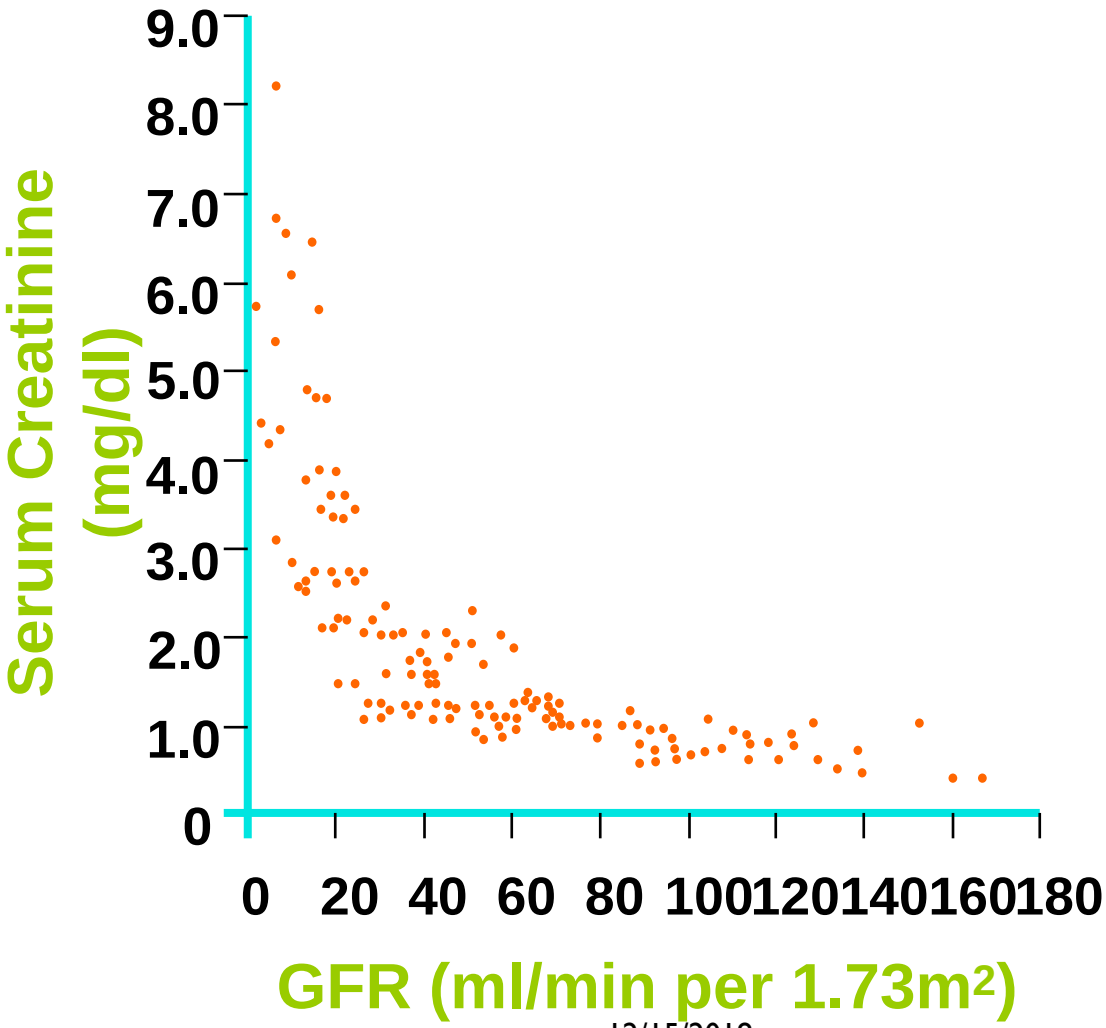
AKIN Staging

AKIN stage	Serum Creatinine Criteria	Urinary Output Criteria	Time
1	↑ Cr ≥ 0.3 mg/dl or ≥26.5 µmol/l or 1.5-1.9 times baseline	< 0.5 ml/kg/hr	> 6 -12 hrs
2	↑ Cr 2-2.9 times baseline	< 0.5 ml/kg/hr	≥ 12 hrs
3	↑ Cr ≥ 3 from baseline or Cr ≥ 4mg/dl (≥353.6 µmol/l) or initiation of RRT	< 0.3 ml/kg/hr or anuria	≥ 24 hrs ≥ 12 hrs

Acute Kidney Injury

Stage	Increase in serum Creatinine
1	≥1.5 x previous result
2	≥2 x previous result
3	≥3 x previous result, RRT Anuria ≥ 12 hours

Relationship between GFR and serum creatinine in AKI



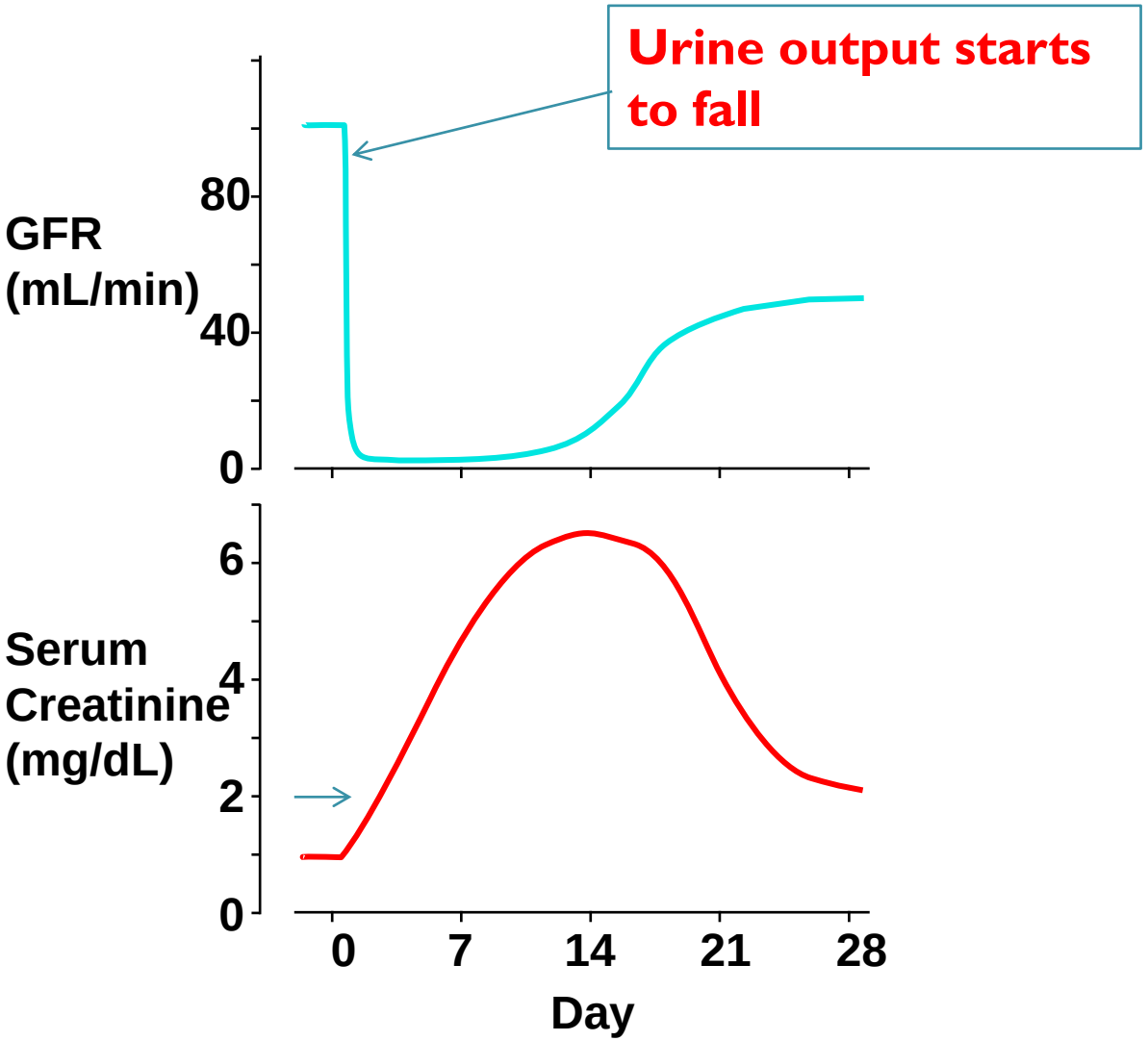


Figure: The abrupt drop in GFR but the S.Cr. does not start going up for 24 or 36 hours after the acute insult .

One glass urine in 12 hours

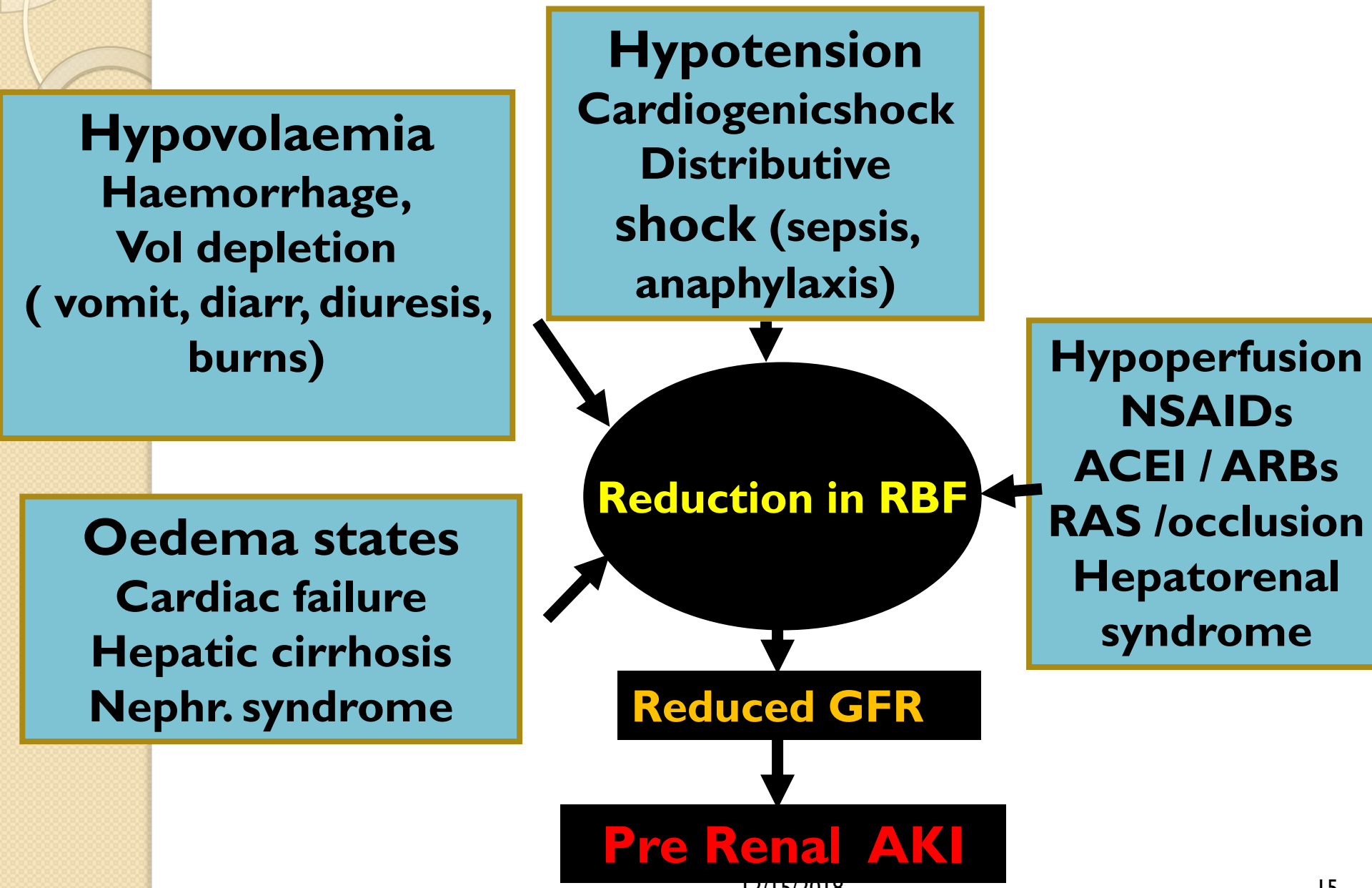
Risk factors of AKI

- eGFR <60 ml/min/1.73m2 or history of AKI
- Diabetes
- Heart failure, liver disease,
- Neurological or cognitive impairment
- Use of nephrotoxic drugs
- Use of iodinated contrast agents within the past week
- Symptoms or history of urological obstruction
- Sepsis
- Age 65years or over

To function properly kidneys require:

- Normal renal blood flow – **Prerenal.**
- Functioning glomeruli,tubules and interstitium – **Intrinsic/Renal.**
- Clear urinary outflow tract – **Postrenal.**

PRE-RENAL (Hemodynamic) AKI



Renal / Intrinsic AKI

Glomerular	Tubular	Interstitial	Vascular
AGN PSGN, SLE, ANCA associated, anti-GBM disease HSP, Cryoglobulinemia, TTP, HUS	ATN *Ischemia-50%* *Toxins -30%*	AIN Drug: NSAIDs, antibiotics Infiltrative : lymphoma Granulomatous-Sarcoidosis, TB Infection : APN	Vascular occlusions - Renal artery occlusion - Renal vein thrombosis - Cholesterol emboli
5- 15%	70-80%	8 -20%	< 2%

Post-renal Urinary outflow tract obstruction

Intrinsic

Intra-luminal
- Stone,
- Blood clots,
- Papillary necrosis

Intra-mural
- Urethral stricture,
- BPH,
- Ca prostate,
- Bladder tumour,
- Radiation fibrosis

Extrinsic

- Pelvic malignancies
- Prolapsed uterus
- Retroperitoneal fibrosis

Acute Kidney Injury

Prerenal
Uosm > 500 mosm/kg
Una < 20 meq/L
FEna < 1%
Microscopy – bland
↑ BUN / S.Cr. Ratio
USG- Normal

Intrinsic/ Renal

Post Renal
Uosm: variable
UNa: low early, high late
FEna: variable
Microscopy – bland
USG - Diagnostic

Ischemic / Toxic ATN
Uosm ~ 300 mosm/kg
UNa > 40meq/L
FEna > 2%
Microscopy – dark pigment cast

Acute Interstitial Nephritis
Uosm: variable, ~300 mosm/kg
UNa > 40 meq/L
FEna > 2%,
↑ Eosinophils
Microscopy – WBC, RBC, leukocyte casts

Acute GN
Uosm: variable
UNa: variable
FEna: variable,
ME – hematuria-dysmorphic, RBC casts, proteinuria

Pre-renal AKI

History

- Any obvious causes of hypotension, hypovolaemia or hypo perfusion.
  a) Haemorrhage/haematoma,
  b) GI loss – diarrhoea, vomiting,
     renal loss,
     skin loss (burns/exfoliation),
  c) Third spacing (pancreatitis).
  d) Evidence of cardiac failure
- Sepsis (and if so what is the source?)

Examination

- **Low BP, rapid pulse.**
- **Cool peripheries – vascular shut down**
- **Capillary refill time – greater than 2 seconds implies volume depletion or poor cardiac function**
- **Lying and standing blood pressure – significant drop implies hypovolaemia**
- **Warm to touch - sepsis?**
- **Peripheral pulses - are they bounding**

Examination

- **Reduced skin turgor, dry lips, mouth and mucous membranes - systemic hypovolaemia**
- **Face – sunken eyes imply dehydration,**
- **JVP: may be low if volume depleted**

Prerenal

- Uosm > 500 mosm/kg
- Una < 20 meq/L
- FEna < 1%
- Microscopy – bland
- ⬆ BUN / S.Cr. Ratio
- USG- Normal

Post-renal AKI

- History
  - Lower urinary tract symptoms (LUTS) – frequency, urgency, dysuria, nocturia, poor stream, hesitancy, terminal dribbling, strangury.
  - Prostatism.
  - Haematuria (visible and non-visible)
  - Loin pain

Examination

- **Look for:**
  - **palpable abdominal masses,**
  - **palpable bladder,**
  - **visible haematuria,**
  - **rectal examination for prostate in males**

Post Renal

- Uosm: variable
- UNa: low early, high late
- FEna: variable
- Microscopy – bland/ haematuria
- Imaging studies - Diagnostic

Renal

History

- Hypovolaemia, hypotension, hypo perfusion, sepsis or toxin/drugs
- Oliguria, haematuria, puffy face, oedema.
- Fever, arthritis, rash etc
- Headache, nausea, vomiting
- SOB
- Altered consciousness
- Presence or history of a primary disease/event.

Renal

Examination

- **Signs of fluid overload- oedema/anasarca**
- **JVP: raised if heart failure or AKI causing significant volume overload**
- **Heart: Listen for an S3**
- **Lungs: signs pulmonary oedema.**
- **Signs of pneumonia / source of sepsis**
- **Abdomen: Organomegaly, ascites, evidence of sepsis**
- **Urine output – catheterize if doubt**

Examination

Evaluation for

- **rashes,**
- **arthritis,**
- **oral ulceration,**
- **skin changes,**
- **uveitis,**
- **epistaxis,**
- **new neurology sign including hearing loss,**
- **stigmata of endocarditis**

Renal

ATN

- Uosm ~ 300 mosm/kg
- UNa > 40meq/L
- FEna > 2%
- Microscopy – Muddy brown granular cast

Renal

Acute Interstitial Nephritis

- Uosm: variable, ~300 mosm/kg
- UNa > 40 meq/L
- FEna > 2%,
- ⇧ Esinophils
- Microscopy – WBC, Eosinophil, RBC, leukocyte casts

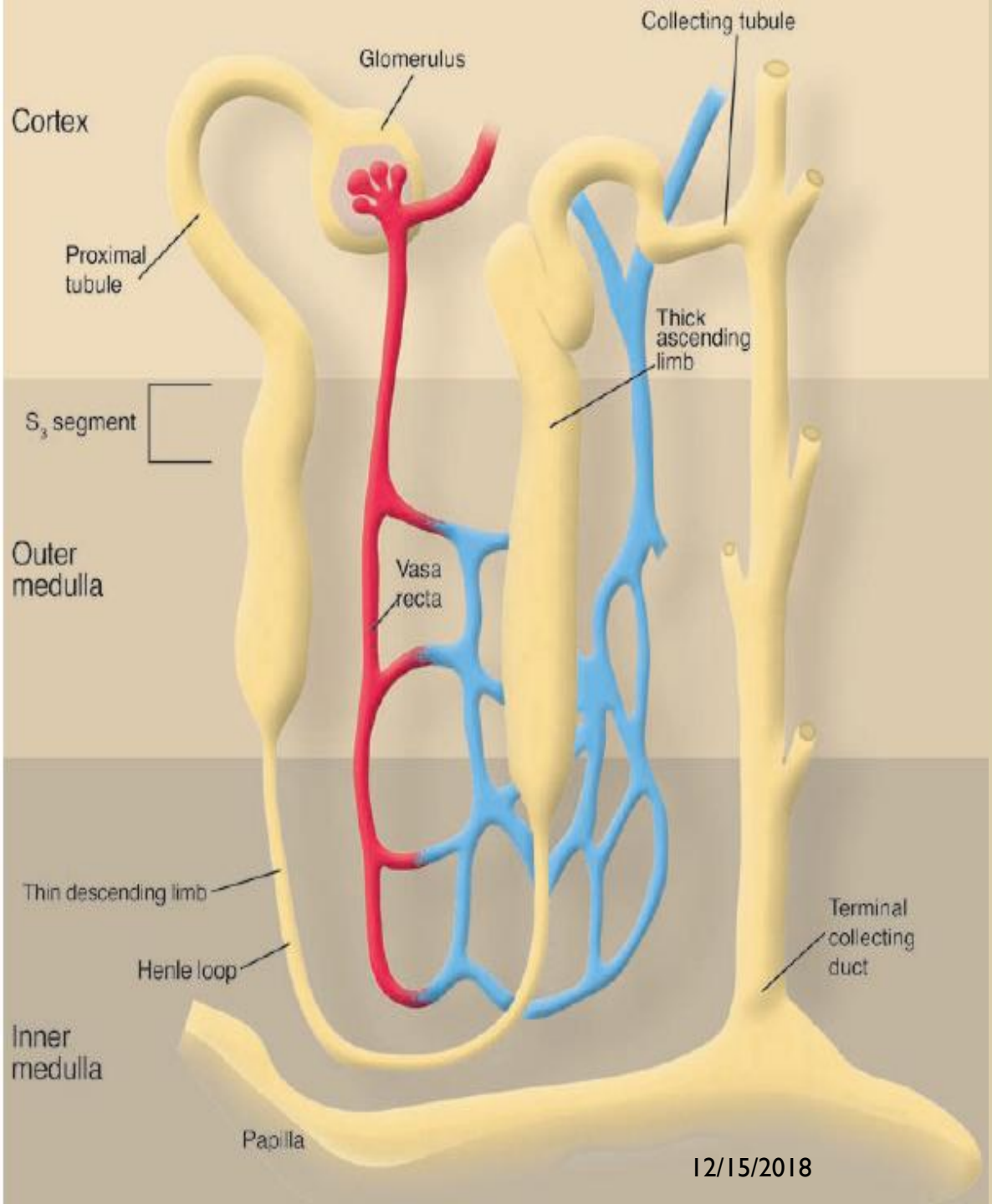
Renal

Acute GN

- Oliguria, puffy
- Uosm: variable
- UNa: variable
- FEna: variable,
- ME – hematuria- dysmorphic, RBC casts, proteinuria

Renal biopsy

- **In specific cases.**
- **Biopsy will guide the management**



PaO2

50 mm of Hg

↓

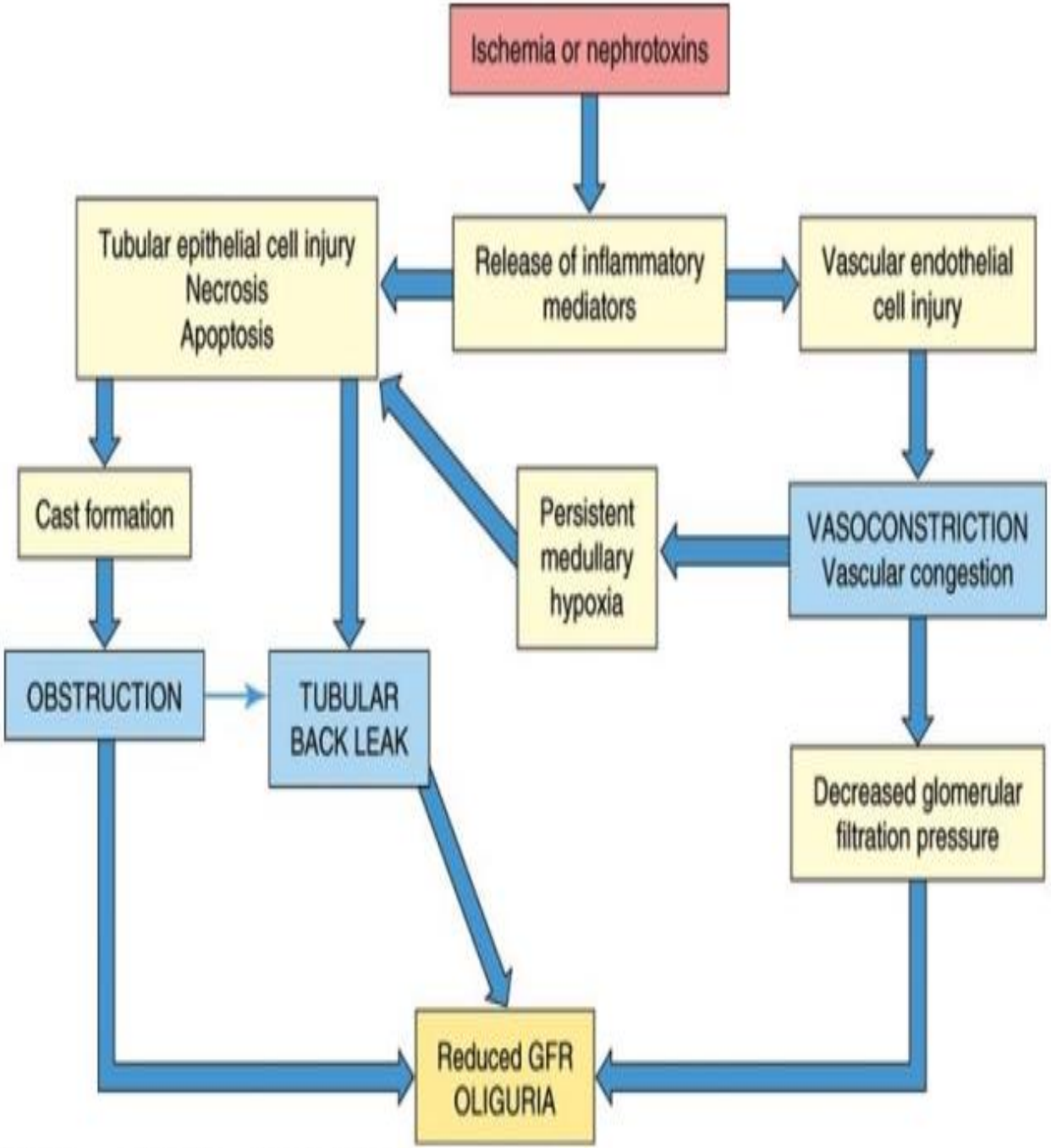
PaO2

20 mm of Hg

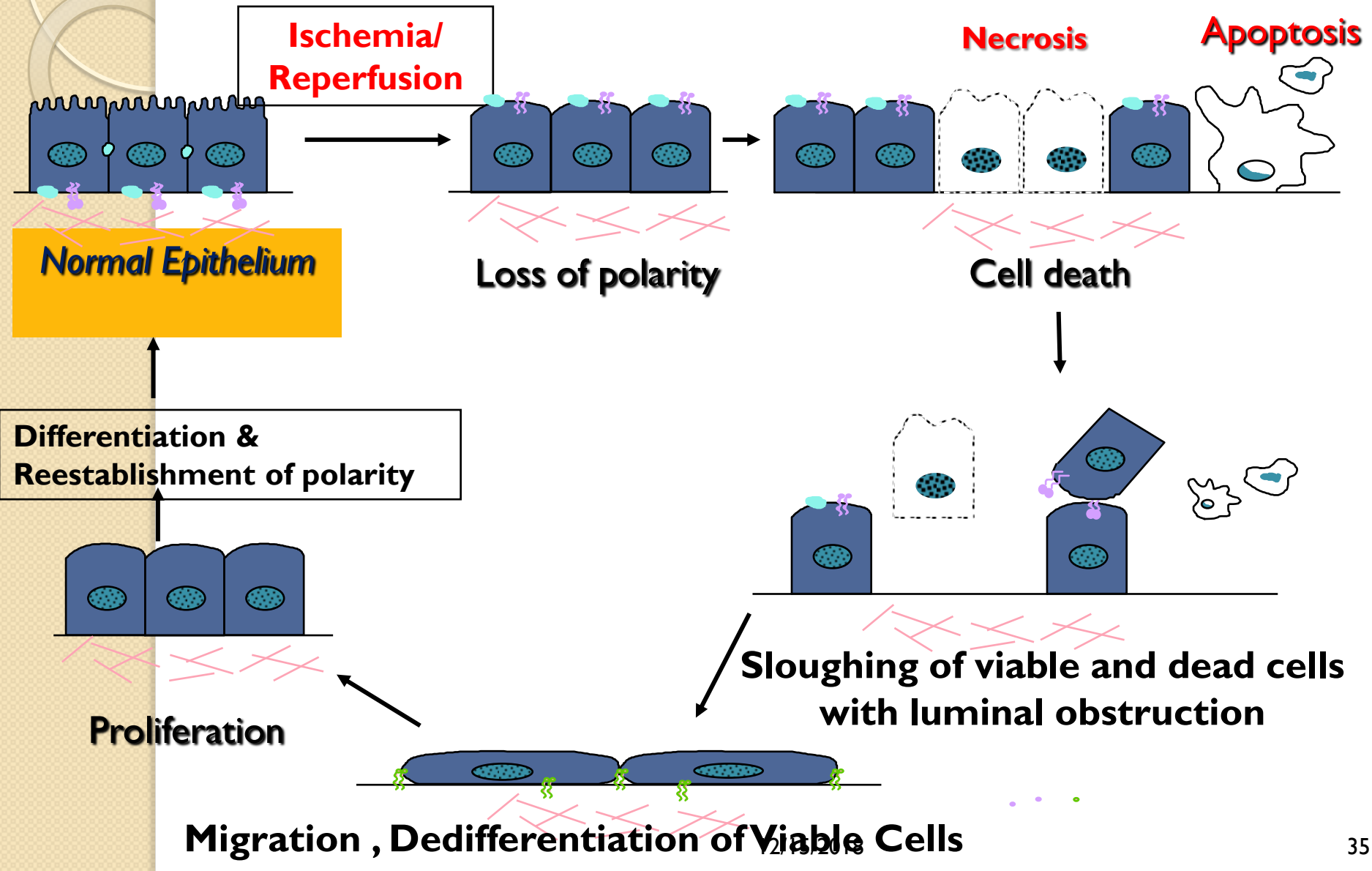
↓

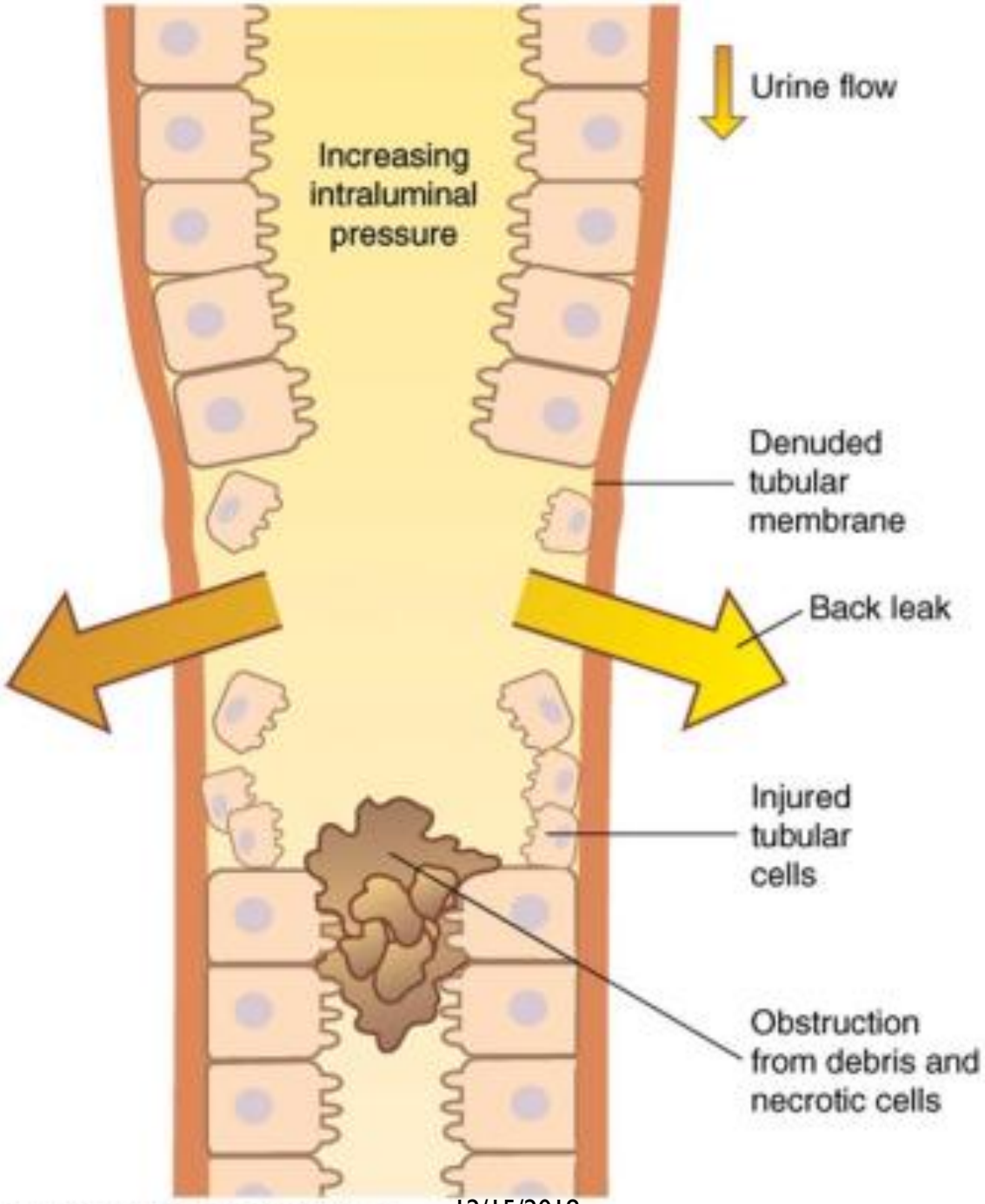
PaO2

10 mm of Hg



Pathophysiology of ATN:
Tubular Epithelial Cell Injury and Repair





What to do with a Raised Creatinine ?

Acute or Chronic ?

- Distinguishing between AKI and chronic renal impairment is important, as –
  - The approach to these patients differs greatly.
  - This may, save a great deal of unnecessary investigation.

Factors that suggest chronicity include –

- History of:
  - HTN,
  - DM,
  - Arthritis and
  - NSAID,
  - Stone disease and obstruction,
  - Congenital diseases .
-

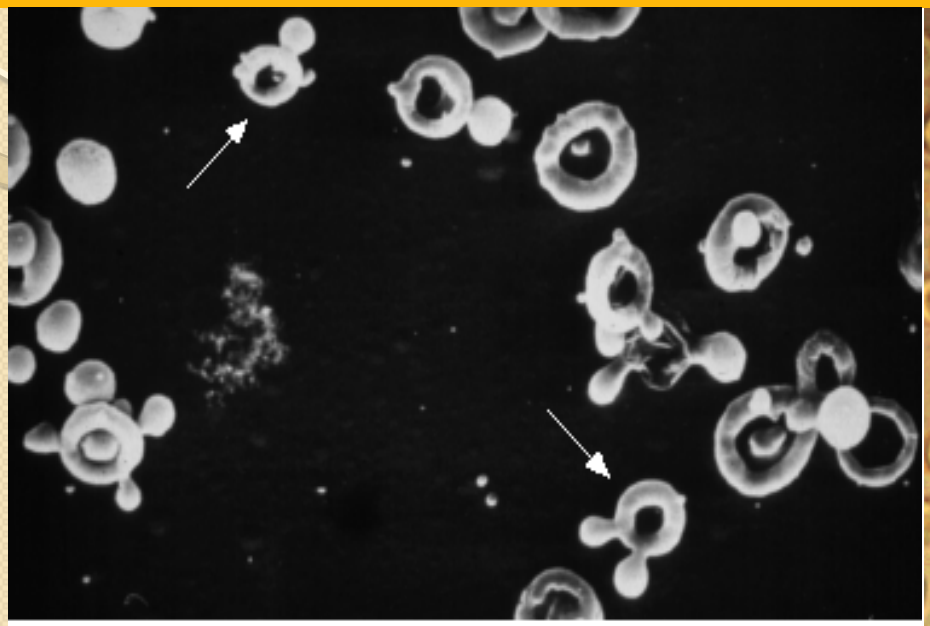
Factors that Suggest Chronicity include –

- Absence of acute illness,
- Long duration of symptoms,
- Nocturia,
- Leuconychia
- Pigmentation
- Anaemia,
- Hypocalcaemia.
- Previous Serum creatinine
- Kidney size.

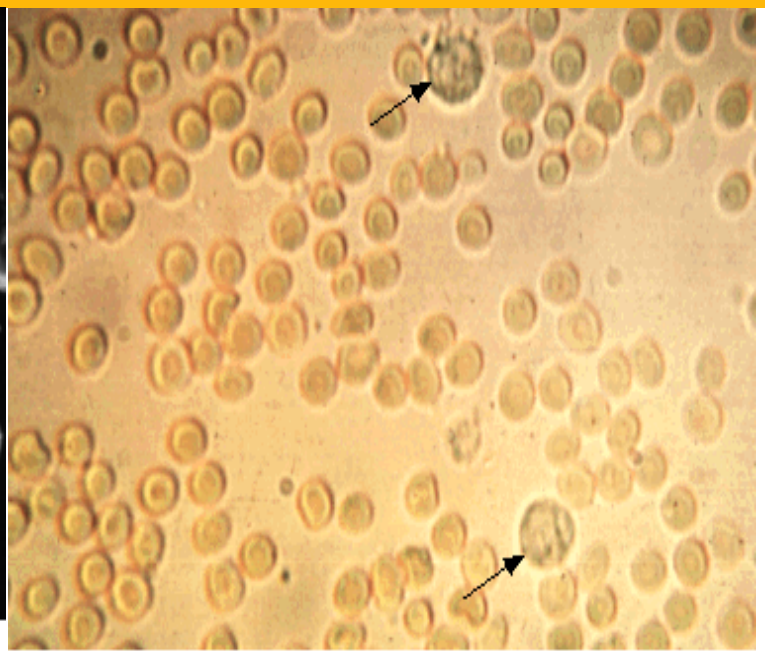
What investigations are most useful in AKI ?

- Urinalysis:
  - Blood,
  - Protein,
  - Cells
  - Casts
  - UNa, FeNa

RBCs



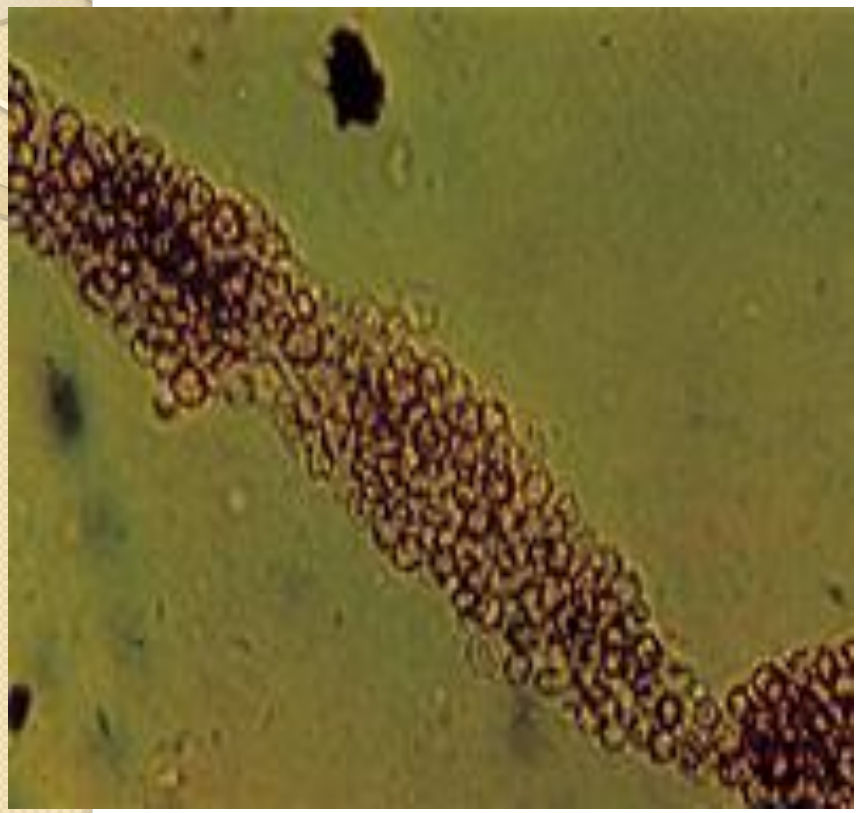
Dysmorphic red cells Scanning microscopy showing dysmorphic red cells in a patient with glomerular bleeding. Acanthocytes can be recognized as ring forms with vesicle-shaped protrusions (arrows). Courtesy of Hans Köhler, MD.

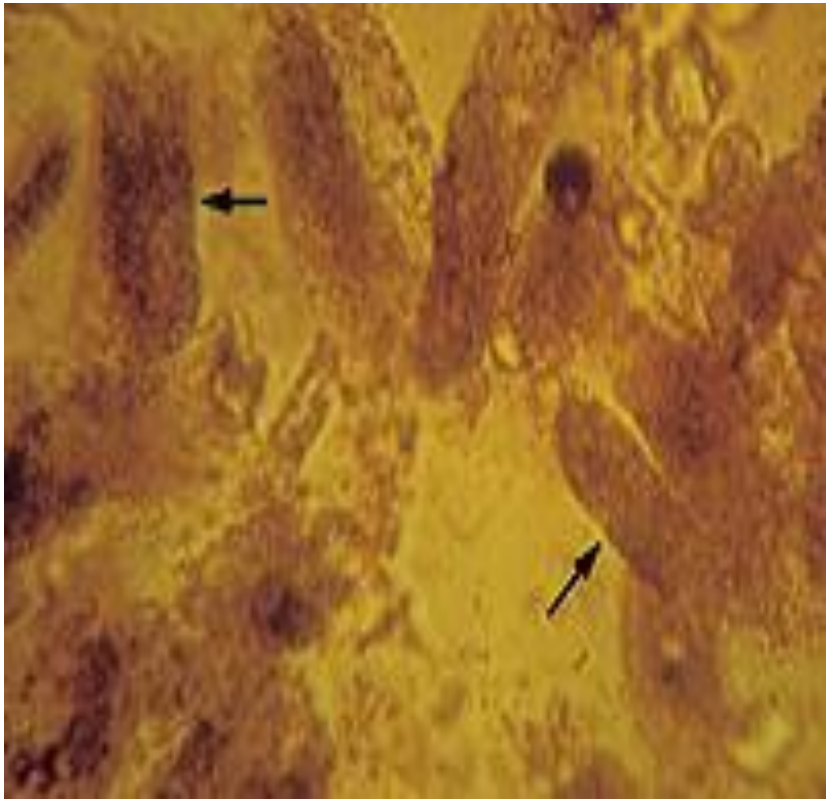


Monomorphic red cells Urine sediment showing many red cells and an occasional larger white cell with a granular cytoplasm (arrows). The red cells have a uniform size and shape, suggesting that they are of nonglomerular origin. Courtesy of Harvard Medical School.

- **Dysmorphic red blood cells suggest glomerular injury.**

Marker of glomerular injury

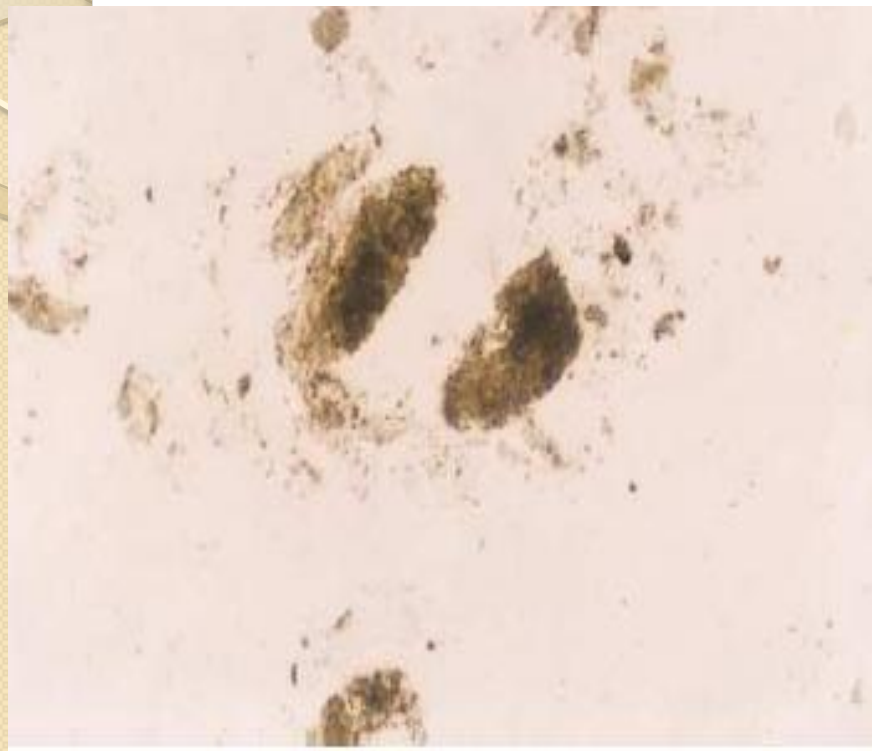


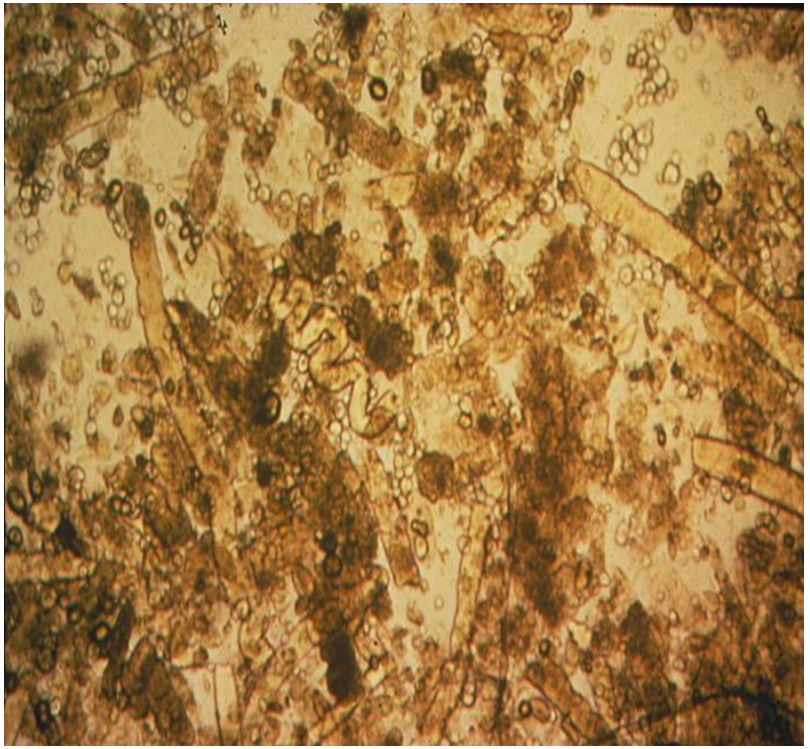


Red blood cell cast

Granular cast

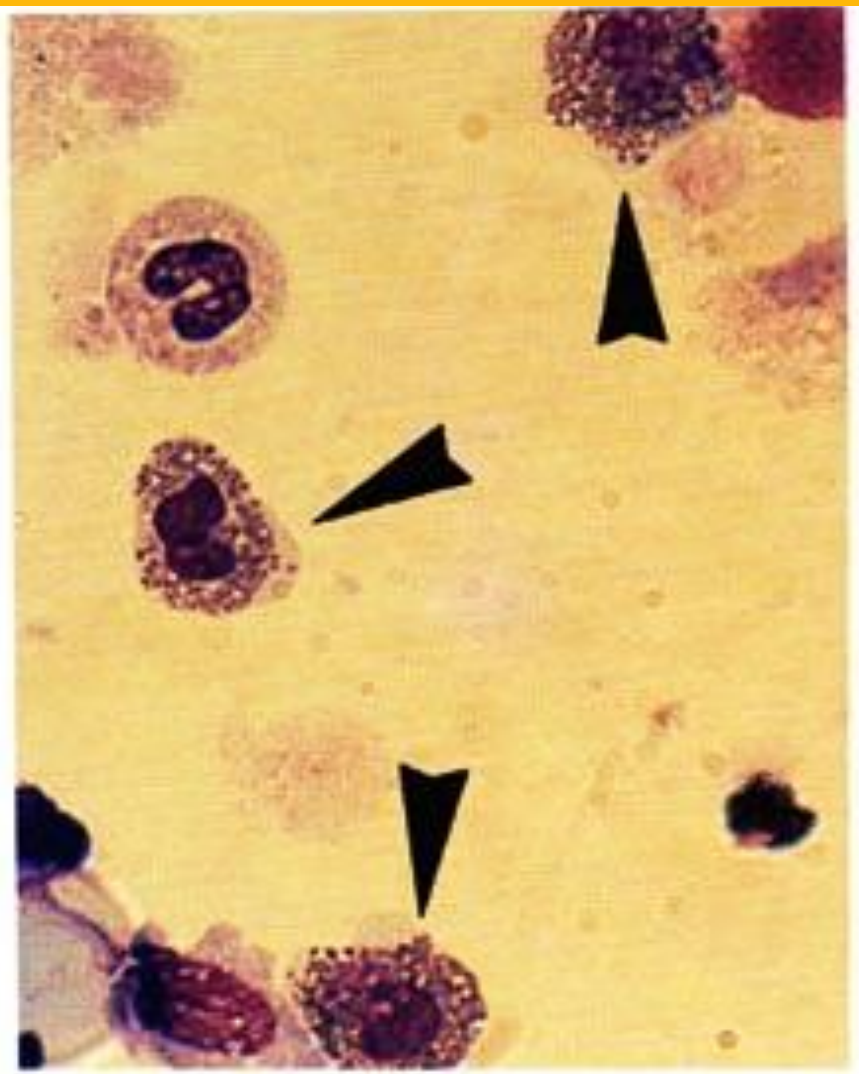
Marker of acute tubular necrosis





Pigmented granular (“muddy brown”) casts

Marker of acute interstitial nephritis.



EOSINOPHILS (BY MGG)

Haematology

- **Full blood count, blood film:**
  - **Neutrophilia in sepsis**
  - **Eosinophilia may be present in acute interstitial nephritis, cholesterol embolization, or vasculitis (CSS)**
  - **Thrombocytopenia and red cell fragments suggest thrombotic microangiopathy –TTP, HUS**

Biochemistry

- Daily
  - urea, creatinine,
  - electrolytes,
  - PH, serum bicarbonate
  - Calcium.

Biochem….

- CPK, myoglobinuria –
  - Rhabdomyolysis
- Serum immunoglobulins, serum protein electrophoresis, Bence Jones proteinuria
  - Myeloma

Haem….

- **Coagulation studies :**
  - **Disseminated intravascular coagulation associated with sepsis**

Immunology

- Antinuclear antibody (ANA) ,Anti-double stranded (ds) antibody .
- C3 & C4 complement concentrations-
  - Low in SLE, acute post infectious glomerulonephritis, Cryoglobulinemia
- ANCA
- Anti GBM antibodies
- ASO and anti-DNAse B titres
  - High after streptococcal infection
- Hepatitis B and C, HIV serology

Imaging

- Renal ultrasonography
  - For renal size, symmetry, evidence of obstruction
- CXR
- X-Ray KUB

Clinical Scenario

A 10 year old girl presented with S Creatinine of 2.0 mg/dL. She has oliguria, haematuria and puffy face. Her BP is 150/100 mmHg.

- AKI or CKD ?
  - *GN*
    1. PSGN
    2. SLE
    3. Vasculitis
- History:
- Investigations:

Clinical Scenario

S Creatinine of a 21 year old farmer is 2.2 mg/dL.

He reported with severe acute watery diarrhoea and vomiting for two days. He has not passed urine since yesterday.

- **AKI or CKD ?**
  - **Pre renal or Renal ?**
    - **History**
    - **Ph Exam**
    - **Investigations**

Clinical Scenario, What to do ?

S creatinine of a 43 old man is 4.9mg/dL.

He was having LBP for last six months along with irregular fever.

His family physician advised Naproxen, which he is taking off and on for last two months.

AKI or CKD ?

- Prerenal or Renal ?
  - History
  - Exam
  - Investigations

Initial 7 Steps of AKI Management Bundle

- Confirm AKI
- Assess emergency: Pulmonary oedema, Hyperkalaemia, Acidosis.
- Undertake ABCDE - full clinical examination
- Stop nephrotoxic drugs
- Urine dipstick test and confirm by RME
- Biochemistry - Check & repeat.
- Renal ultrasound and consider urinary catheter
- Urgent senior review

Management principles…

- Identify the source of infection and treat aggressively keeping dose adjustment.
  - Minimise indwelling lines
  - Remove bladder catheter if anuric.
- Identify and treat bleeding tendency:
  - PPI, H2 antagonist, avoid aspirin
  - transfuse if required

Optimise nutritional support

- **Maintaining adequate nutrition enhances patient survival**
- **Maintain protein intake about 1gm/Kg/Day**
- **Protein intakes of > 1.2 g/kg/ day can dramatically increase azotaemia.**

RRT

- **Initiate dialysis before uraemic complications set in.**
- **Early RRT improves mortality and recovery .**
- **Specific types of therapy are available for critically ill patients.**

Conclusions

- **AKI is increasingly common, particularly among hospital inpatients and critically ill patients.**
- **It carries a high mortality**

Conclusions..

- **Patients at risk are - elderly people; patients with diabetes, hypertension, or vascular disease; and those with pre -existing renal impairment**

Conclusions..

- **AKI is often preventable.**
- **Rapid recognition of incipient AKI and early treatment of established AKI is life saving and prevent irreversible loss of nephrons.**

Questions?


Thanks For
Presence & Patience