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 DISALYSIS QUALITY INITIATIVE GROUP

AKI - differenciate from CKD

AKI- on - CKD.

RIFLE classify- AKI

Three levels

R- I- F

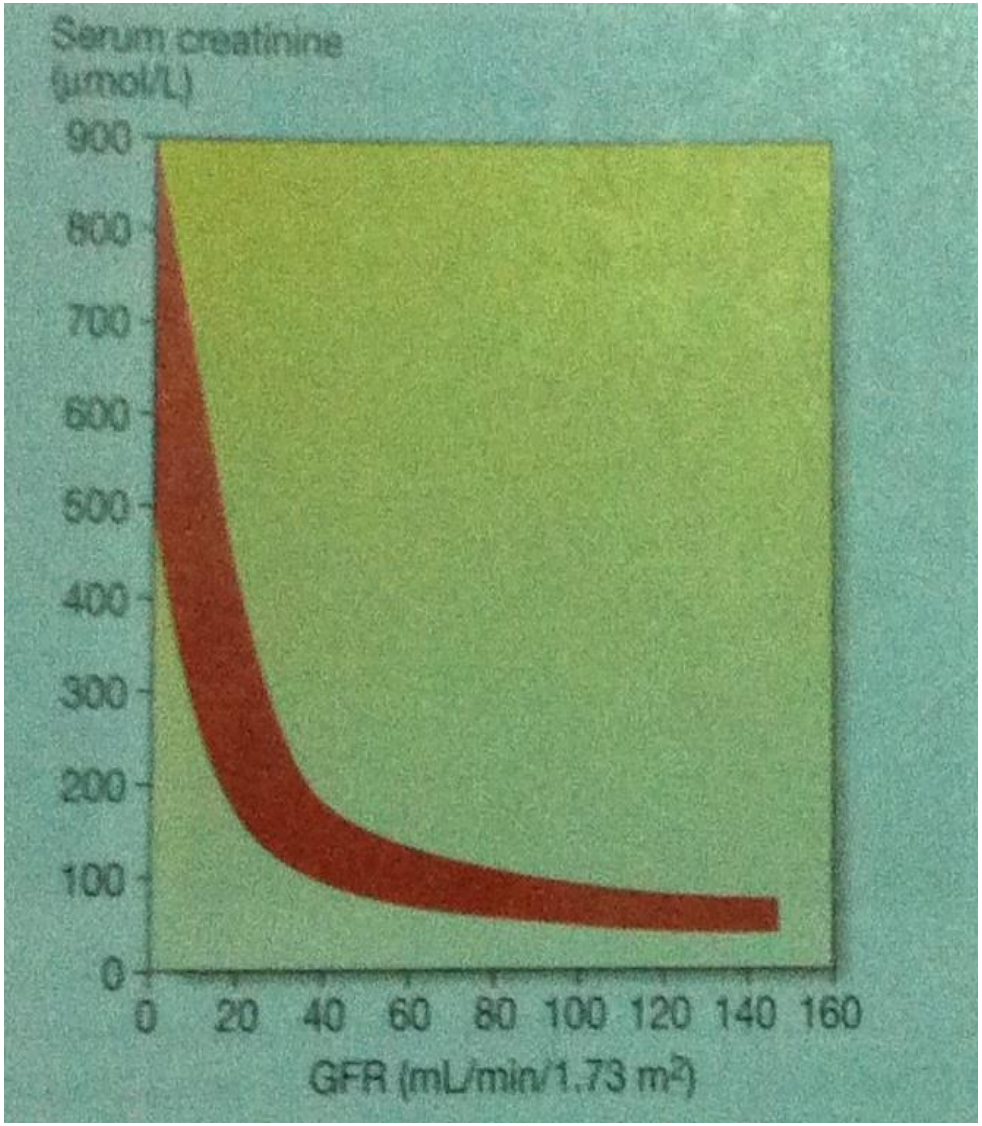
Two out comes

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	


Serum creatinine
(μmol/L)
900
800
700
600
500
400
300
200
100
0
0 20 40 60 80 100 120 140 160
GFR (mL/min/1.73 m²)

GFR-CREATININE

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

EPIDEMOLOGY

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 micr-mol/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 seq. surface area.

  DAILY alteration and variation of renal perfusion pressure.

- AUTOREGULATION

  Kidney releases RENIN from JUXTA-GLOMERULAR-APPARATUS

  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-

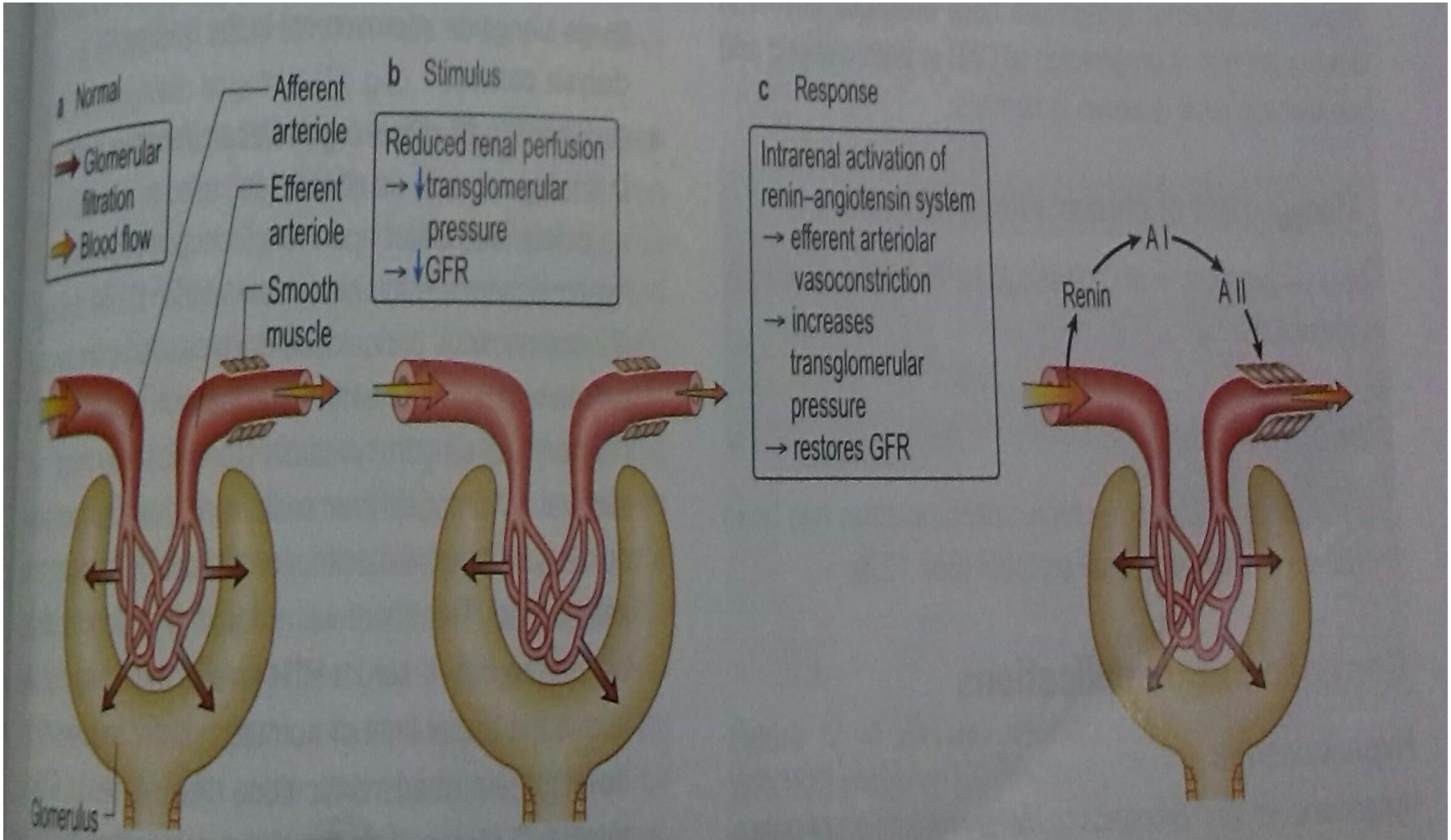
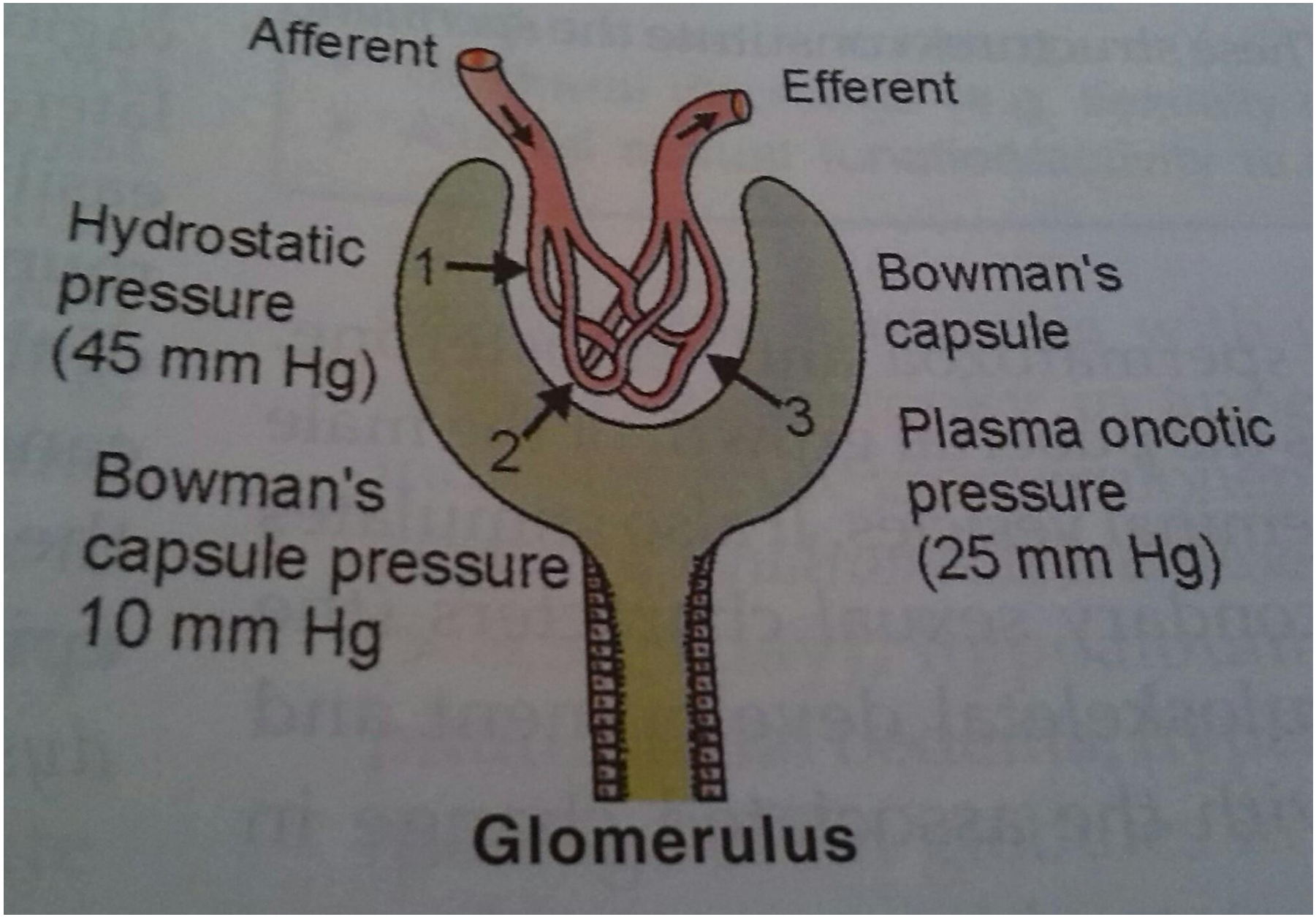
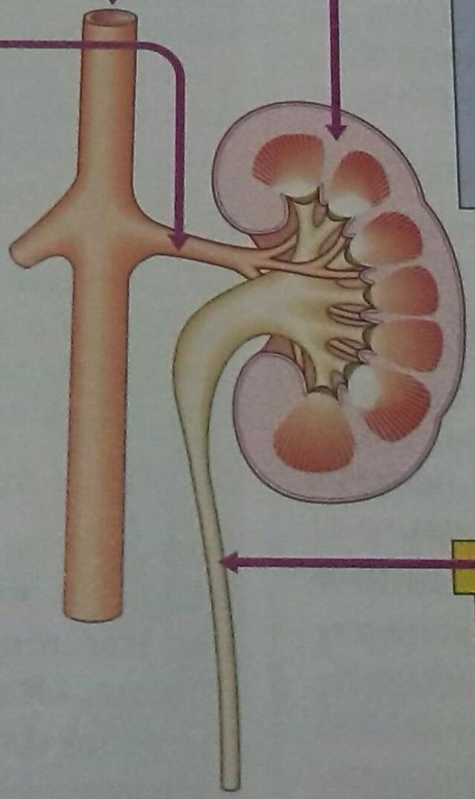


Figure 12.48 **Glomerular dynamics:** effect of the renin-angiotensin system. AI, angiotensin I; AII, angiotensin II.


Afferent
Efferent
Hydrostatic pressure (45 mm Hg)
1
Bowman's capsule
2
3
Plasma oncotic pressure (25 mm Hg)
Bowman's capsule pressure 10 mm Hg
Glomerulus

CLSSIFICATION OF AKI

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



Pre-renal

Systemic
- *Heart failure*
- *Blood or fluid loss*

Local
- *Renal artery occlusion/stenosis*
- *Diseases affecting arterioles*

Under-perfusion initially causes rapidly reversible changes. Subsequently, 'acute tubular necrosis' or other changes cause longer-lasting, but usually temporary, intrinsic renal failure

Intrinsic renal disease

Acute tubular necrosis/ toxic/septic renal failure	85%
Glomerular disease	5%
Primary	
Component of systemic disease	
Interstitial disease	10%

Post-renal

Obstruction
- e.g. Stones
- Tumour
- Prostatic enlargement

15 Causes of acute renal failure.

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 Tamponade

  Massive Pulmonary Embolism

PRE-RENAL- AKI

III - Altered renal- systemic vascular resistance-
DROP- EFFECTIVE ARETERIAL 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

  livedo-reticularis-

  eosinophila - 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.

-
-

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- Infilteration-

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- Sever and prolonged- renal Ischemia - AKI.

B- Nephrotoxic - AKI-

EXO - TOXINE-

Radio-contrast agents- sodium phosphate

Drugs- Aminoglycosides , Cyclosporine-

Chemotherapy- HEROIN.

ATN

- ENDO - TOXINE –
- Myoglobulin- 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 debrise necrotic epithelial cells .
- Tubular block - dilatation- tubule-glom. feedback.

Interstitial odema

sever microvascular vasoconstriction .

- Leucocytes infilteration .
- Reversible within 6 weeks.

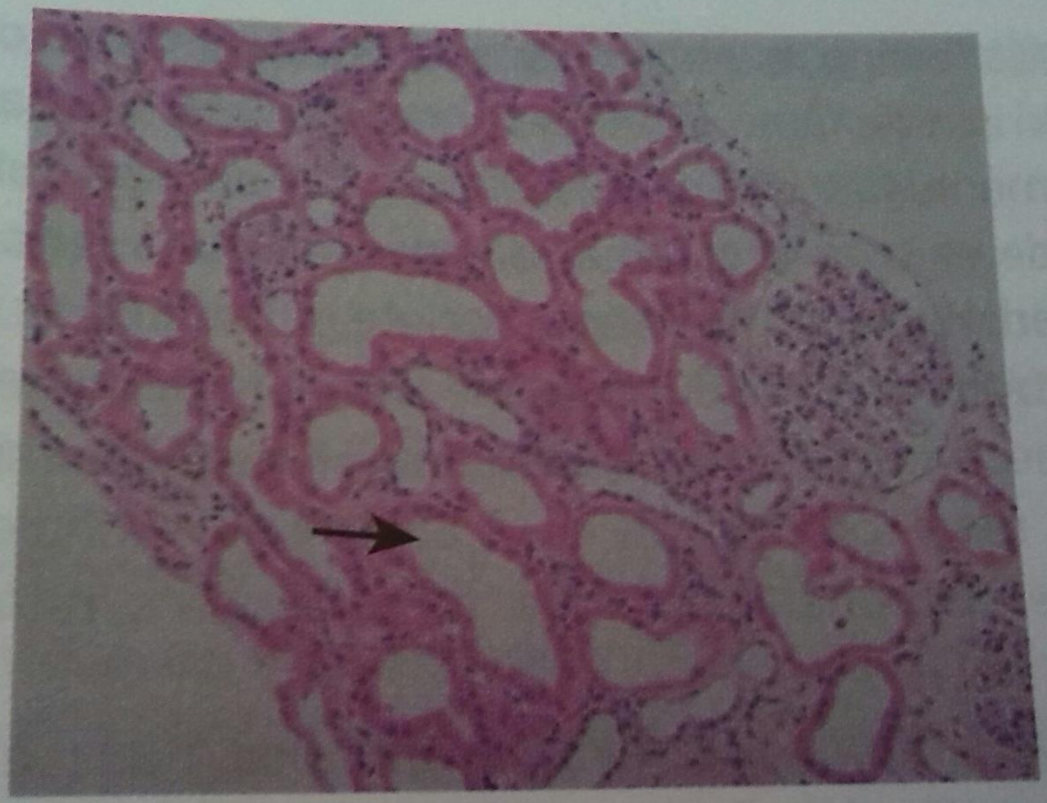


Figure 12.44 **Acute tubular necrosis** showing effacement and loss of the proximal tubule brush border, patchy loss of tubular cells and focal areas of proximal tubule dilatation (arrow).

CLINICAL PRESENTATION-AKI

1- Early Oliguric phase

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

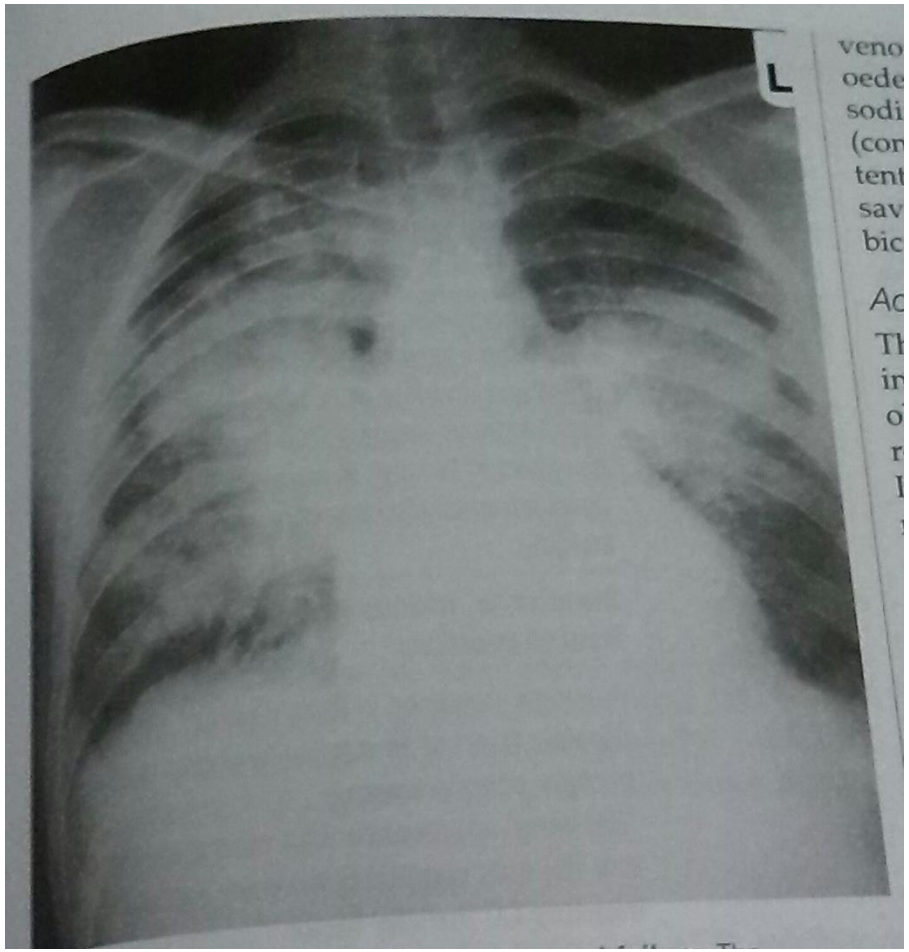
2- URAEMIC- Symptoms,

- Anorexia, Nausea, Vomiting,
  Hiccups, Pruritis, Drowziness, 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, myoglobulin, and electrolytes into blood.

AETIOLOGY

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

DRUGS- COCAINE, STATINS, COLCHICINE, ANESTHESIA.

INFECTIONS- VIRAL INFLUENZA,

ENDOCRINE HYPO AND HYPERTHYROIDISM- ALCHOHOL

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 Hemofilteration – 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
- Haemofilteration 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 .

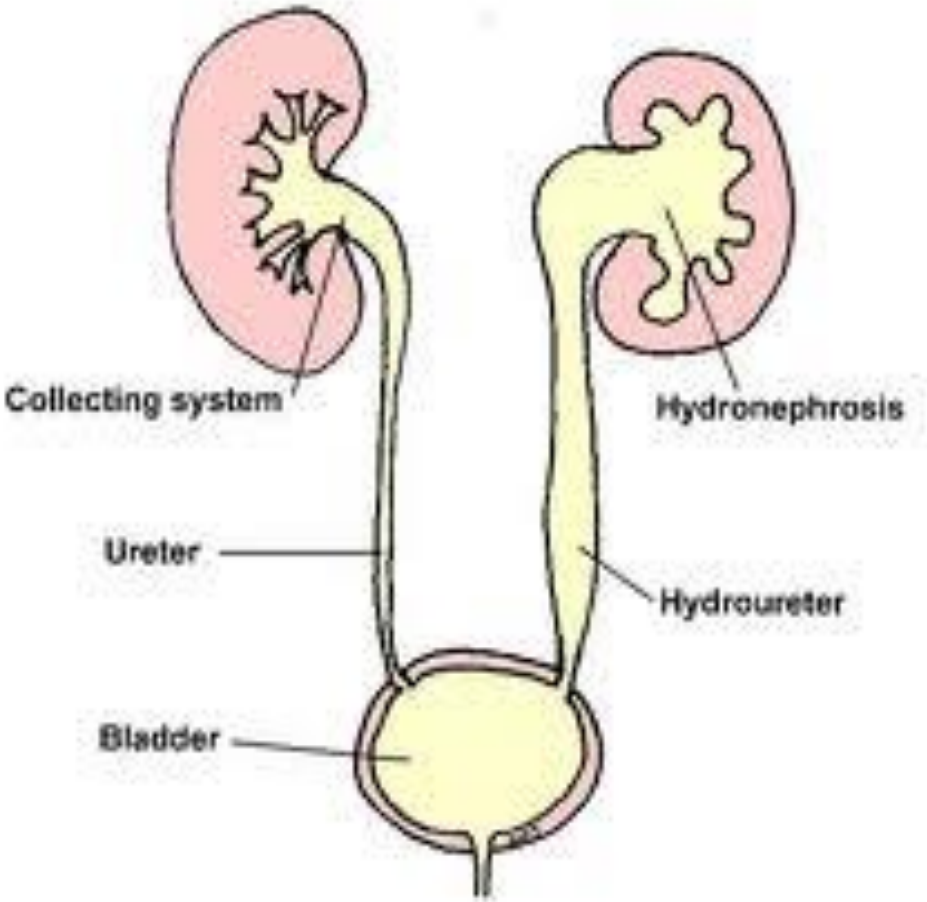
- Cong. pelviureteric junction dysfunction.
- Ureteric or Urethral STRICTURE

Schistosomiasis - Post-Surgery- GC.

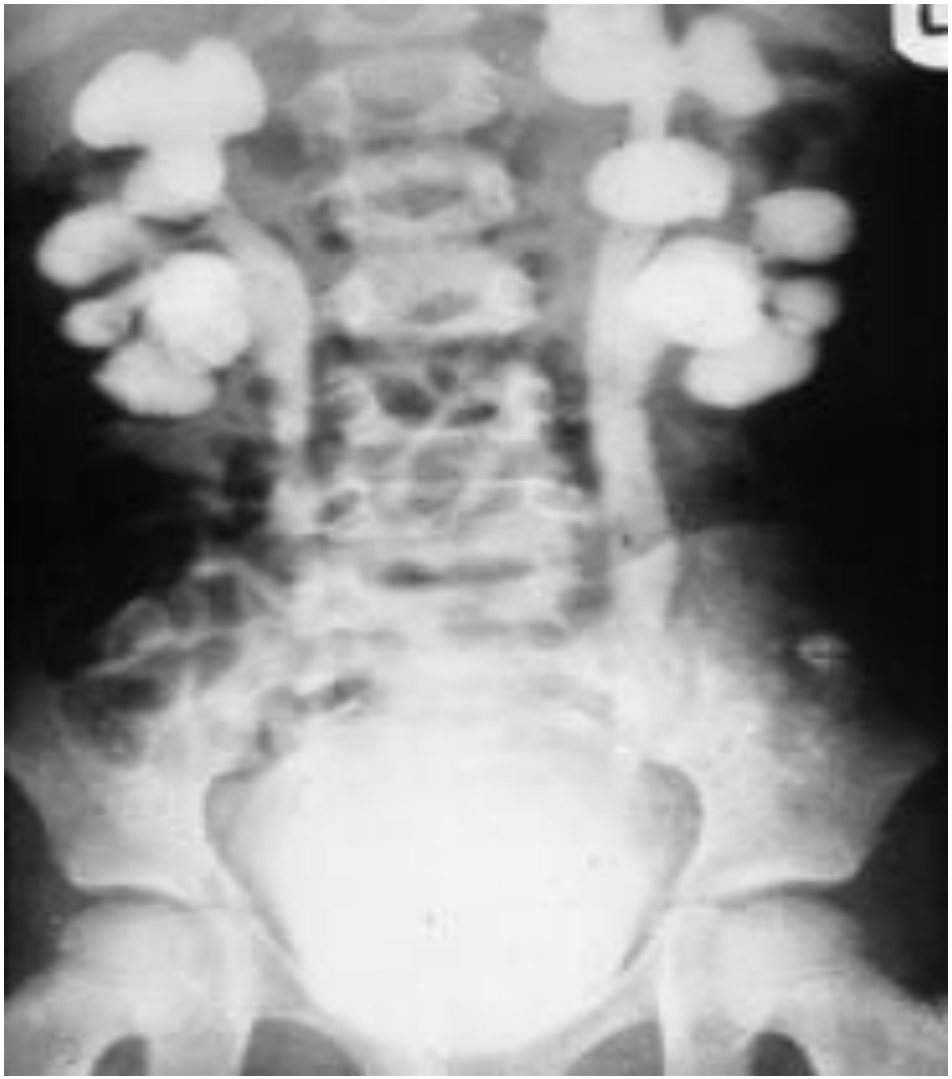
3- Pressure from out side-

- Aberrant artery- BPH
- Retroperitoneal tumor and Fibrosis.

POST-RENAL ARF-



HYDRONEPHROSIS



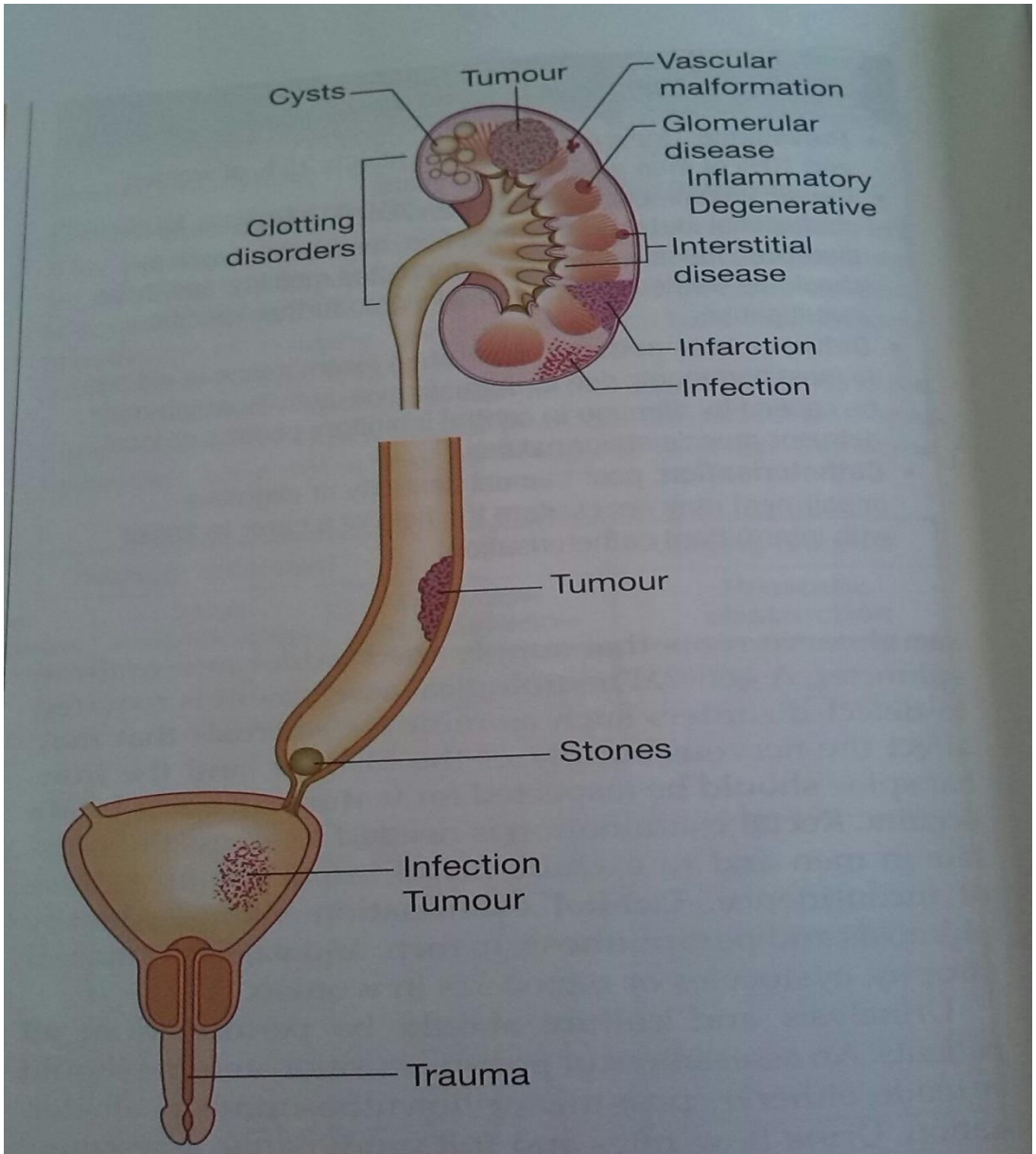


Fig. 17.12 Causes of haematuria.