

# ACUTE KIDNEY INJURY – AKI

CHARACTERISED by -

- USUALLY acute reversible loss of renal function
- due to rapid decline in GFR within days-weeks.

ACCOMPANIED -

- Oliguria - Non-oliguric- or Anuria
- Retention of nitrogenous waste products.

DISTURBANCES -

- Body fluid
- Electrolytes
- Acid base homeostasis.

# RIFLE criteria

The ACUTE DIALYSIS QUALITY INITIATIVE GROUP

AKI - differentiate from CKD

AKI - on - CKD.

RIFLE classify - AKI

Three levels

R - I - F

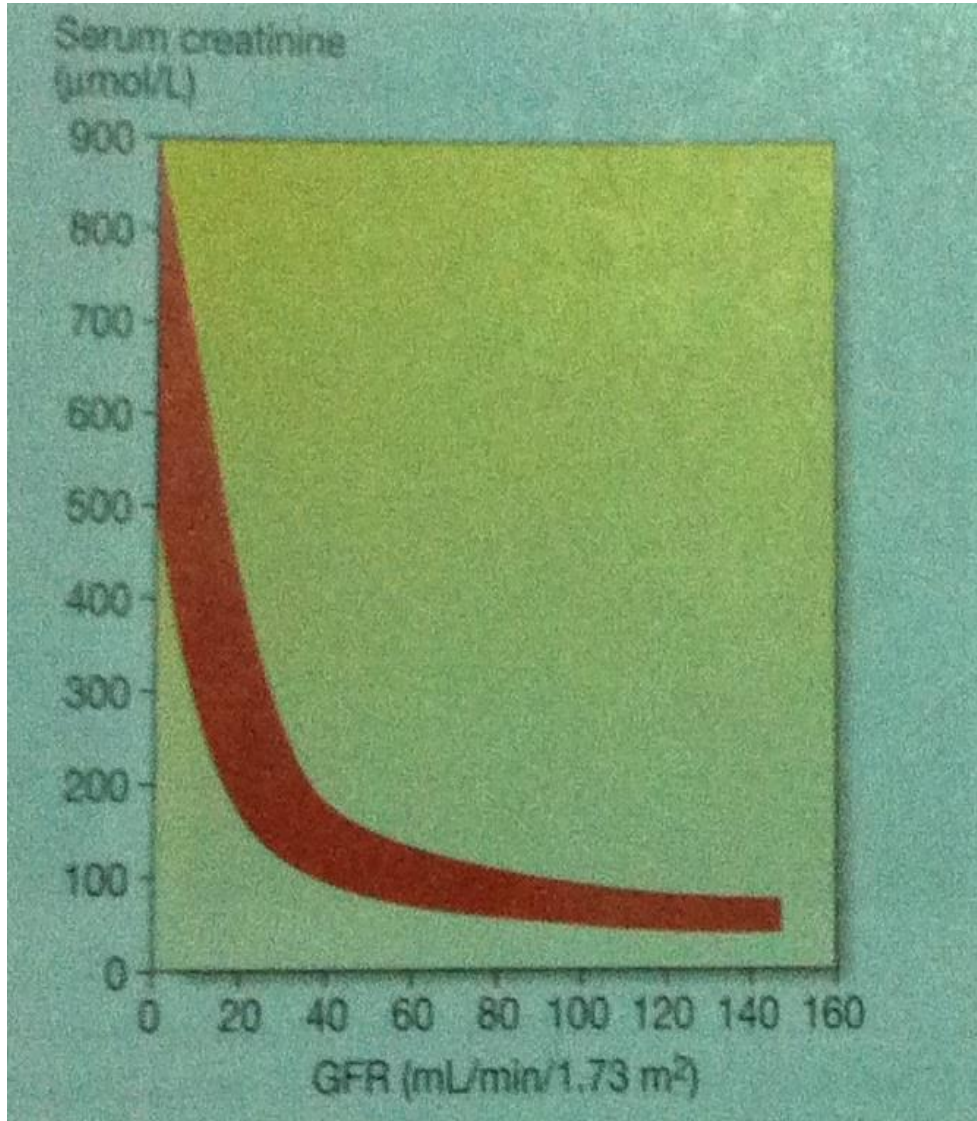
Two outcomes

L - E.

Assess the degree of renal damage and prognosis

## RIFLE classification for ARF-

Grade	GRF criteria	UO criteria
Risk	S.Cr. 1.5 times normal Within 48hr	UO <0.5 mL/kg/ hour within 6h
Injury	S.Cr. 2-3 times	UO <0.5mL/kg/ hour within 12h
Failure	S. Cr. 3 times or S.Cr >350micro mol/L with Acute rise >40micro mol/L	UO <0.3mL/kg/ hour within 24h
Loss	Persistent Aki >4 weeks	
CKD	Persistent renal failure >3 months	



# GFR-CREATININE

- NORMAL-GFR- is 120-130ml/min/1.73 m<sup>2</sup> surface area.  
plasma-ultra-filtered from intra-Glomerular capillary  
into Bowmans capsule .  
CREATININE - ideal marker for GFR  
Endogenous sub. derived from skeletal muscle- CREATIN-  
released at CONSTANT rate.
- It is freely filtered in the Glomeruli-  
Neither metabolised Nor absorbed by renal tubules.  
UREA- NO CONSTANT level  
varies with protein intake- GIT-bleeding –  
liver function , Catabolism- state and Drugs.

# EPIDEMIOLOGY

AKI- has variable clinical presentation.

- 1- COMMUNITY ACQUIRED- AKI.

Presented in two kinds

- A- less sever AKI-
- S. Creatinine rises  $> 50\%$ - of normal level
- 177micomol/L.
- Good prognosis
- Managed
- Medical ward.

# Epidemiology-

B- Sever complicated AKI-

Multi - Organ failure or sepsis .

- S. Cr. > 500 micromol/L .
- Managed in - ICU- MOINTERING.
- Poor prognosis
- Mortality 50-70%.

2- HOSPITAL ACQUIRED - AKI.

Presented in two form

- less sever AKI
- Sever complicated AKI

– RENAL AUTO-REGULATION- MECHANISM-  
PATHOPHYSIOLOGY-OF PRE-RENAL-AKI-

- Normally the kidneys are able to maintain GFR 120-130-ml/min./ 1.73 m<sup>2</sup> surface area.

DAILY alteration and variation of renal perfusion pressure.

- AUTOREGULATION

Kidney releases RENIN from

JUXTA-GLOMERULAR-APPARATUS – MACULA DENSATA

RENIN- Angiotensinogen - Angiotensin-I

ANGIOTENSIN-II- ALDOSTERONE.



## — RENAL AUTO-REGULATION- MECHANISM- PATHOPHYSIOLOGY-OF PRE-RENAL-ARF-

- ANGIOTENSIN II-

1- A potent and powerful vasoconstrictor

- A- systemic vessels
- B- Efferent Post- Glomerular arterioles.
- Causing increase of intra-glomerular cap. pressure and maintain GFR.

2- Angiotensin-II- release ALDOSTERONE H.

Enhances Na- re absorption from collecting duct-  
maintaining-BP- renal perfusion.

– RENAL AUTO-REGULATION- MECHANISM-  
PATHOPHYSIOLOGY-OF PRE-RENAL-AKI-

Kidney also synthesis and release

PROCTAGLANDIN - PROSTACYCLIN- and NO .

Potent Afferent pre-glomerular arterioles

Vasodilators increasing renal perfusion and GFR.

AKI - happened

AUTOREGULATION - compromised or impaired

SEVER and PROLONGED

drop of Intra-vascular volume - and LOW - BP-

EFFECTIVE ARTERIAL BLOOD VOLUME AND FLOW- EABV.

sever and prolonged Hypotension.

Systolic BP- < 80mmHg .

# RENAL AUTO-REGULATION- MECHANISM- PATHOPHYSIOLOGY-OF PRE-RENAL-AKI-

Both NSAIDS- and ACEI- can cause AKI-

- -NSADI
  - blocks Prostaglandin-
  - USEFUL Afferent pre- glomerular Renal Vasodilators
- ARBs- ACEI-
- blocks- Angiotensine II-
  - USEFUL Efferent post-glomerular Renal vasoconstrictors.
- Especially- when renal function is compromised -
  - Elderly
  - Diabetic nephropathy
  - CKD

# PATHOPHYSIOLOGY-OF PRE-RENAL-AKI-

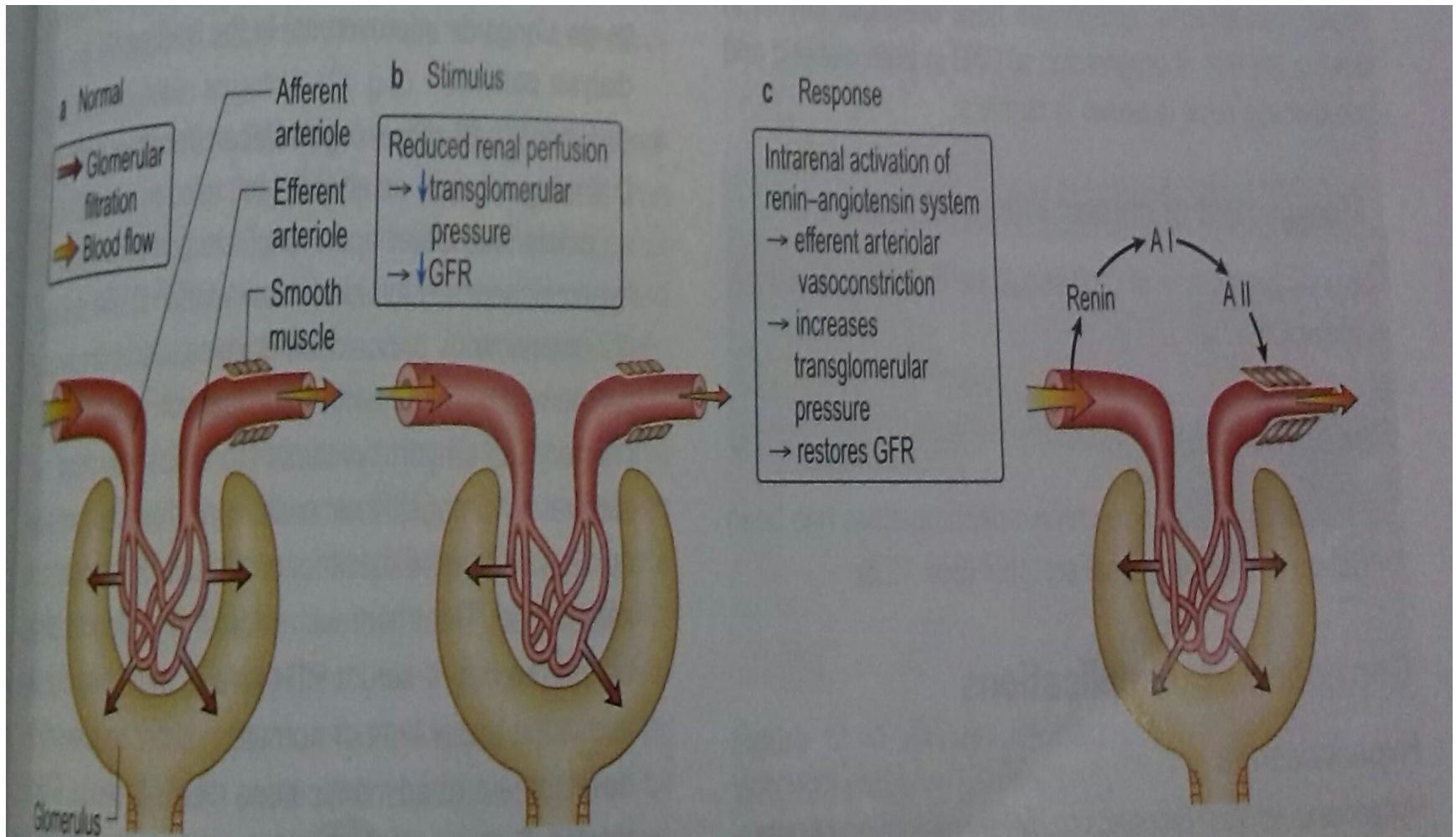
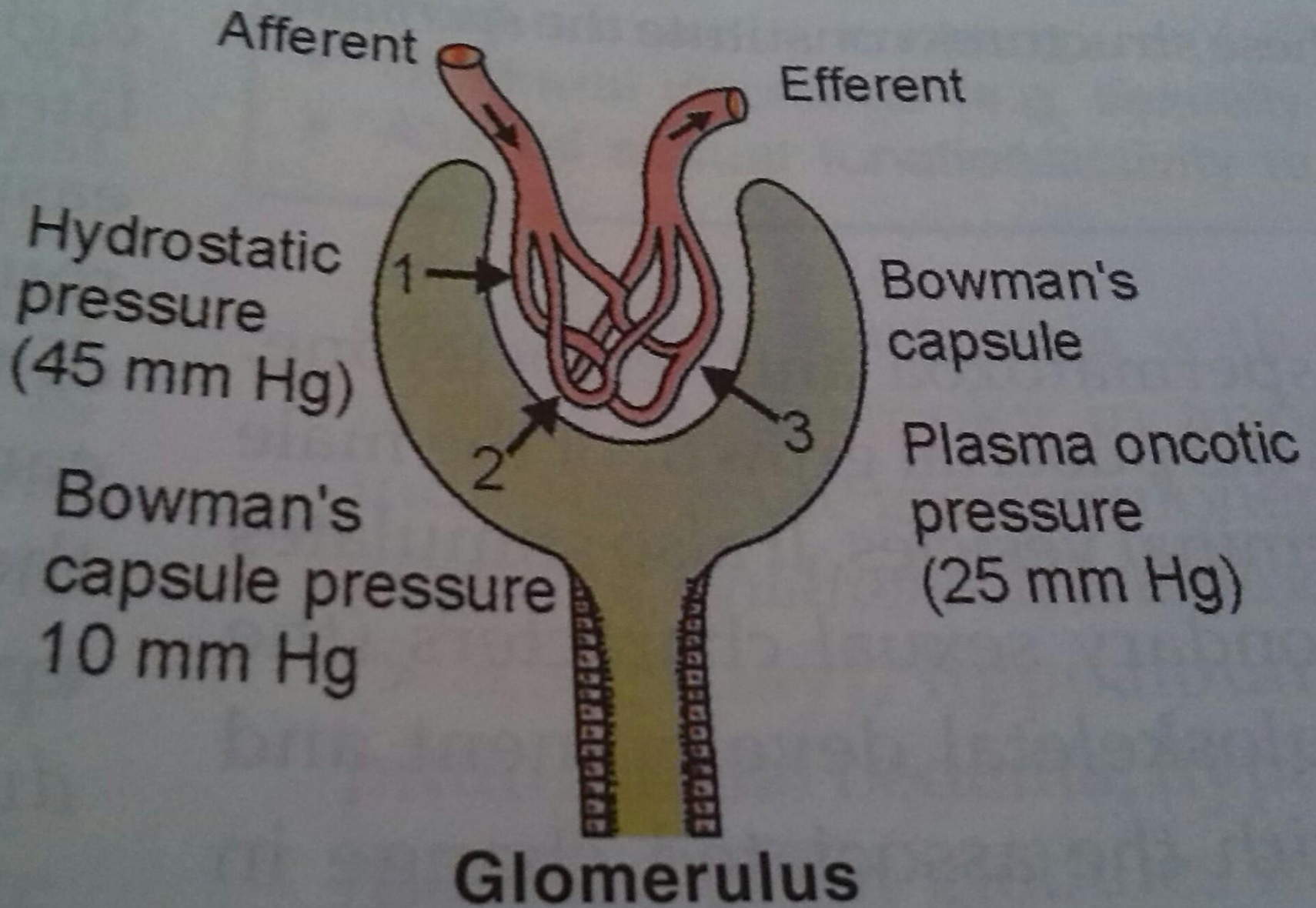


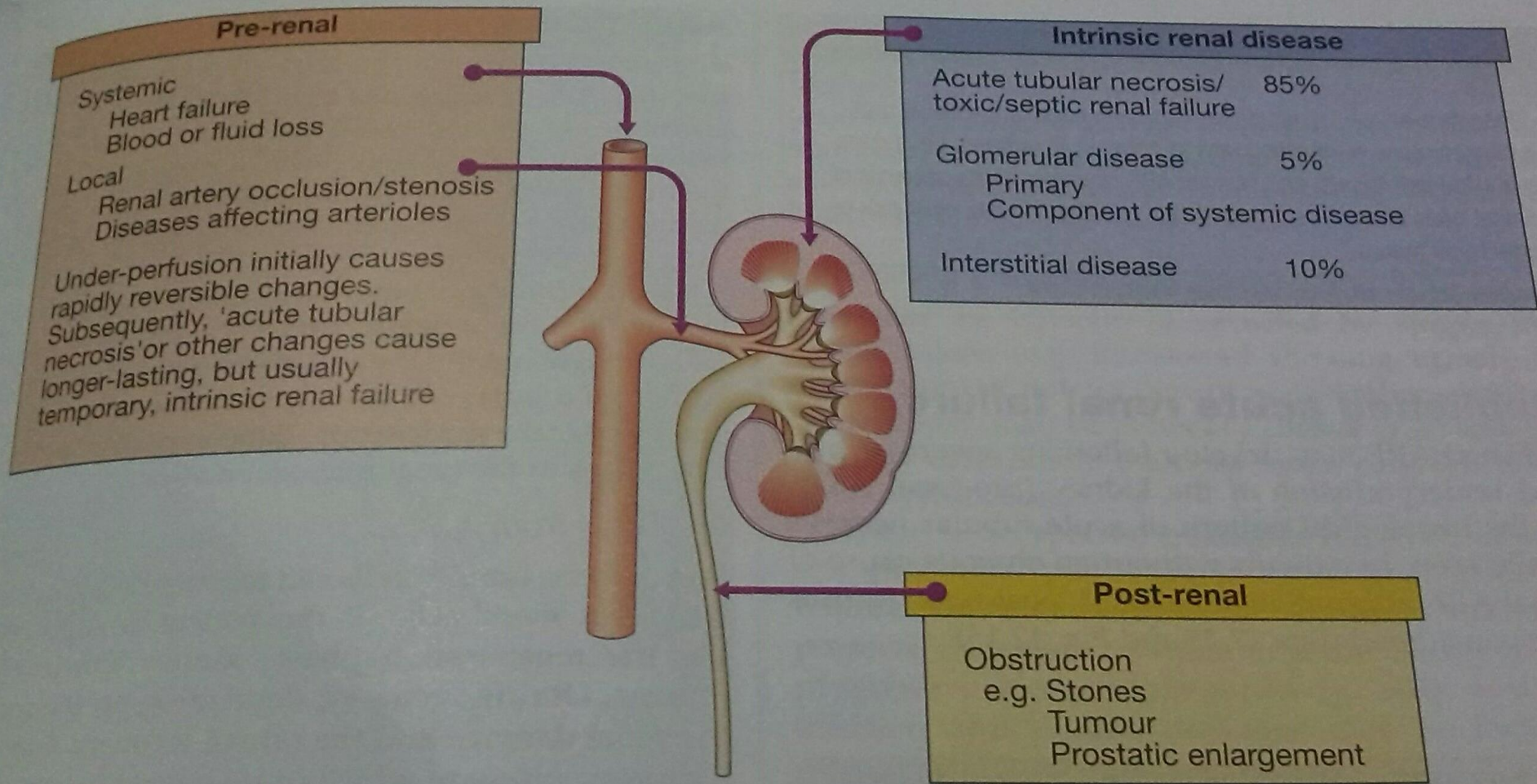
Figure 12.48 Glomerular dynamics: effect of the renin-angiotensin system. A I, angiotensin I; A II, angiotensin II.



# CLASSIFICATION OF AKI

- 1- PRE-RENAL –
  - HYPOVOLAEMIA- HYPOTENSION-EABV-TOXIN
- 2- RENAL-AKI-
  - GLOMERULI- TUBULES- INTERSTITIUM
- 3- POST-RENAL- AKI-
  - URINARY OBSTRUCTION
- Overlap more than one group

Presenting problems in renal and urinary tract disease



# PRE-RENAL- AKI

## AETIOLOGY

I - HYPOVOLAEMIA- COMMONEST

A- Hamorrhage - BURN

B- GIT- Fluid loss- vomiting- diarrhea- dehydration-  
Surgical wound drain- NGT- tube aspiration.

C- Renal- Fluid loss- diuretics- Osmotic diuresis-

- Diabetic keto-acidosis

D- Sequestration fluid in extra vascular space-

THIRD SPACE-

ABDOMINAL COMPARTMENT SYNDROME –ACS-

HIGH Intra- Abdominal pressure—

Organs dysfunction- ISCHAEMIA -AKI

intra- peritoneal bleeding – Massive Ascitis-

Intestinal obstruction- Acute Pancreatitis- Trauma.



# PRE-RENAL- AKI

II- LOW CARDIAC OUT PUT.

HAEMODYNAMICALLY UNSTABLE-CARDIO - RENAL- SY.

Acute – extensive - MI – CARDIOGENIC SHOCK

RV- MI

- CHF

Serious Arrhythmia

- AF- VT- VF

Pericardial Tamponad

Massive Pulmonary Embolism

# PRE-RENAL- AKI

- III - Altered renal- systemic vascular resistance-  
DROP- EFFECTIVE ARTERIAL BLOOD FLOW- EABF-
  - A- Systemic vasodilatation.
    - Septic shock - Anaphylaxis.
    - Anesthesia- Vasodilator drugs.
  - B- Liver cirrhosis- HEPATO-RENAL SY.
    - Sever Vasomotor disturbances
    - splanchnic vasodilatation –
    - intra-abdominal pooling of blood
    - Following liver cirrhosis- portal hypertension-ascites-
    - Reversible condition After restoring hepatic function.

# PRE-RENAL-AKI

- 1V- Large renal artery disease.
  - A- Atherosclerotic renal artery disease
- Renal artery stenosis
- Athero - emboli
  - Multiple Cholesterol emboli - KIDNEY damage
  - livedoreticularis-
  - eosinophilia - eosinophiluria
  - low complements- blue toes
- B- Renal vein occlusion.

# PRE-RENAL-AKI

V- Small vessels occlusion –MICRO-ANGIOPATHY

HUS- T T P- DIC - Scleroderma - RENAL CRISIS-

Malignant - HPT

- Toxemia of pregnancy-
- Pre-eclampsia - Eclampsia.

• VI- Glomerular diseases- vasculitis

NEPHRITIC PRESENTATION

- Acute Proliferative- POST- INFECTION - GN-

RPGN - Crescentic GN- SLE

WEGNERS GRANULOMA - Good Pastures syn.

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# RENAL-AKI

## GLOMER.-TUBULES-INTERSTIAL

### VII- Tubulo-Interstitial nephritis- TIN

- A- Allergic-interstitial nephritis.
  - Drugs- Acute phosphate nephropathy-
  - bowel purgative- sodium phosphate
  - Antibiotics- -Sulfa- Refampicin-
  - Pencillin- Diuretics- NSAIDS- PPI.
- B- Infection—
  - Bacterial UTI- Reflux Uropathy – Vesico-ureteric reflux
  - Viral- CMV- EPV- HIV- KORONA VIRUS
- C- Infiltration-
  - lymphoma- leukaemia- Sarcoidosis.

# ACUTE-TUBULAR-NECROSIS-ATN

Acute tubular necrosis- ATN.

- This is the most common cause of
- RENAL- AKI- 85% of the cases.
- Usually REVERSIBLE recovers within 6 weeks.

- AETIOLOGY-

A- Severe and prolonged- renal Ischemia - AKI.

B- Nephrotoxic - AKI-

EXO - TOXINE-

Radio-contrast agents- sodium phosphate

Drugs- Aminoglycosides , Cyclosporine-

Chemotherapy- HEROIN.

# ATN

- ENDO - TOXINE –
- Myoglobin- Rhabdomyolysis-
- Haemoglobin- Intravascular haemolysis.
- UA Hype uracemia-
- Oxalat- Hyperoxalurea.
- Light chain- MM
- Hypercalcemia- Hyperparathyroidism-
- Nephrocalcinosis
- calcium Precipitate in side renal tubules.

# ATN- histopathology-

Structural renal tubular cells damage .

tubular cells effacement- flat- with necrosis.

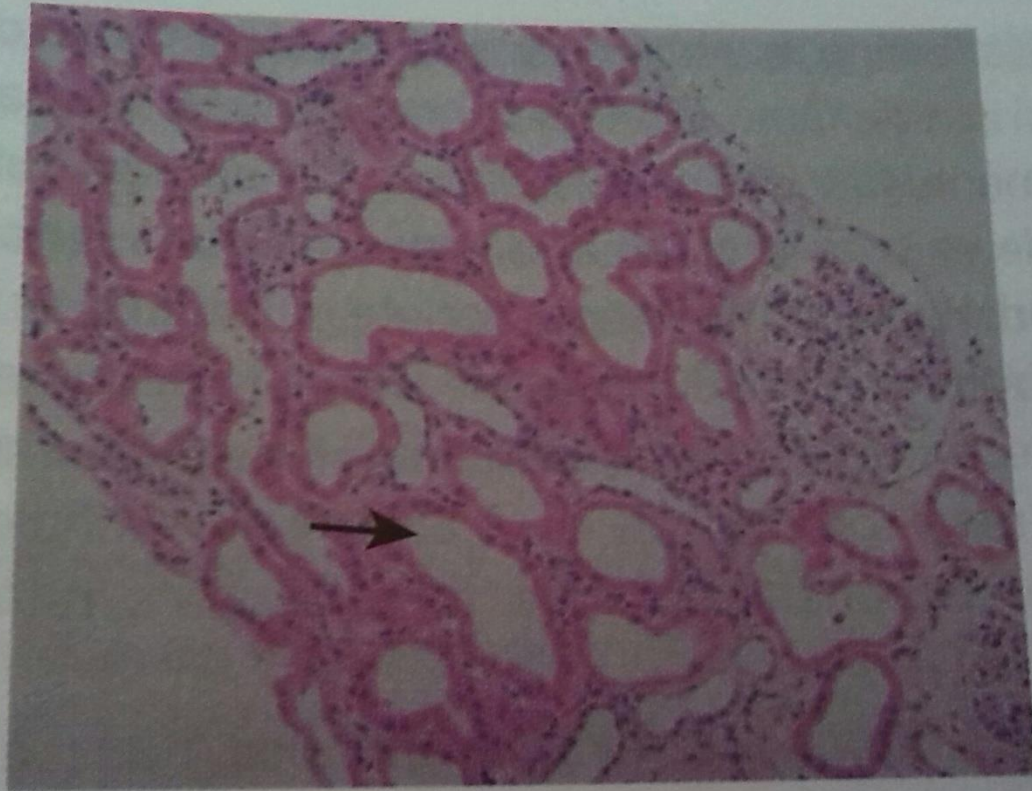
- Prox. tubular obstruction
- by desquamated debris necrotic epithelial cells .
- Tubular block - dilatation-
- tubule-glom. Feedback .

Interstitial odema

sever microvascular vasoconstriction .

- Leucocytes infiltration .
- Reversible within 6 weeks.





**Figure 12.44 Acute tubular necrosis** showing effacement and loss of the proximal tubule brush border, patchy loss of tubular cells and focal areas of proximal tubule dilatation (arrow).

# CLINICAL PRESENTATION-AKI

## 1- Early Oliguric phase

- Followed later on by Polyuric diuretic phase
- loss of renal tubular medullary urine
- concentration function.

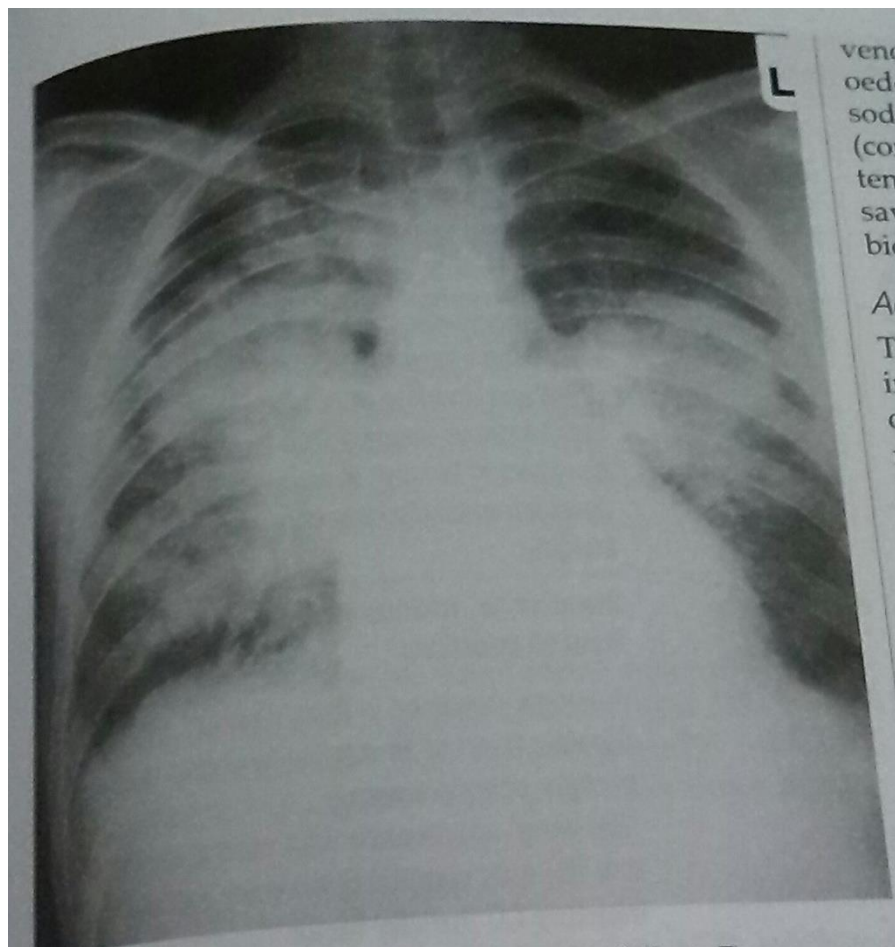
## 2- URAEMIC- Symptoms,

- Anorexia, Nausea, Vomiting,  
Hiccups, Pruritis, Drowsiness, Muscle-twitching-
- Apathy, Confused, Fit, Coma.

## 3- Metabolic acidosis- HYPERKALAEMIA

BUN/CREAT. ratio typically increased to  $> 20$

## 4-Acute Pul.Oedema due to acute fluid salt retention and high BP, causing- Acute-LV-FAILURE



# LAB- CRITERIA- DIFFERENCIATE BETWEEN-PRE-RENAL AND-RENAL ARF

	Pre-renal	Intrinsic
Urine specific gravity	>1.020	<1.010
Urine osmolality (mOsm/kg)	>500	<350
Urine sodium (mmol/L)	<20	>40
Fractional excretion of Na- ratio of Na clearance To creatinine clearance	<1%	>1%

# RHABDOMYOLYSIS-AKI

muscle damage resulting in release  
muscle enzymes, myoglobin, and electrolytes into blood.

## AETIOLOGY

TRAUMA- CRUSH INJURY- COMA- SEIZURES - HEATSTROKE-  
HEAVY EXERCISE- MARATHON RUN- FOOTBALL.

DRUGS- COCAINE, STATINS, COLCHICINE, ANESTHESIA.

INFECTIONS- VIRAL INFLUENZA,

ENDOCRINE HYPO AND HYPERTHYROIDISM- ALCOHOL

ELECTROLYTES - HYPOKALEMIA- HYPOPHOSPHATEMIA.

CLINICALLY- MUSCLE PAIN- AND DARK URIN - OLIGURIA

LAB.- HIGH- CPK- AST- ALT- HYPERKALEMIA-  
HYPERPHOSPHATEMIA- HYPERURICEMIA.

URINE- MYOGLOBLIN PIGMENT-COARSE GRANULAR CASTS IN URIN

# MANGEMENT OF AKI-

- 1- IV- fluid replacement is the treatment of choice.
  - Restoring normal GFR .
  - Close cardiovascular monitoring-
  - BP- HR- JVP- guided by CVP-LINE-
  - to avoid fluid over load and pulmonary edema.
  - SEVER cases Hemofiltration – HAEMODIALYSIS.
- 2- U/S-ABD. Is important .
- 3- Treat the underling cause stop offending drugs.
- 4- Treat Emergency complications
  - ACCELERATED HYPERTNSION-HIGH-BP
  - ACUTE PULMONARY OEDEMA
  - Metabolic Acidosis- Hyper-kalaemia-Sepsis-blood loss.

# CONTRAST NEPHROPATHY

It is a common clinical problem.

- Iatrogenic complication.
- Caused by iodinated radioactive contrast agents used for X- RAY-procedures.
- Cor. and peripheral Angiography- PCI.
- This contrast agents have both Nephrotoxic and Vasoconstrictor effects .
- Especially in poorly prepared
- Elderly- Dehydrated -
- DM- pre-existing CKD-

# CONTRAST NEPHROPATHY

- PREVENTION-
  - 1- Using iso- or hypo-osmolar agents- to avoid kidney injury.
  - 2- Good rehydration measures .
    - IV- 1L- 0.9% N/S
    - 12 h before and after contrast agents.
  - 3- CKD-patients .
    - Peri - X-RAY- during procedure
    - Haemofiltration should be done.



# POST-RENAL AKI-

- Any acute renal obstructing cause from renal calyces down to external urethral orifice- AKI.
  - Clinical Presentations- U/S- abd. Should be done
    - Renal colic
    - Haematuria
    - UTI- UROSEPSIS - Fever
    - Hydro-nephrosis-
    - Urine- Retention
- Urological consultation.

# Aetiology-

1- Within urinary tract lumen.

- Stones- Blood clots
- Papillary Necrosis
- Renal pelvis tumor-
- Urinary bladder tumor.

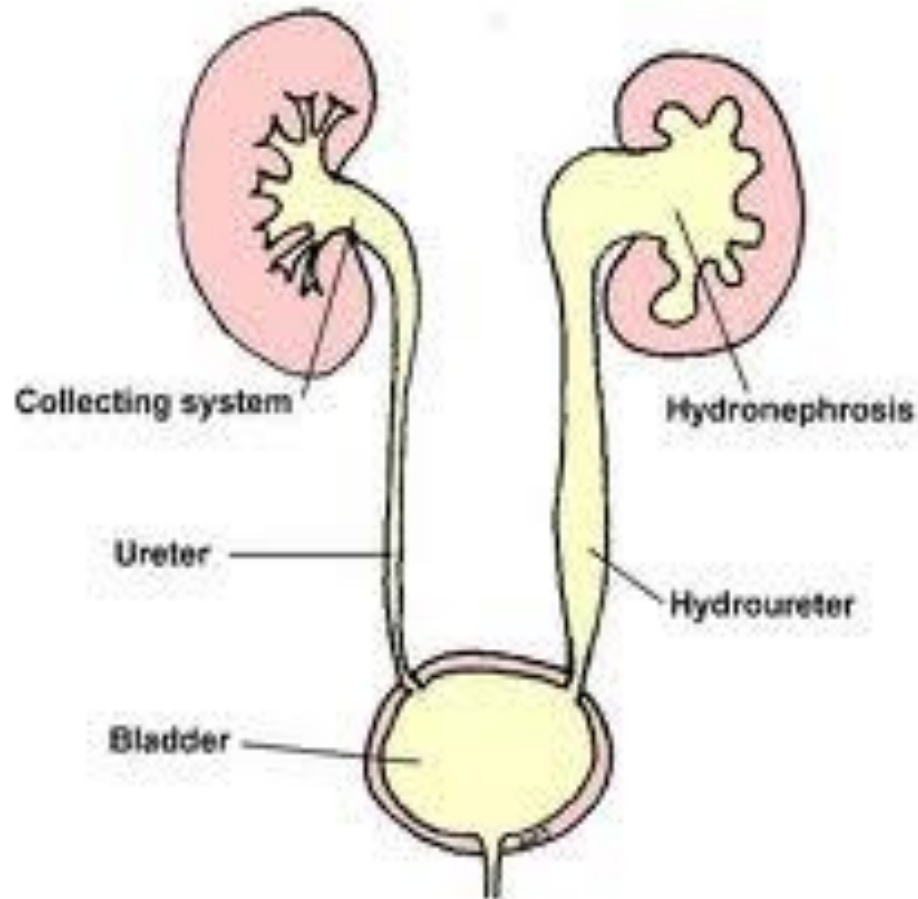
2- Within the wall of urinary tract .

- Cong. pelviureteric junction dysfunction.
- Ureteric or Urethral STRICTURE
- Schistosomiasis - Post-Surgery- GC.

3- Pressure from outside-

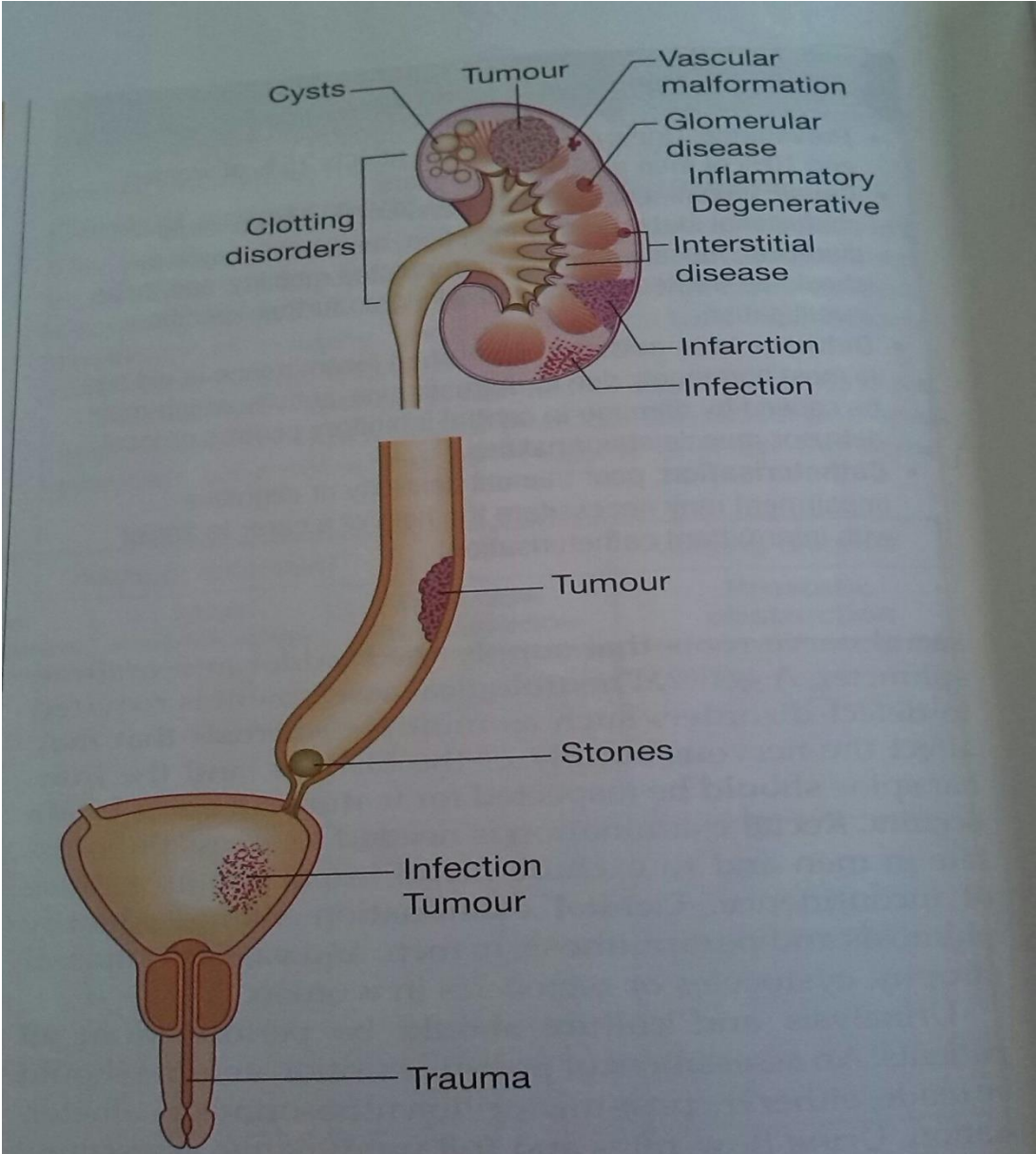
- Aberrant artery- BPH
- Retroperitoneal tumor and Fibrosis.

# POST-RENAL ARF-



# HYDRONEPHROSIS





**Fig. 17.12** Causes of haematuria.