Integrated Teaching
On

Glomerulonephritis

Date: 23-05-2023, Time: 9:30 am
Venue: Lecture Gallery-2

Glomerulonephritis

Anatomy

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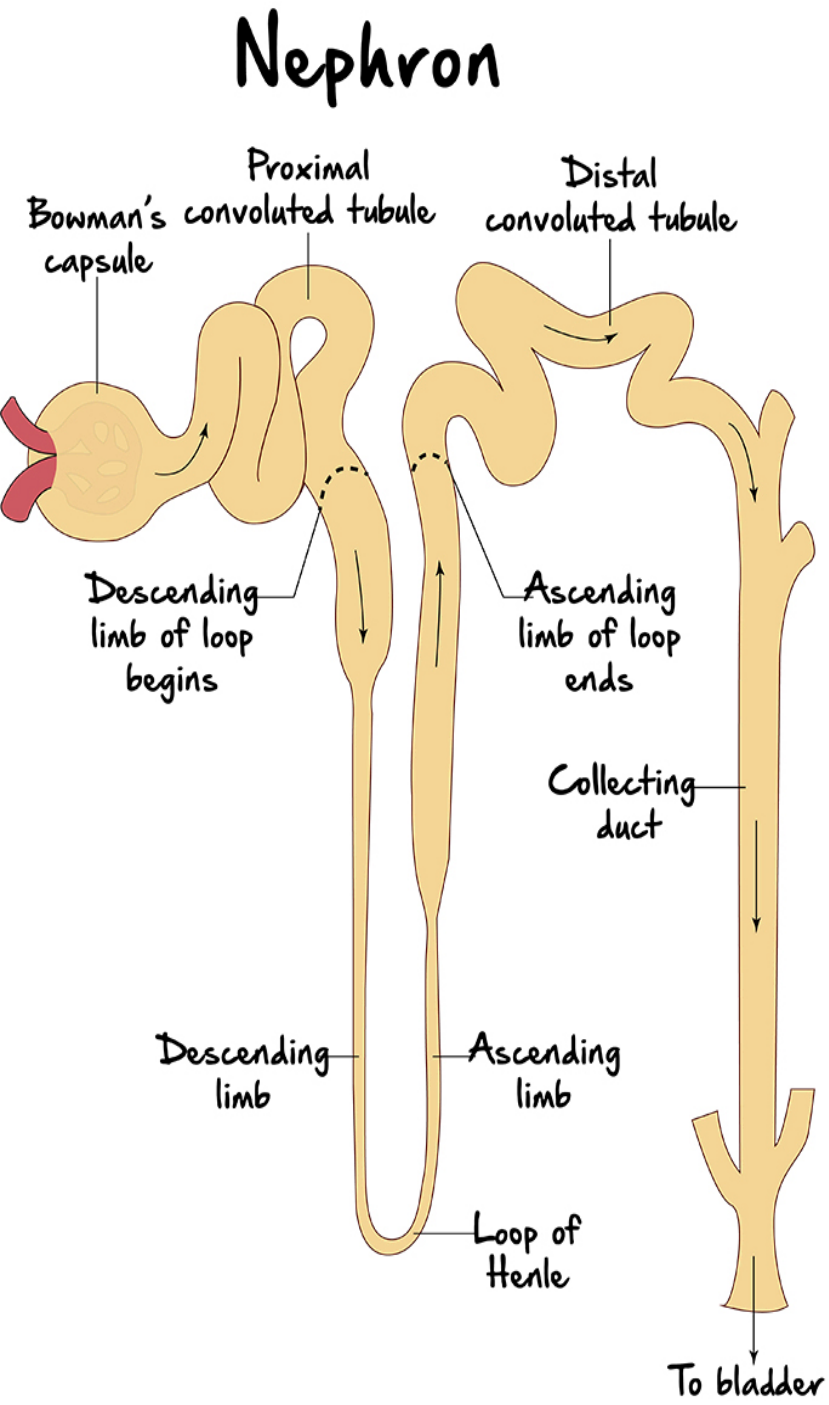
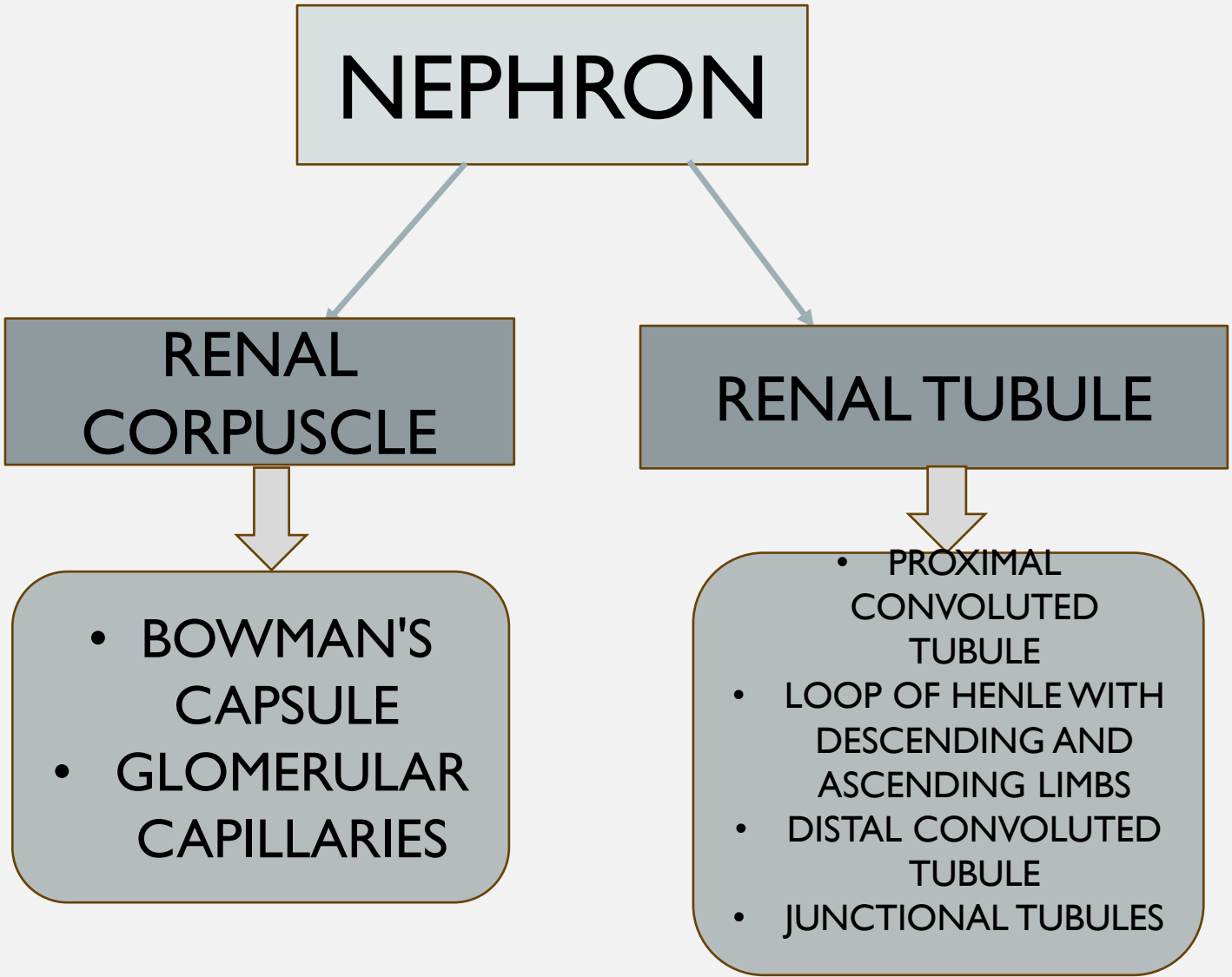
NEPHRONS

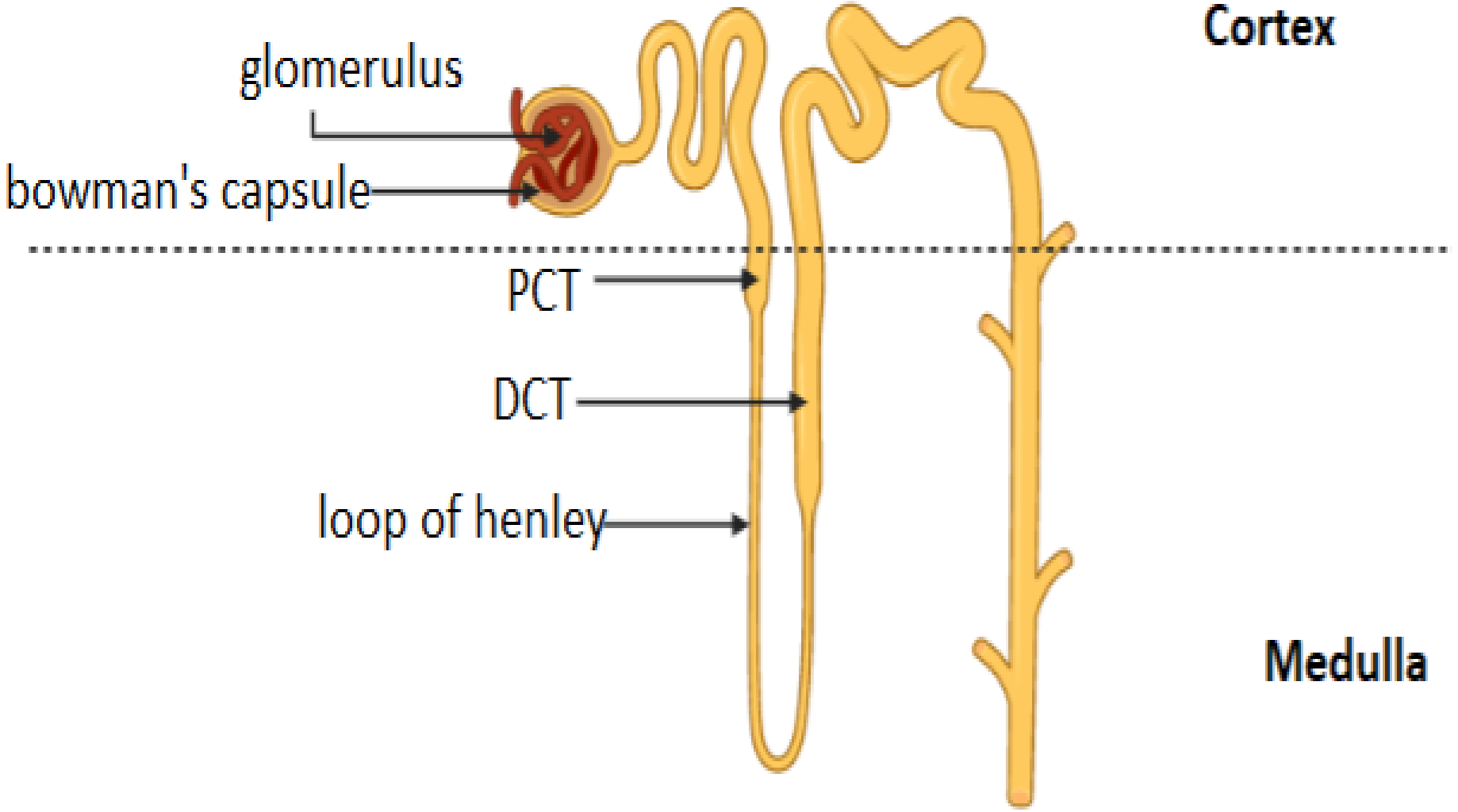


-The nephron is the functional unit of the kidney

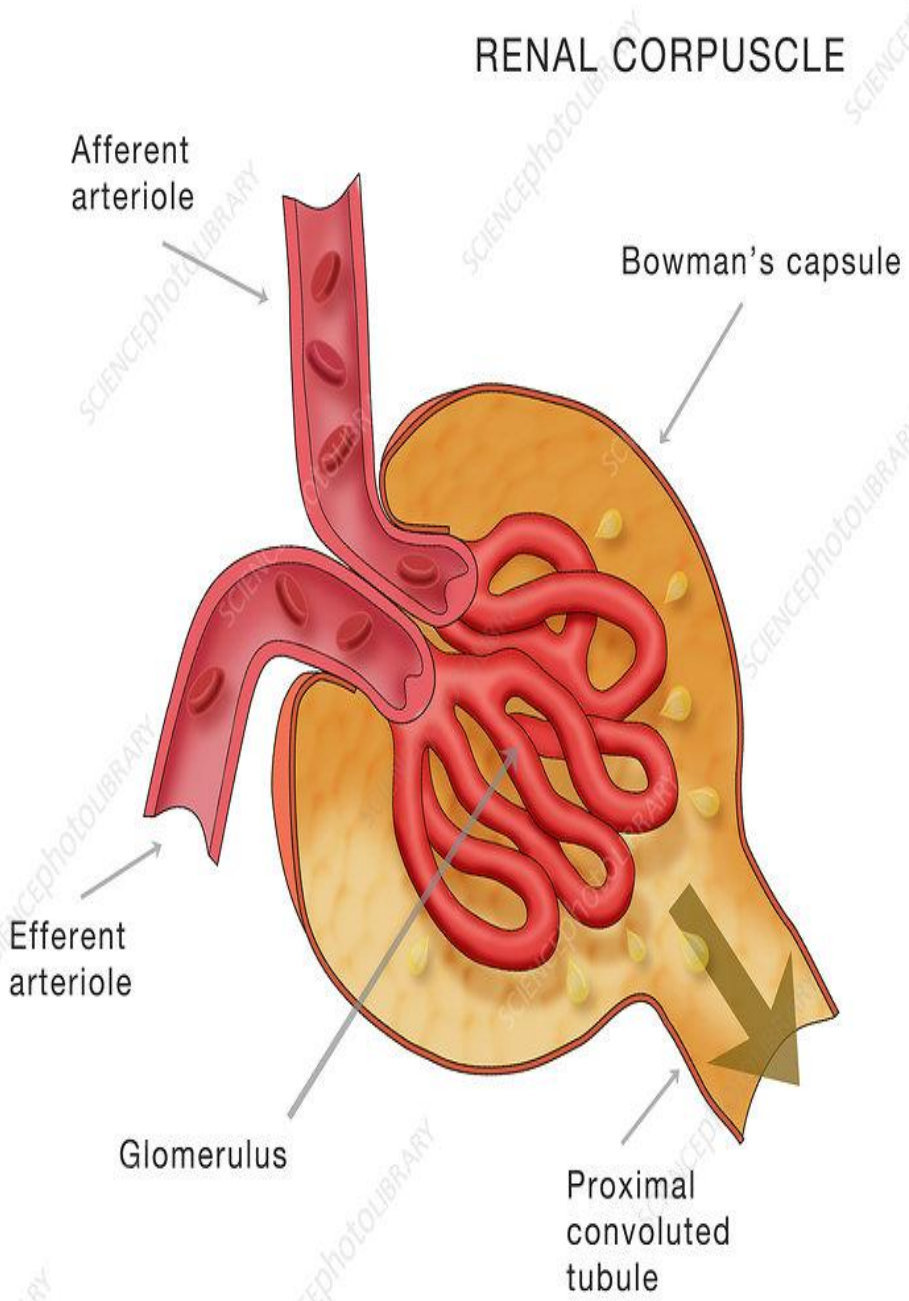
-In adults, each kidney contains approximately one million nephrons

PARTS OF NEPHRON



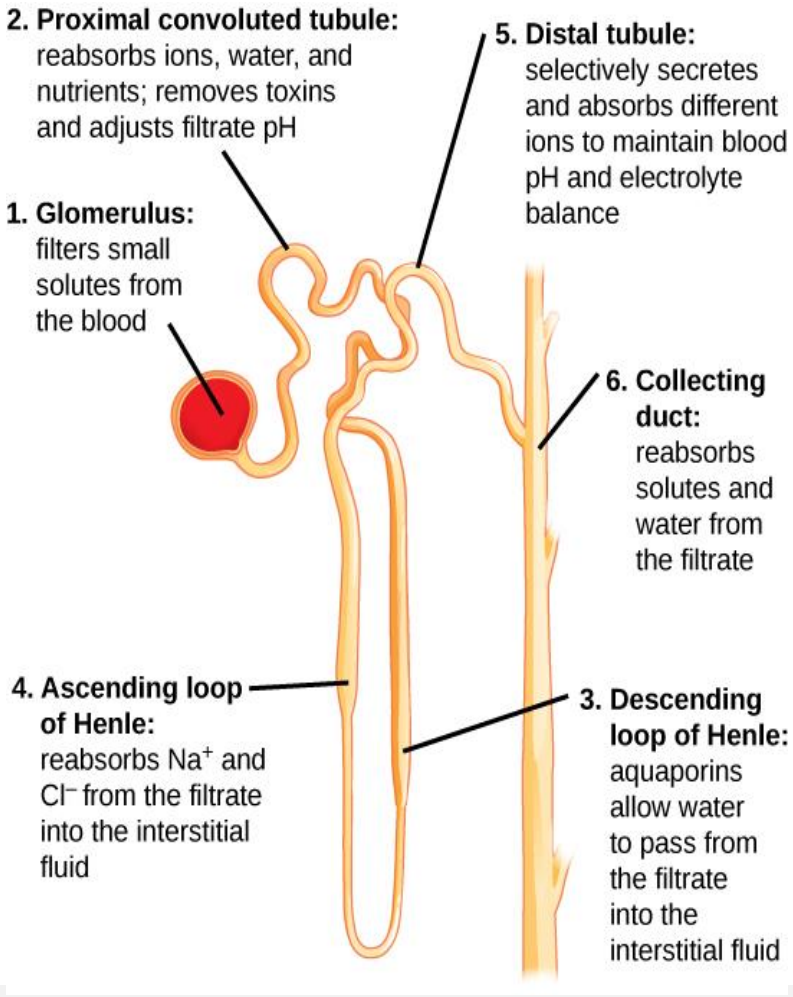

Cortex
glomerulus
bowman's capsule
PCT
DCT
loop of henley
Medulla

Nephron



RENAL CORPUSCLE FUNCTION

The capsule helps the glomerulus to filter blood


2. Proximal convoluted tubule: reabsorbs ions, water, and nutrients; removes toxins and adjusts filtrate pH
5. Distal tubule: selectively secretes and absorbs different ions to maintain blood pH and electrolyte balance
1. Glomerulus: filters small solutes from the blood
6. Collecting duct: reabsorbs solutes and water from the filtrate
4. Ascending loop of Henle: reabsorbs Na^+ and Cl^- from the filtrate into the interstitial fluid
3. Descending loop of Henle: aquaporins allow water to pass from the filtrate into the interstitial fluid

Glomerulonephritis

Pathology

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Principal
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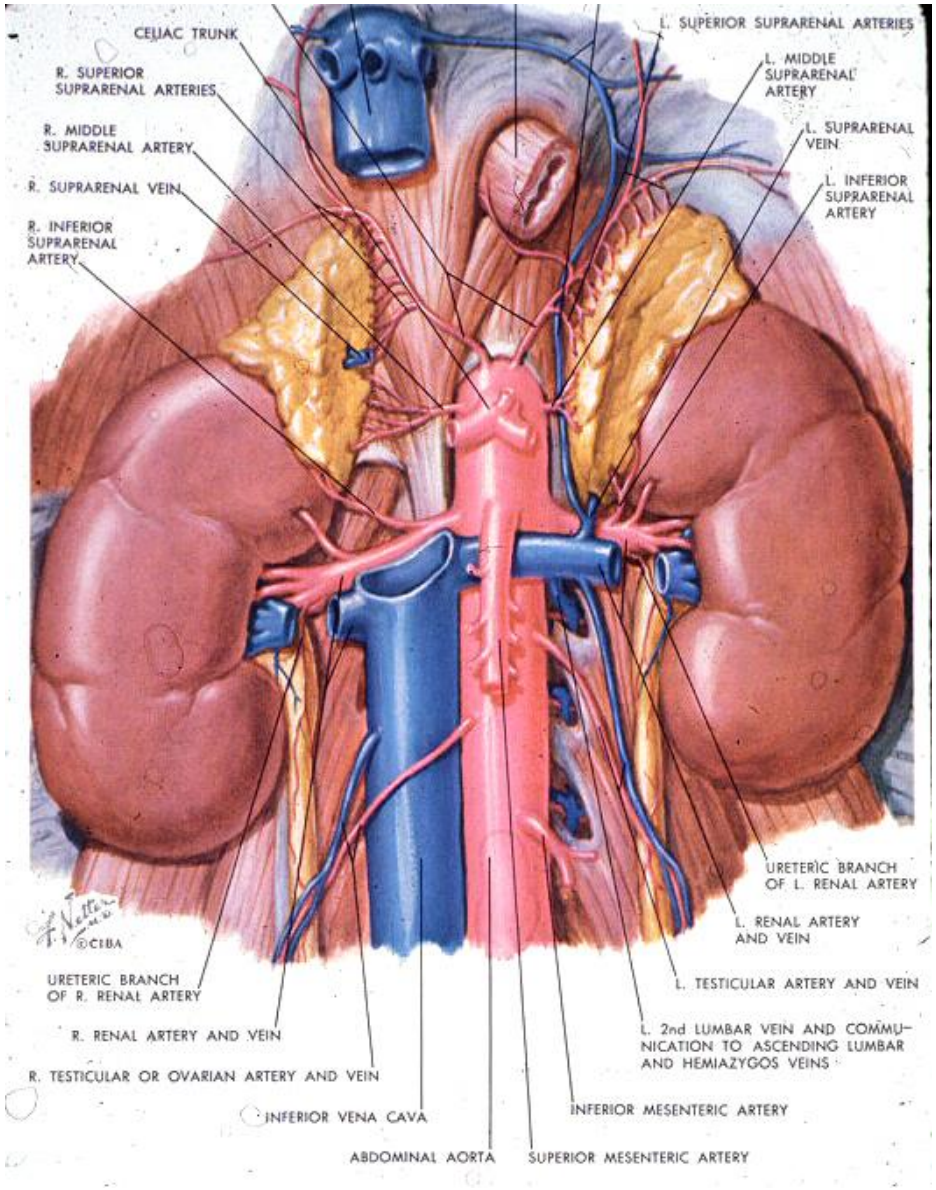
Objectives

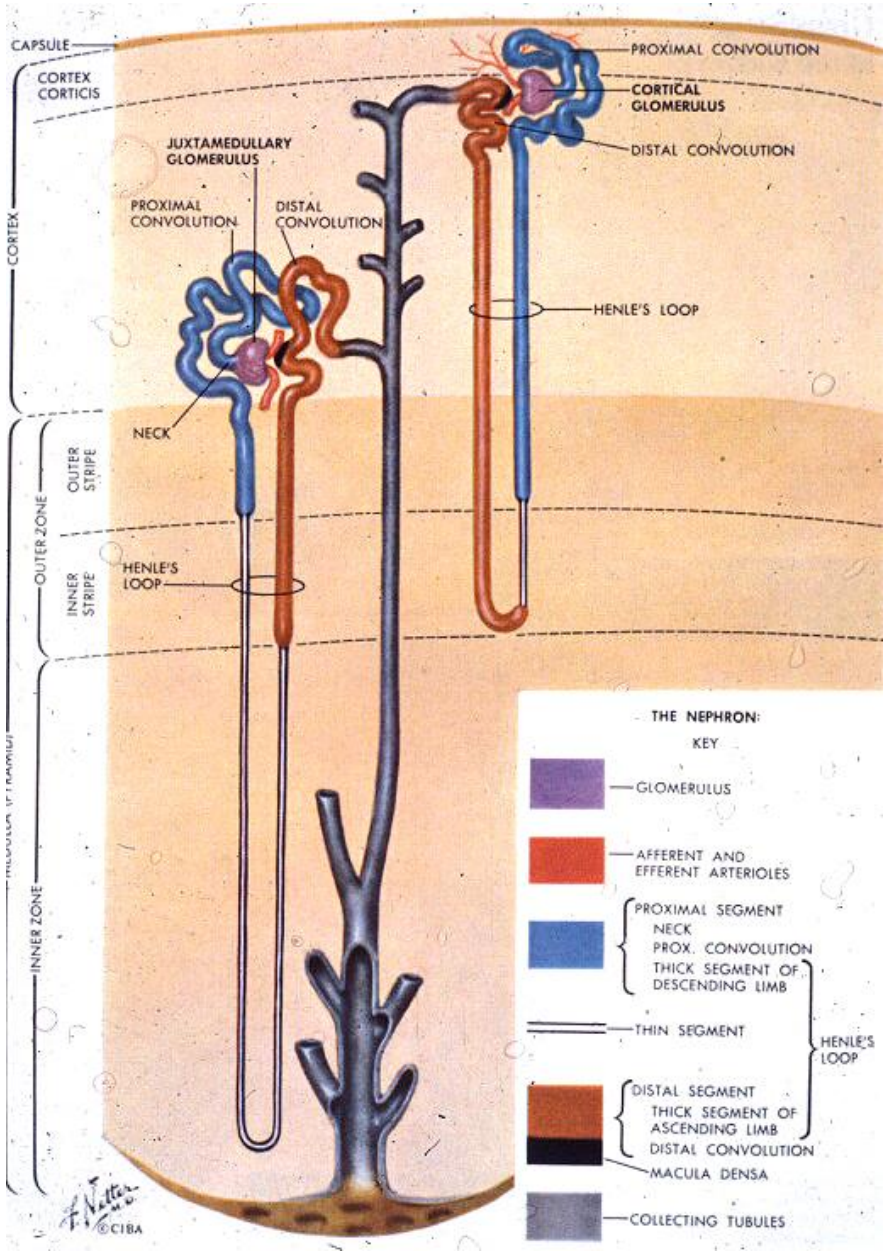
After completion of the class students will be able to

- Introduce the glomerulonephritis.
- Define glomerulonephritis
- Enlist the types of glomerulonephritis
- Explain the Causes of glomerulonephritis
- Explain the Pathophysiology of glomerulonephritis
- Enumerate the Clinical manifestations glomerulonephritis.

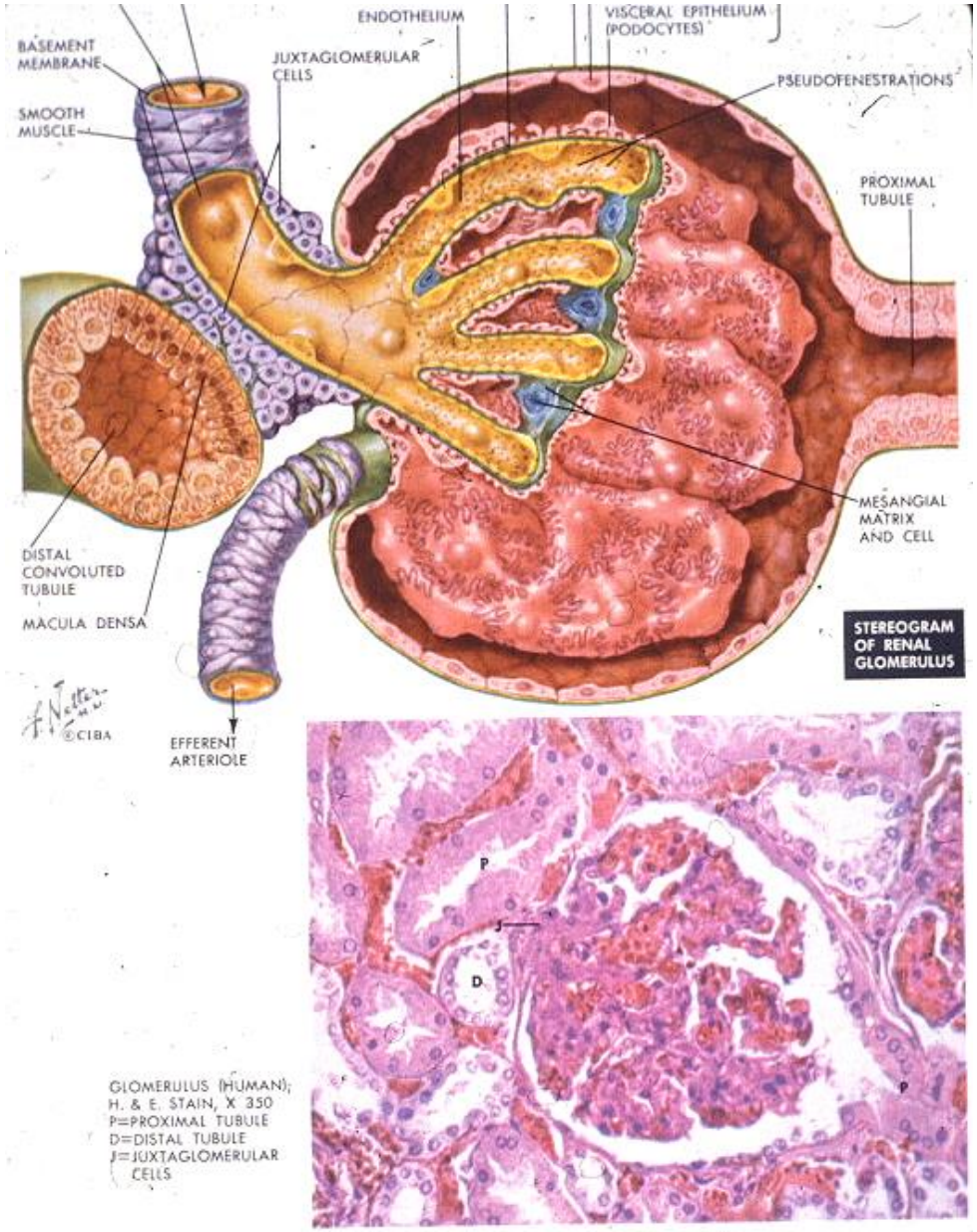
Basic Anatomy



Renal Microanatomy

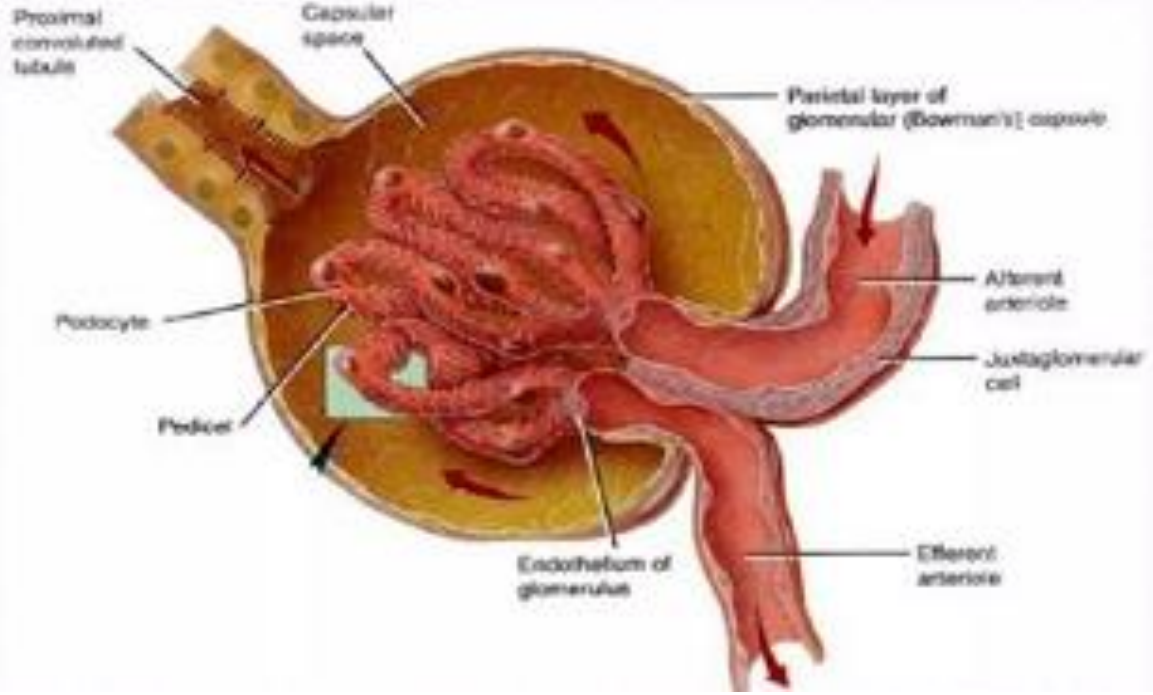


Glomerular Microanatomy

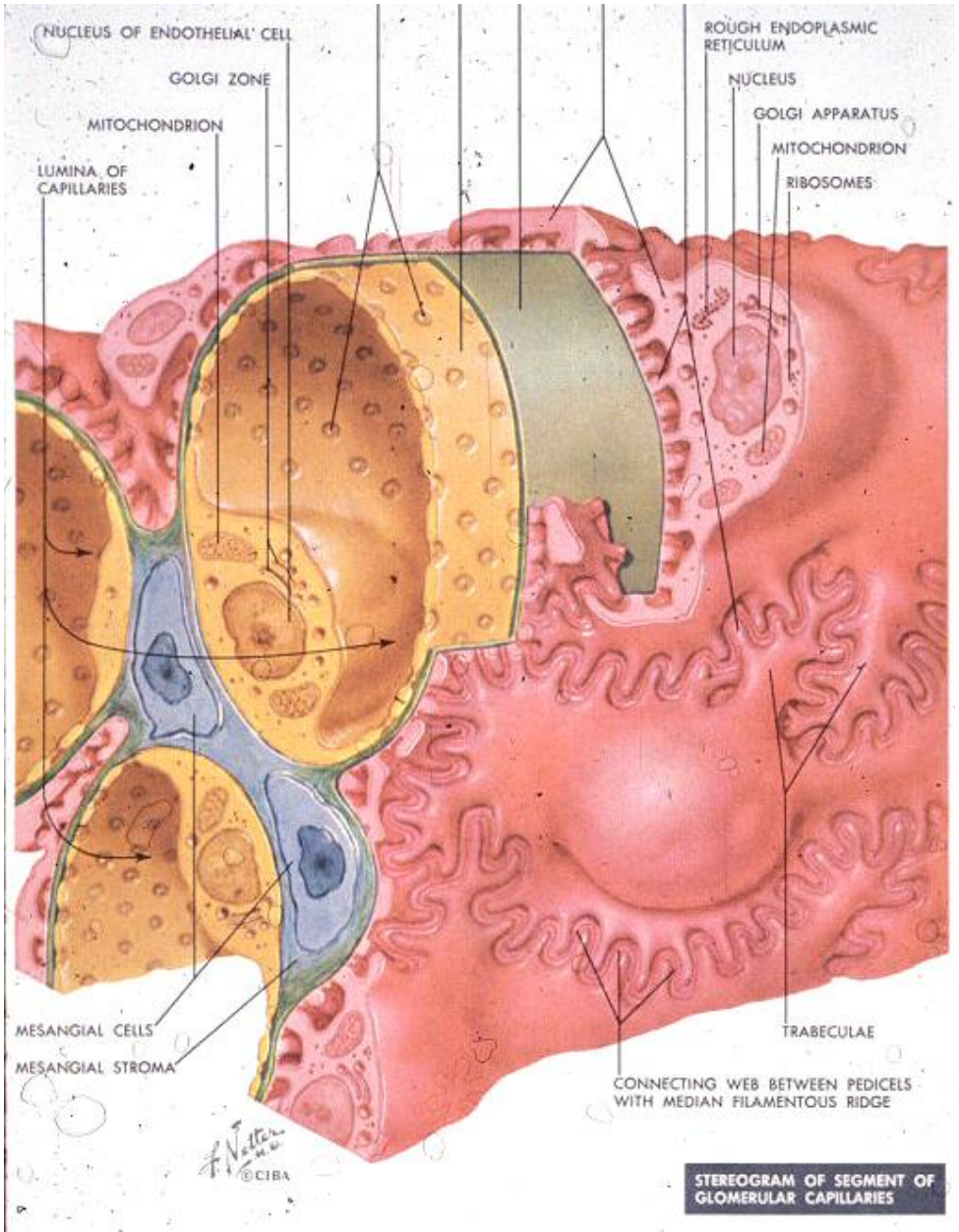


Glomeruli

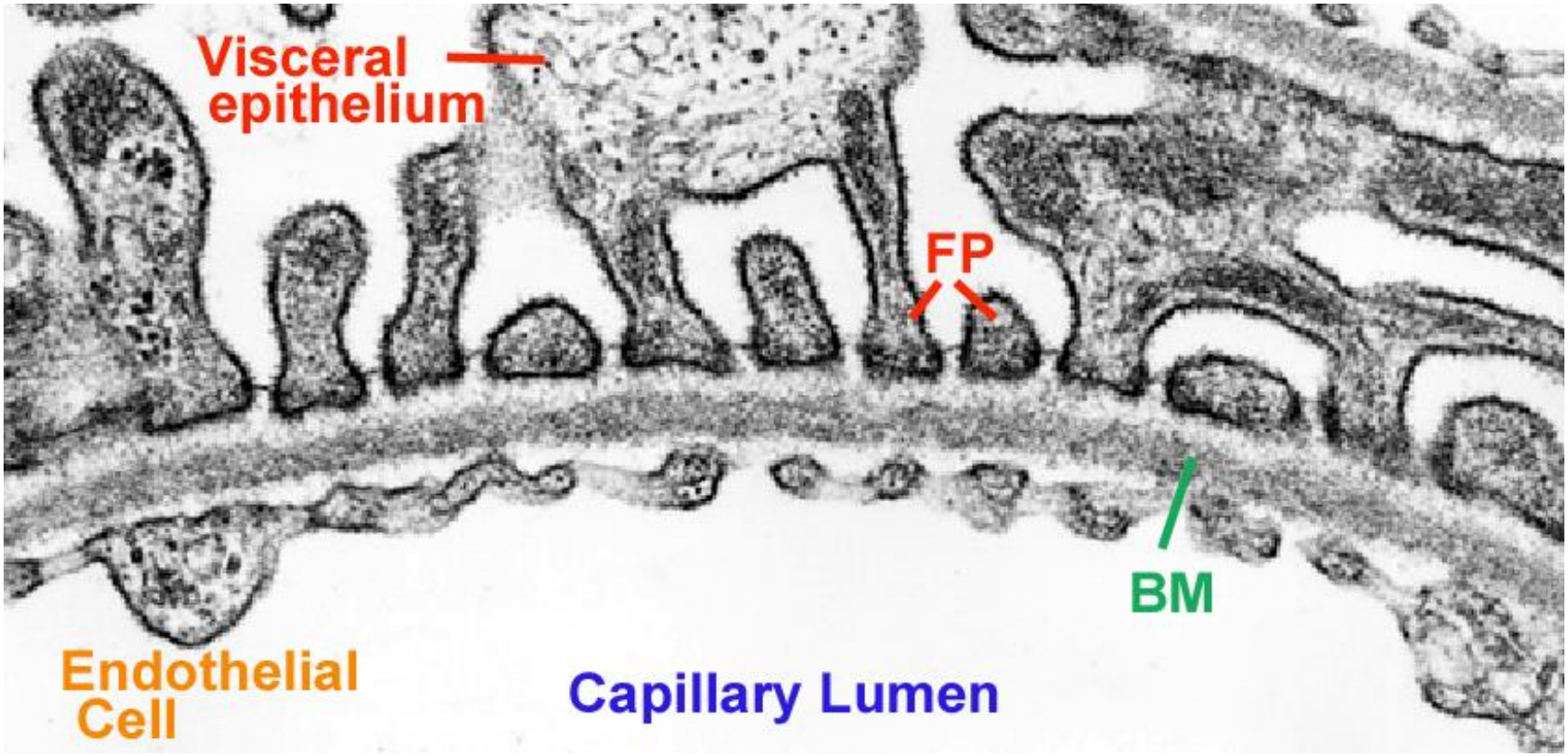
- Glomeruli – The filters of the kidneys which filter the blood and make urine.



The Business End of the Kidney



The Details


Visceral epithelium
FP
BM
Endothelial Cell
Capillary Lumen

Renal Functions

- The Kidney does lots of stuff
  - RBC production
    - Erythropoietin
  - Calcium metabolism
    - By means of phosphate elimination.
  - Acid-Base balance.
  - Na^+ and K^+ balance
  - Makes urine

Histological zones

- *diffuse,* involving all glomeruli;
- *focal,* involving only a proportion of the glomeruli;
- *segmental,* affecting a part of each glomerulus;

PRIMARY GLOMERULAR DIASEASES

- **Acute proliferative glomerulonephritis**
  - **Post-infectious**
  - **Other**
- **Rapidly progressive (crescentic) glomerulonephritis**
- **Membranous glomerulopathy**
- **Minimal-change disease**
- **Focal segmental glomerulosclerosis**
- **Membranoproliferative glomerulonephritis**
- **IgA nephropathy**
- **Chronic glomerulonephritis**

SECONDARY GLOMERULAR DIASEASES

- **Systemic lupus erythematosus**
- **Diabetes mellitus**
- **Amyloidosis**
- **Goodpasture syndrome**
- **Microscopic polyarteritis/polyangiitis**
- **Wegener granulomatosis**
- **Henoch-Schönlein purpura**
- **Bacterial endocarditis**

Common

- Immunological injury
  - Glomerulus
  - Interstitial tissue
- Vascular injury or compromise
  - Diabetes
  - Hypertension
- Infections
  - Upper urinary tract
  - Lower urinary tract

Nephritic Syndrome

- Inflammation of the glomeruli
- Glomerular diseases presenting with a nephritic syndrome are characterized by inflammation in the glomeruli
- Main features of nephritic syndrome include

  Hematuria, Proteinuria, Azotemia,

  Hypertension

Types of glomerulonephritis

- Acute glomerulonephritis

  - begins suddenly.

    It occurs after 5-21 days of streptococcal Infection.

- Chronic glomerulonephritis

  -develops gradually over several years. It occurs after the acute phase.

INCIDENCE

- More common in male than females.
- Most common in preschool and early school age children with a peak age of onset of 6-7 years.
- Rare in children under two years of age.
- On average responsible for 2 to 4% of pediatric admissions in India.
- Accounts for about 90% of renal diseases in childhood
- Varies with the prevalence of nephritogenic strains of streptococci and the likelihood of cross – infection.

Etiology & risk factor

Streptococcal infection of the throat (strep throat) or skin (impetigo)

Hereditary diseases

Immune diseases, such as SLE

diabetes

High blood pressure

Vasculitis (inflammation of the blood vessels)

Viruses (HIV, hepatitis B virus, and hepatitis C virus)

Endocarditis (infection of the valves of the heart)

Pathologic responses of the glomerulus Injury

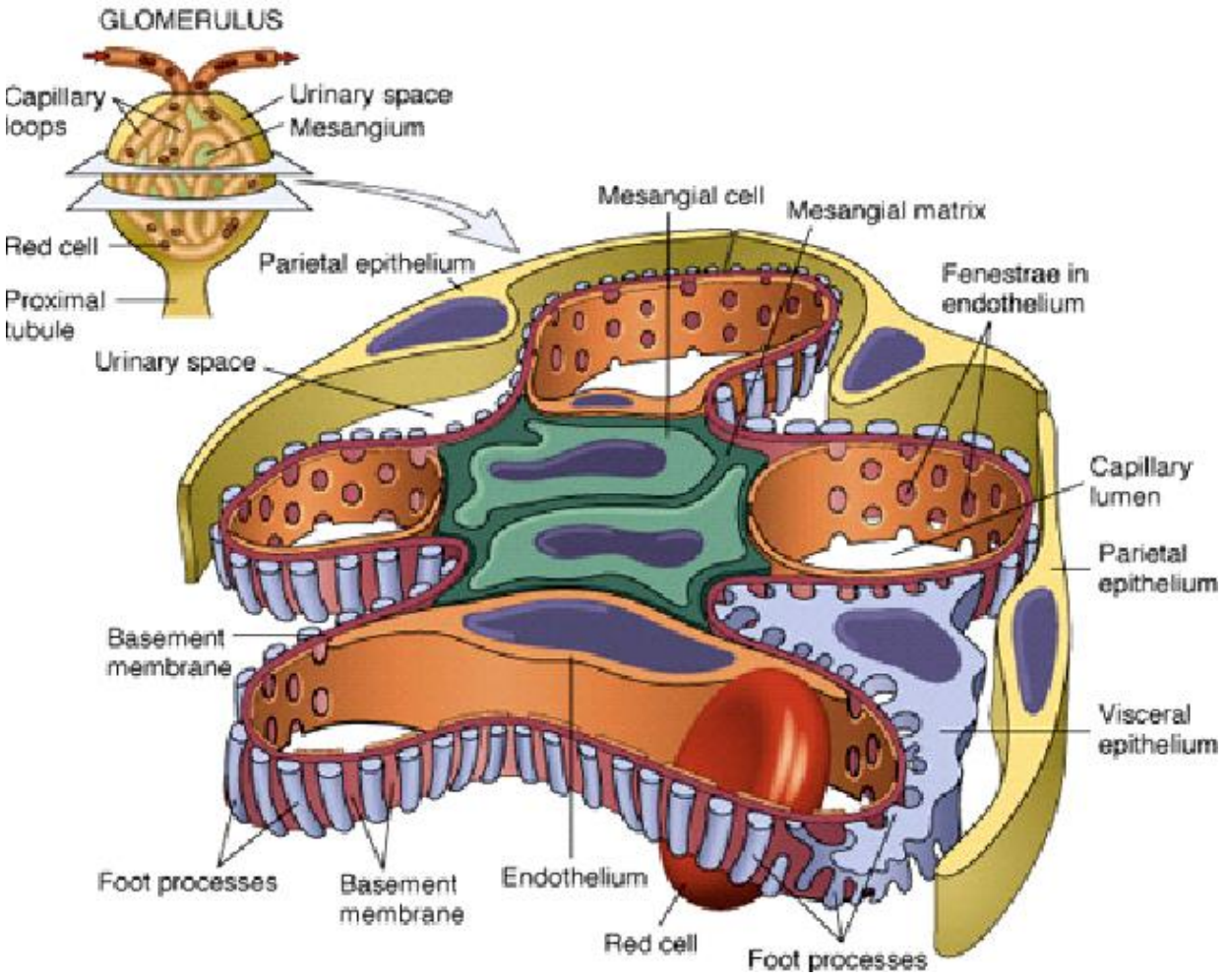
1.HYpercellularity

- Mesangial/End. proliferation
- Leukocyte infiltration

2.BM thickening

3. Hyalinosis

4. Sclerosis



Antibody associated injury

- Circulating Ab reaches glomerulus &binds with in situ Ag (Ag+Ab-locally formed) **
- Circulating Ag+Ab complex deposited in glomeruli

↓

Inflammatory reaction

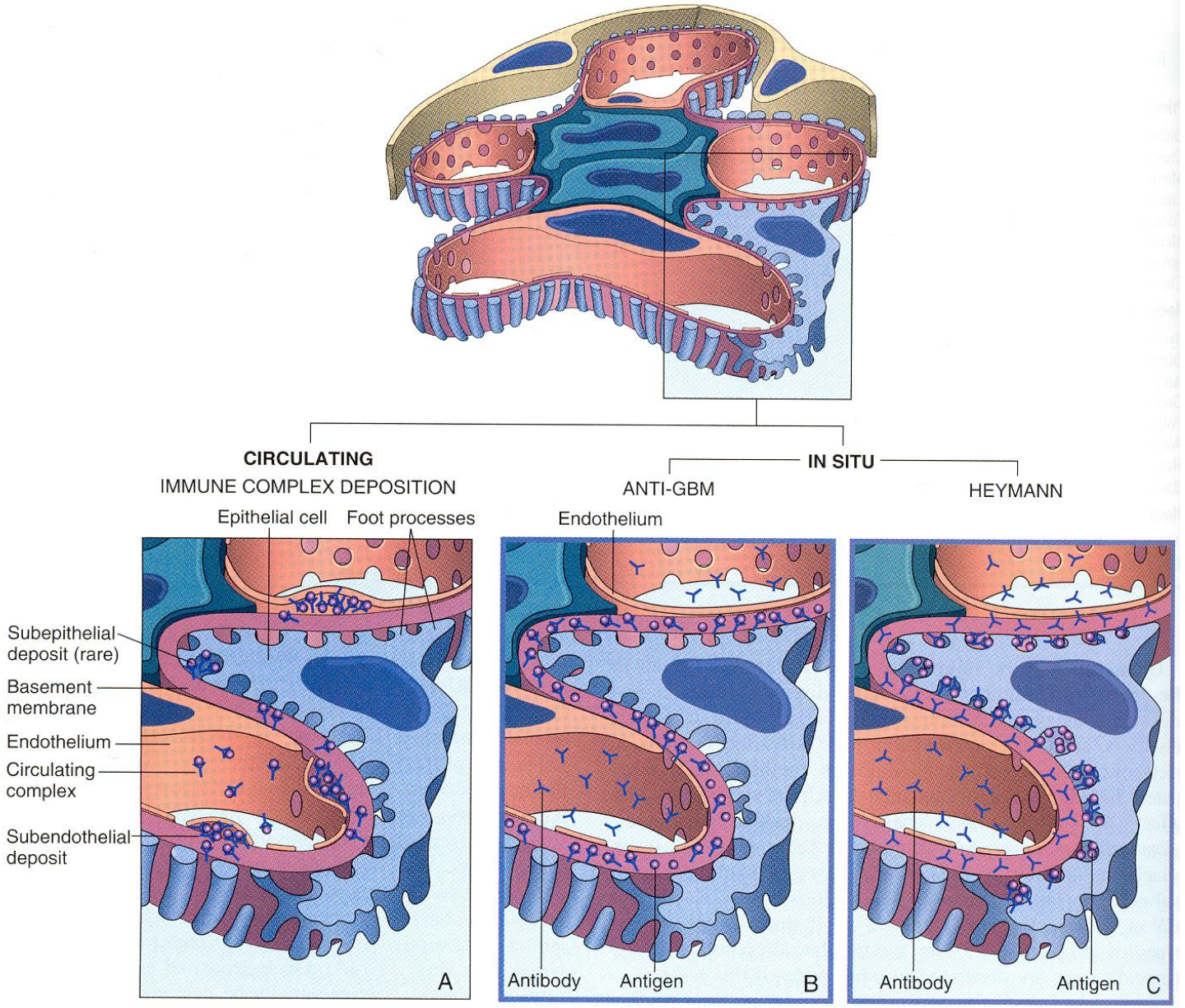
↓

Increased no. WBC, Endothelial cells,

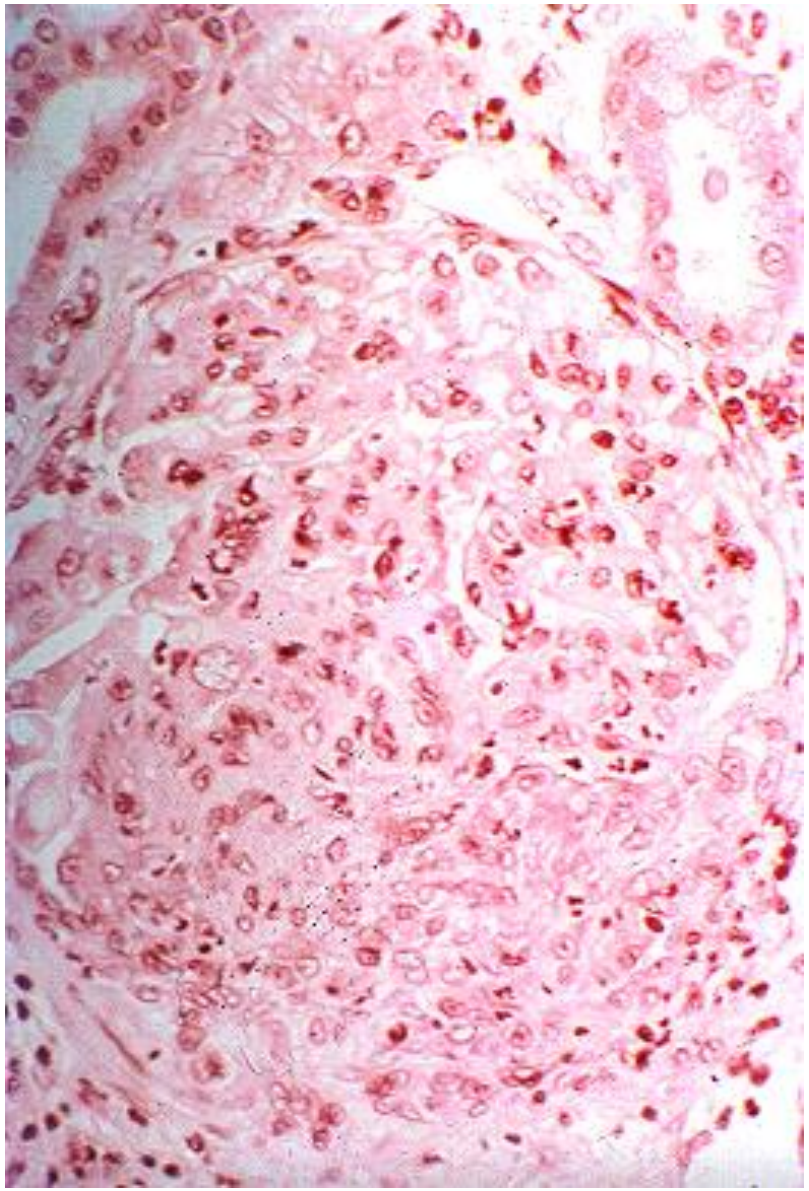
Mesangial cells

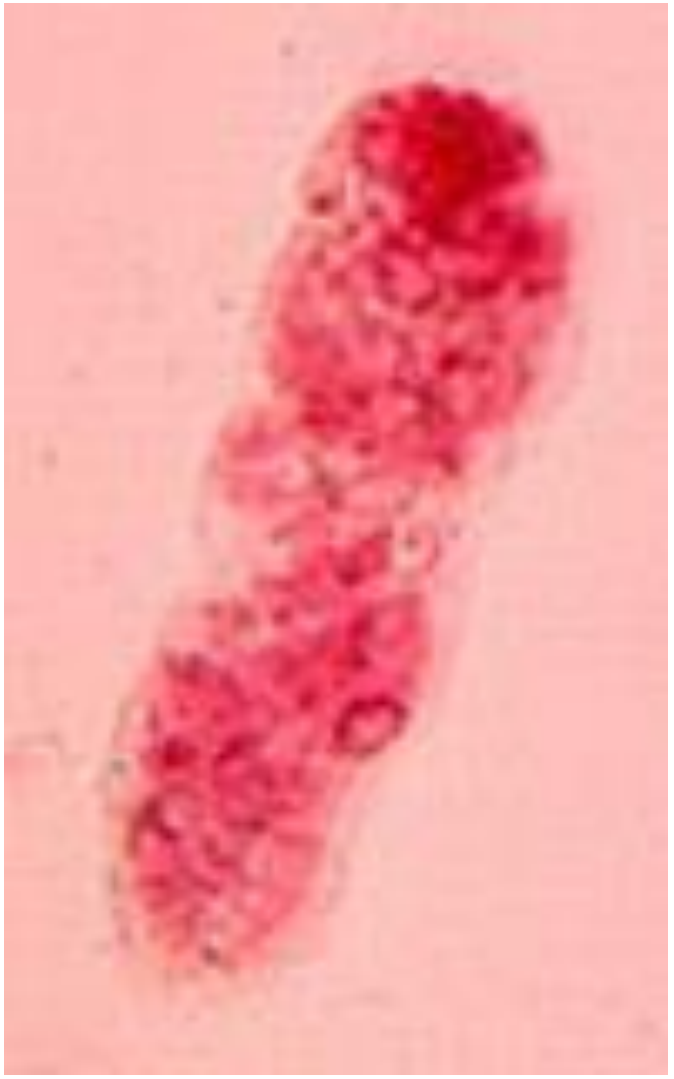
Pathogenesis of Glomerular injury

Immunologic Injury



Acute Glomerulonephritis





Pathophysiology

Due to any etiological factor

↓

Release of Ag substance into the circulation

↓

Formation of Ab

↓

formation of Ag and Ab complex in the glomerulus

↓

Inflammatory response

↓

proliferation of epithelial cells lining the glomerulus

↓

Leukocytes infiltration of the glomerulus

↓

Thickening of the glomerular filtration membrane

Cont…

↓

scarring and loss of glomerular filtration membrane

↓

decrease GFR and glomerulus plasma flow

↓

Retention of sodium and water

↓

Edema and hypertension

INVESTIGATION

- **History of illness** and physical examination help in clinical diagnosis.
- The confirmation of diagnosis is done by the following:

Urine examination:

It shows increased specific gravity, smoke dirty brown colour urine with reduced total amount in 24 hrs. Mild to moderate or severe albuminuria is detected. Microscopic examination reveals presence of red cells, WBCs, pus cells, epithelial cells and granular cast. Proteinuria (3+ to 4+)

Blood examination:

- Blood examination demonstrates increased level of urea, creatine, ESR, ASO titer and anti – DNAase 'B'. There is decreased level of Hb%, serum complement and albumin in blood. Hyponatremia and hyperkalemia may occur in persistent oliguria.

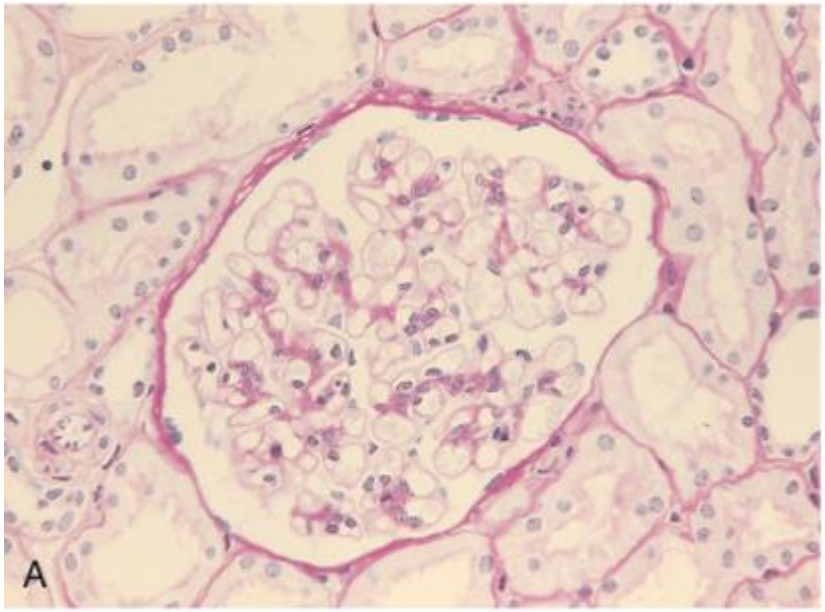
Throat swab culture:

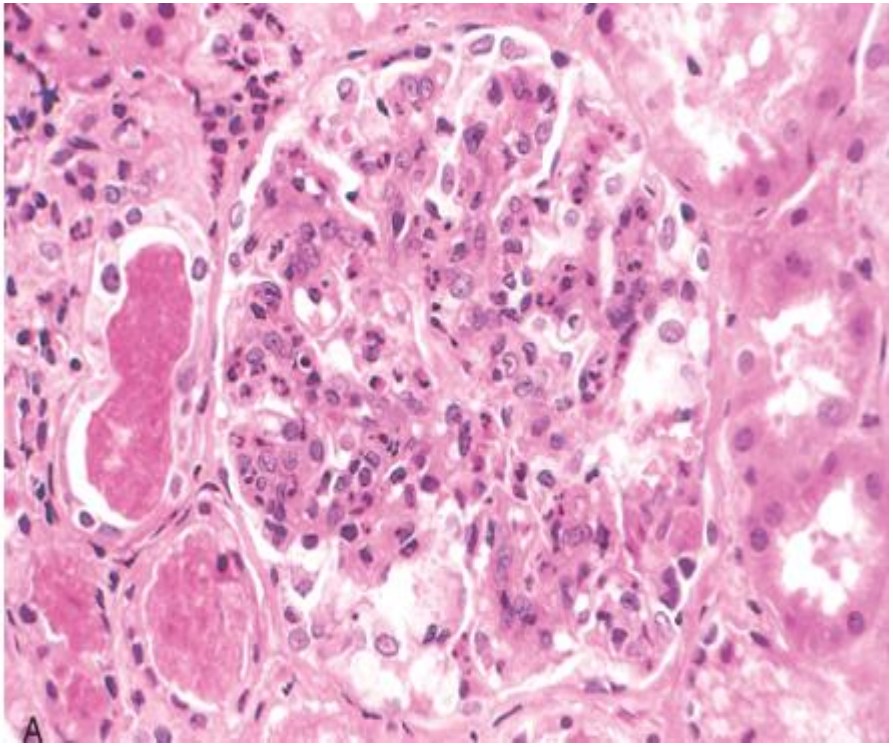
- Throat swab culture may show presence of beta – hemolyticus streptococcus in some children.

Chest X-ray:

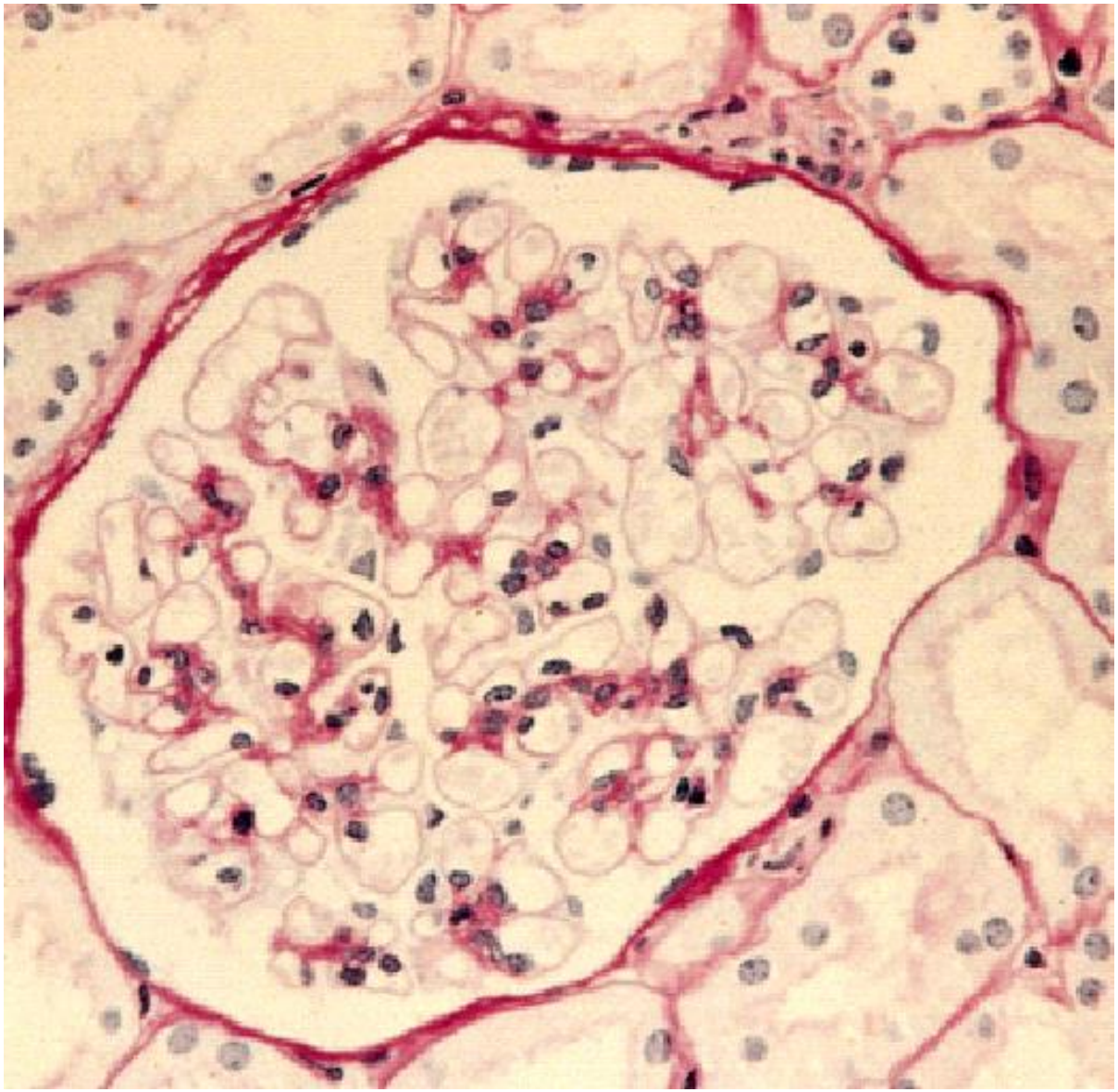
- It may show pulmonary congestion

Normal Gl. Vs AGN



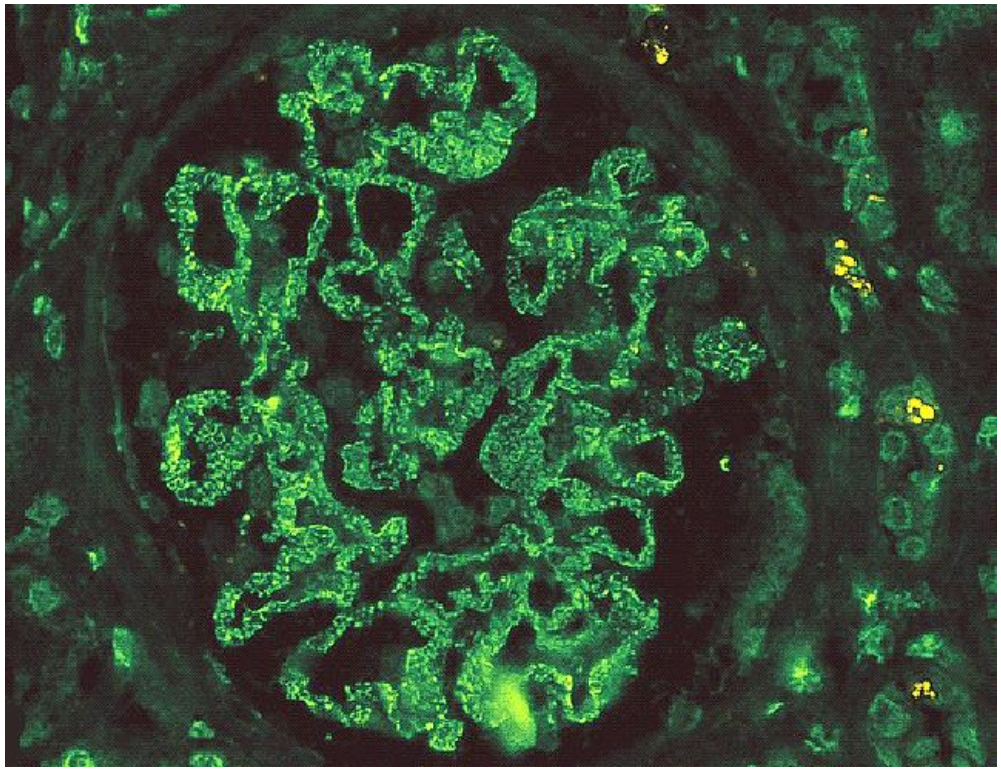


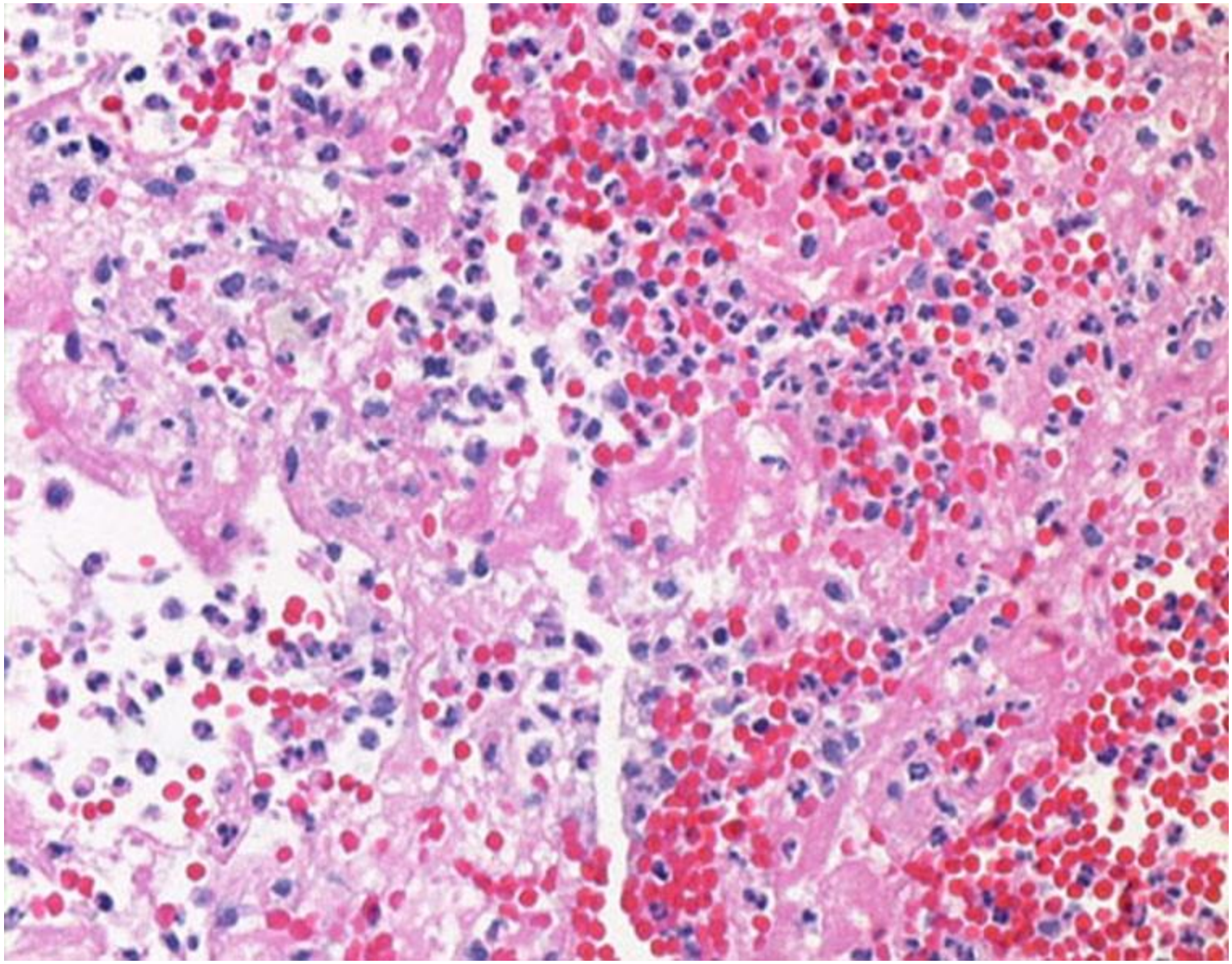
Minimal Change Glomerulonephritis

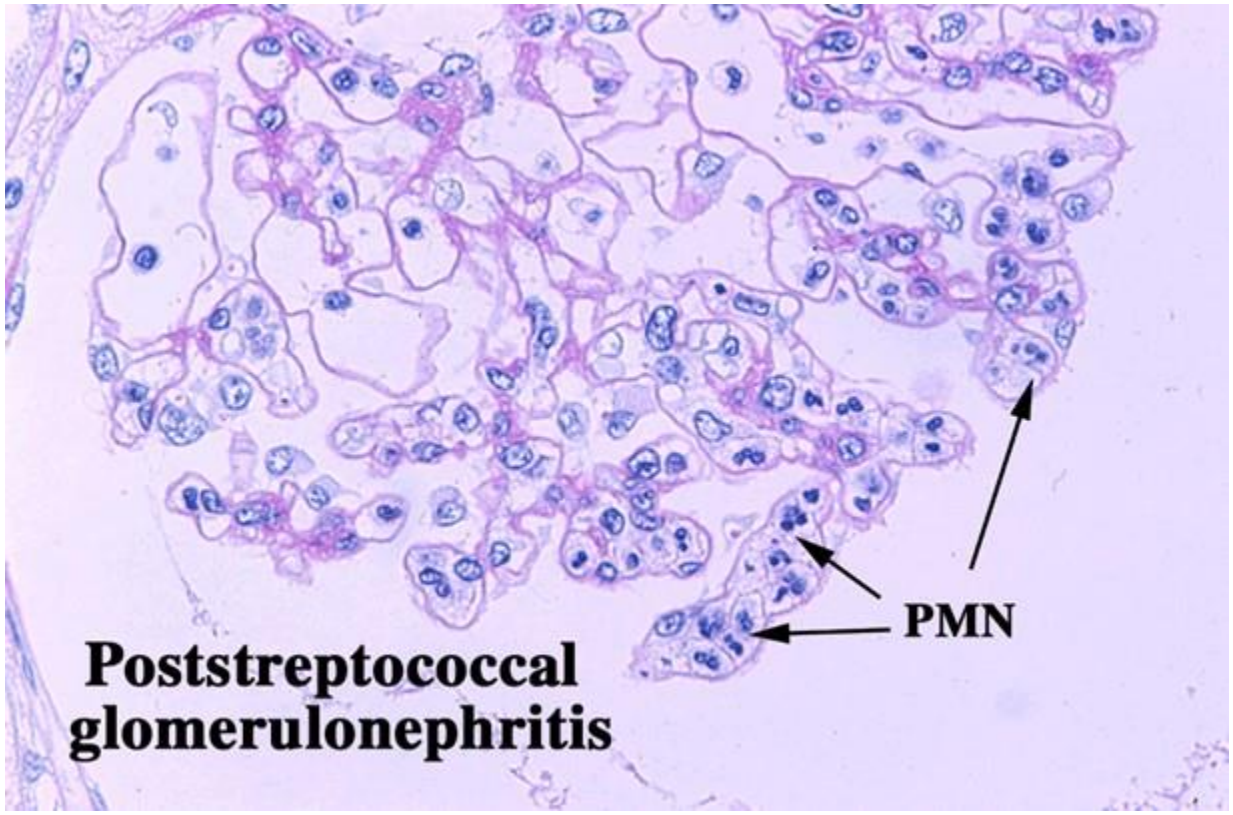


Acute Glomerulonephritis

- Anti-human IgG labeled with fluorescence.
- Identifies the immune complexes
- Granular pattern
  - Irregular clumps
  - Fix C’
  - Membrane damage

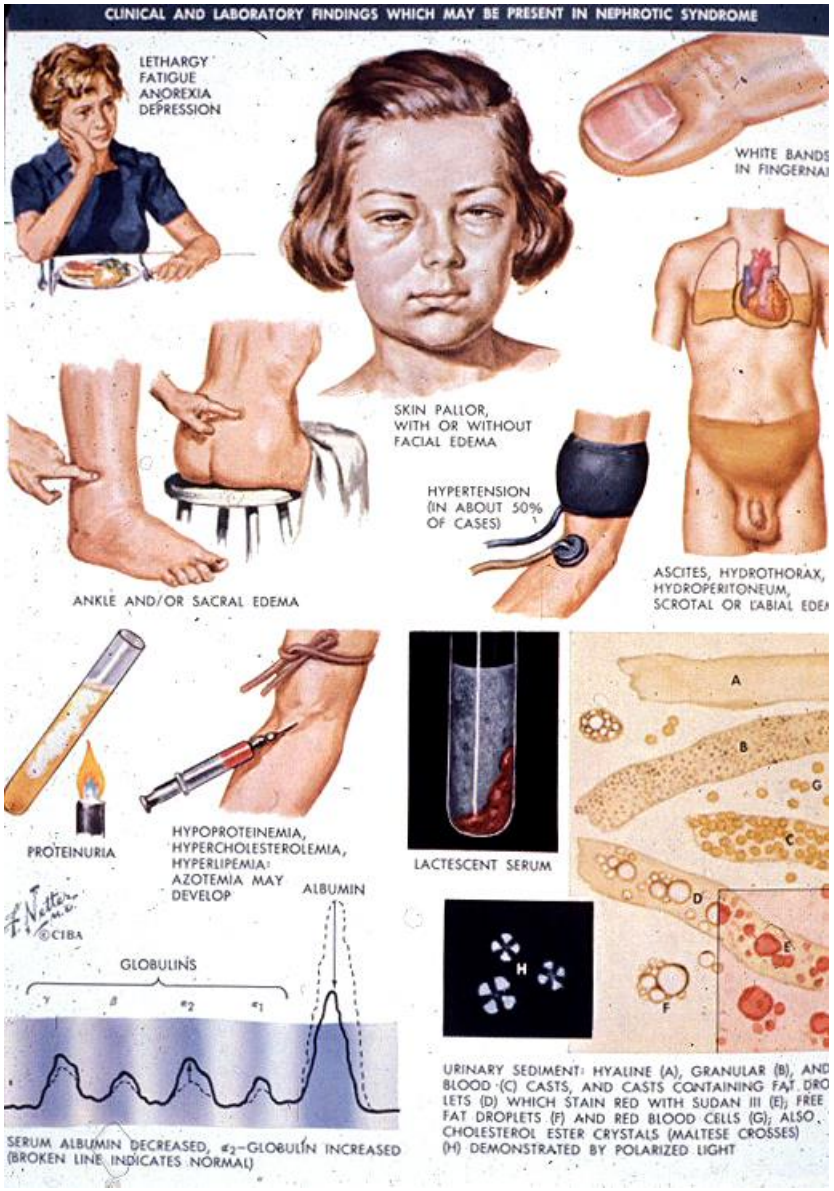





PMN
Poststreptococcal
glomerulonephritis

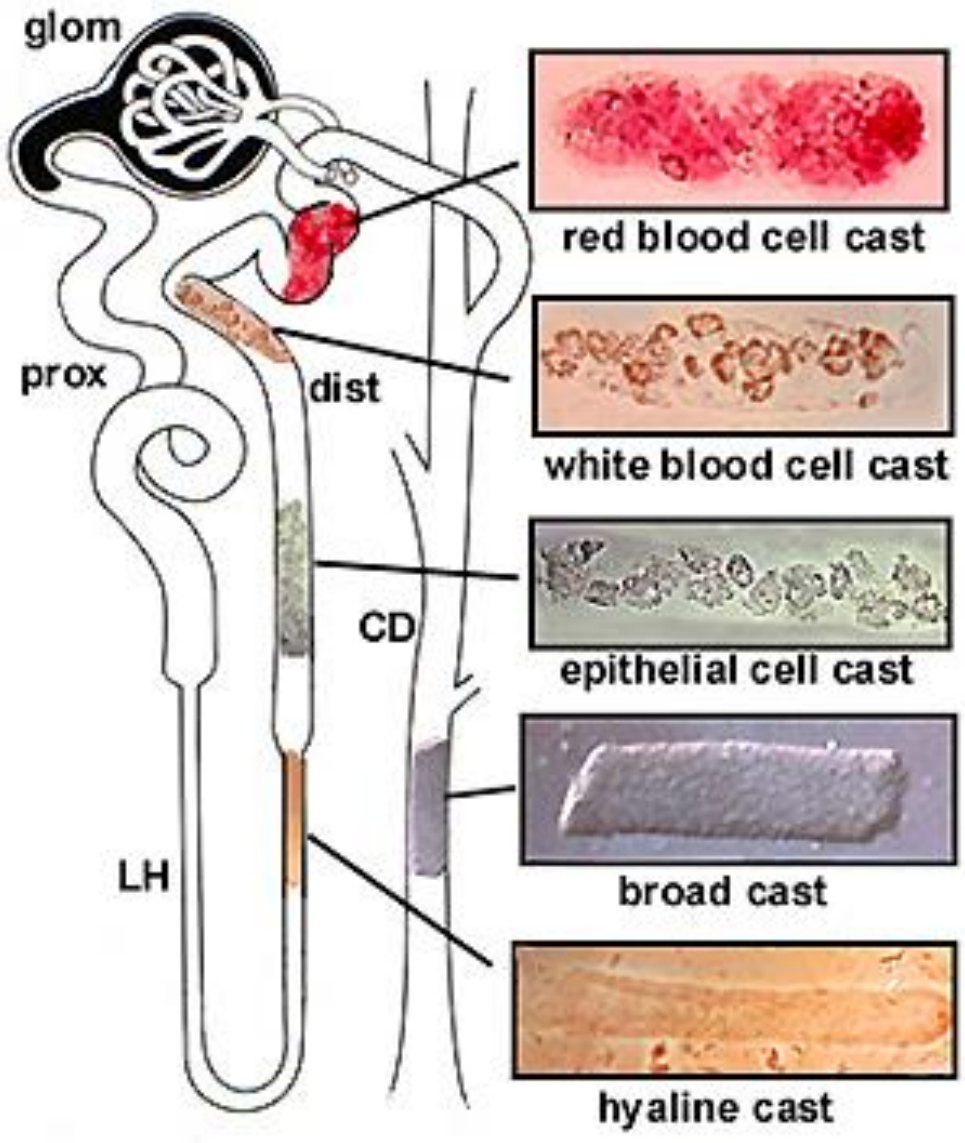
Nephrotic Syndrome

- **<u>Chronic</u>** injury of the glomerulus.
  - Many causes
- Basic clinical pattern
  - <u>Proteinuria</u> (>3.5 gm)
  - High serum lipids
  - Lipiduria
  - Low serum albumen
  - Edema



Urinary Casts

- Material cleared or shed by a sick glomerulus.
- Congeals within the
  - Convoluted tubules or
  - Collecting ducts
- Creates a ‘cast’ of the interior of the duct it formed in.
- Is Cleared in urine.
- Observed microscopically



Glomerulonephritis

Pharmacology

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Patuakhali Medical College, Patuakhali

Diuretics

Loop diuretics

Aldosterone antagonist

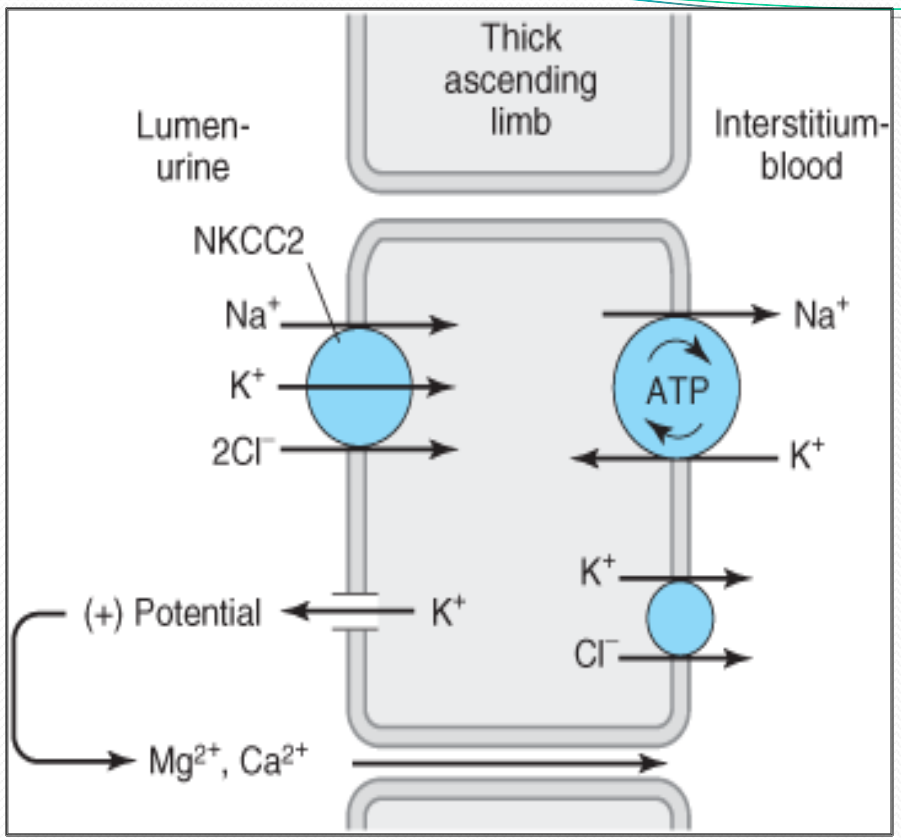
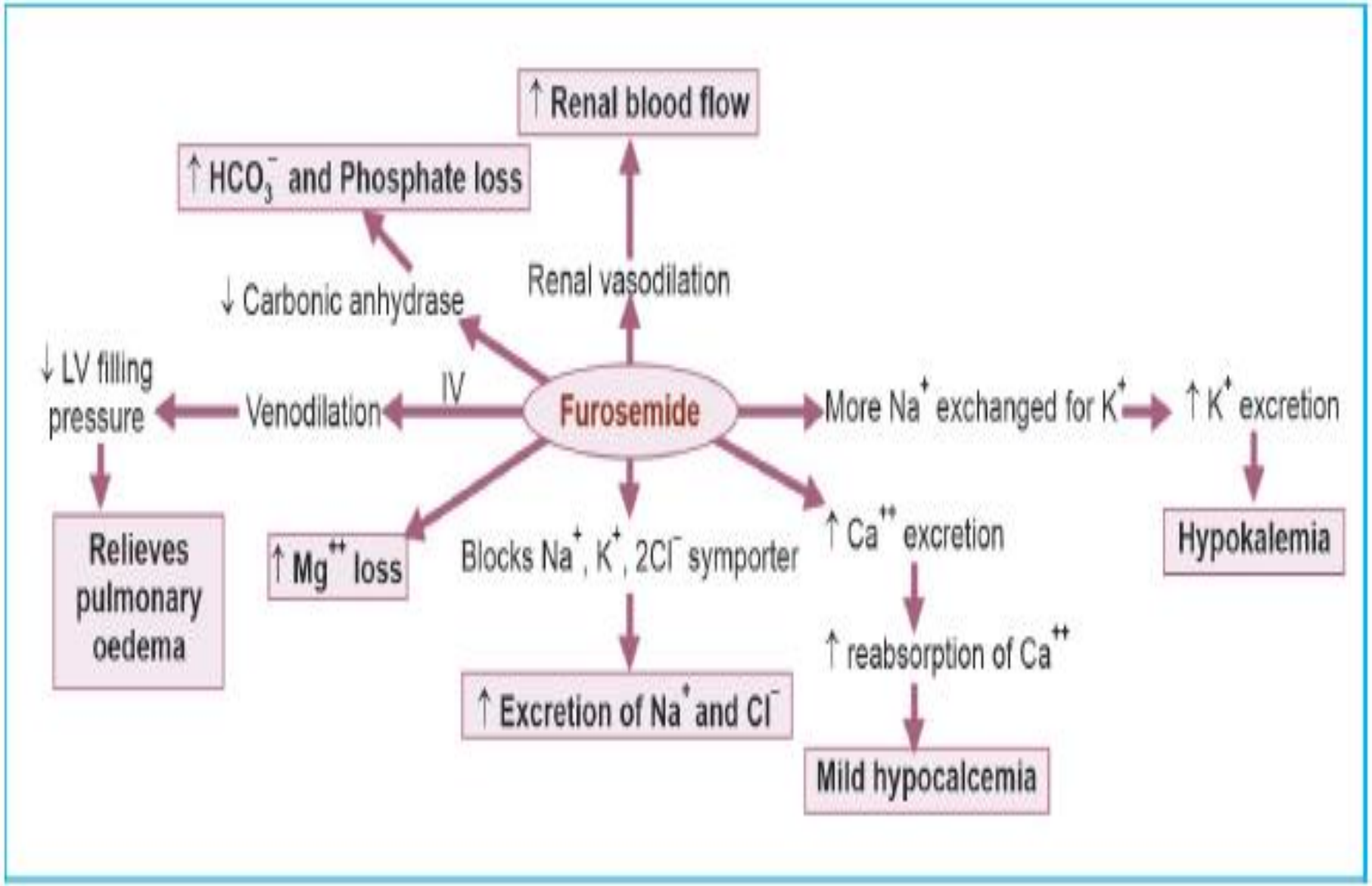


Figure: Ion transport pathways across the luminal and basolateral membranes of the thick ascending limb cell. The lumen positive electrical potential created by K + back diffusion drives divalent (and monovalent) cation reabsorption via the paracellular pathway. NKCC2 is the primary transporter in the luminal membrane.

Flowchart 22.1: Actions of frusemide (loop diuretics)



A/E of loop diuretics:

- Hypovolemia ,hyponatraemia,dehydration,hypotension
- Hypokalemia
- Metabolic alkalosis (due to excess H^+ loss)
- Hyperuricaemia (hypovolemia-associate enhancement of uric acid reabsorption in the PCT).
- Hypomagnesemia
- Hypersensitivity

Adverse effect of Aldosterone antagonist

- Hyperkalemia
- Hyperchloremic metabolic acidosis
- Endocrine abnormalities(estrogenic effect)-

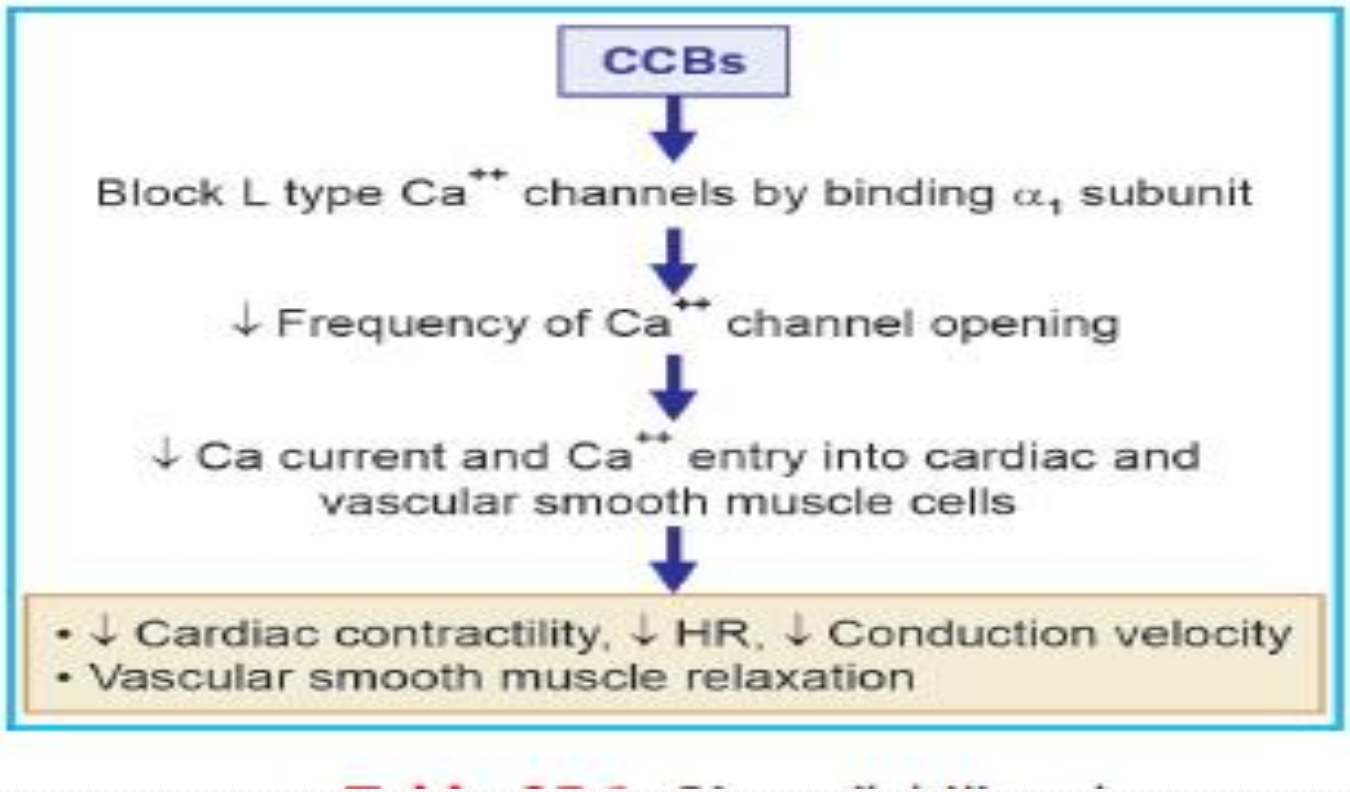
- **Glomerular disease is** often associated with hypertension.

- Control of hypertension is very important because of its close relationship with further decline in renal function and exacerbated cardiovascular risk.

Calcium channel blockers

Nifedipine

Works by blocking the voltage dependent L-type calcium channels, thereby inhibiting the initial influx of calcium.


CCBs
Block L type Ca^{++} channels by binding α_1 subunit
↓ Frequency of Ca^{++} channel opening
↓ Ca current and Ca^{++} entry into cardiac and vascular smooth muscle cells
• ↓ Cardiac contractility, ↓ HR, ↓ Conduction velocity
• Vascular smooth muscle relaxation

Adverse effects:

- Throbbing Headache
- Tachycardia, Palpitation
- Ankle oedema

Angiotensin-converting enzyme inhibitors

- Captopril
- Lisinopril
- Perindopril

Angiotensin receptor blockers

- Losartan
- Irbesartan
- Valsartan

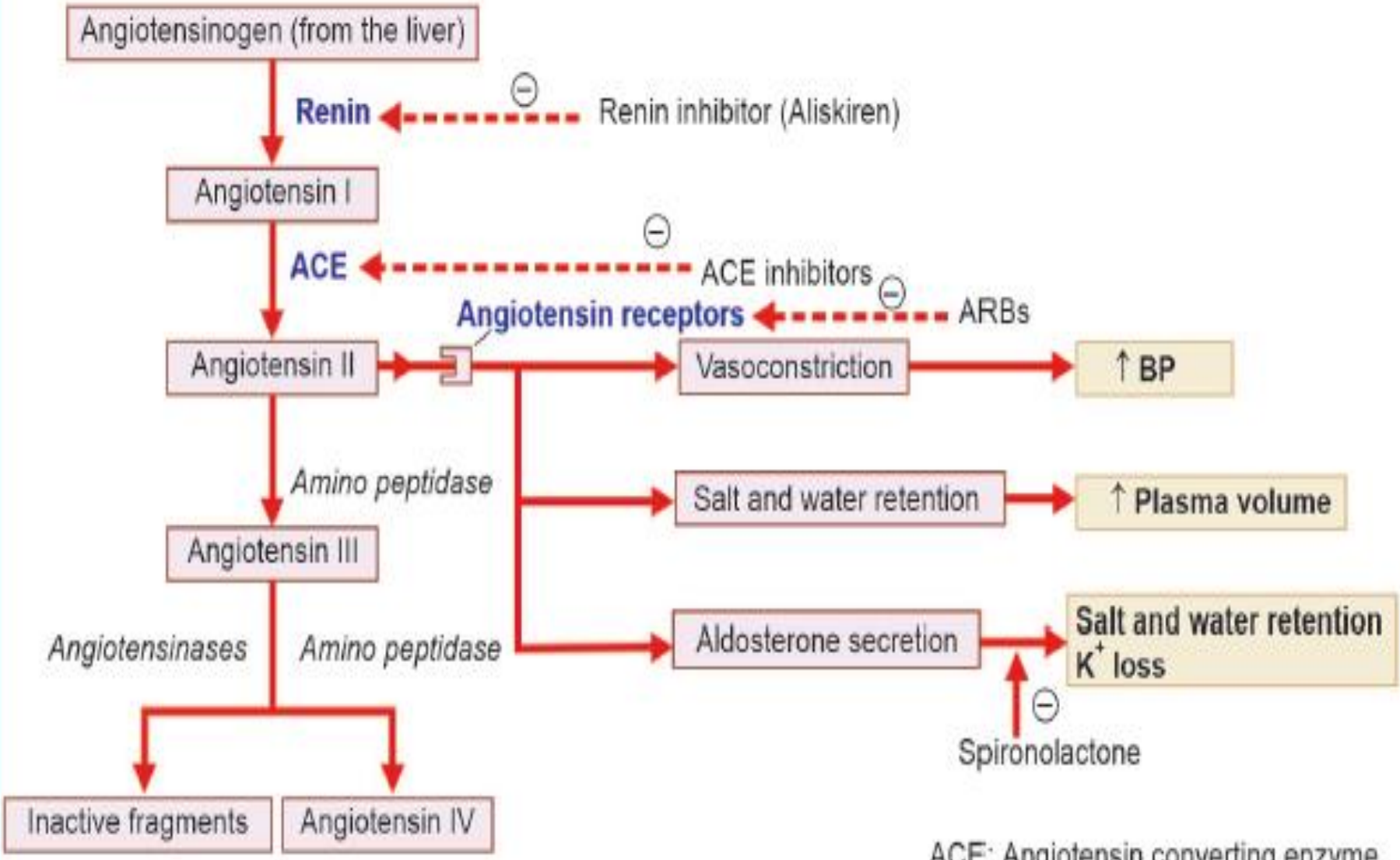


Fig. 24.1: Synthesis and metabolism of angiotensins and drugs acting at various sites

Side-effects of of ACE Inhibitors

- Hypotension

(1st dose can cause hypotension specially who is also taking diuretics)

- Persistent dry cough . (No significant dry cough in ACE receptor blocker)

- Angioedema…rapid swelling (edema) of the skin, mucosa and submucosal tissues…. another cause of this edema allergy.

Anti-inflamatory and Immunosuppressive agent

Corticosteroids- prednisolone is used to suppress inflammation, allergy and immune responses.

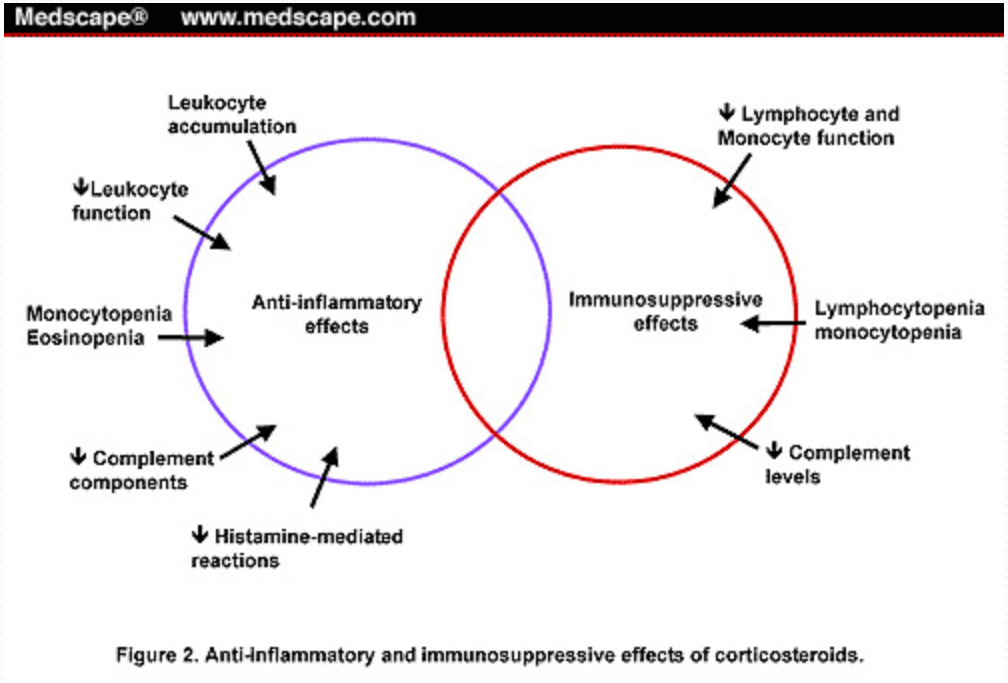


Figure 2. Anti-inflammatory and immunosuppressive effects of corticosteroids.

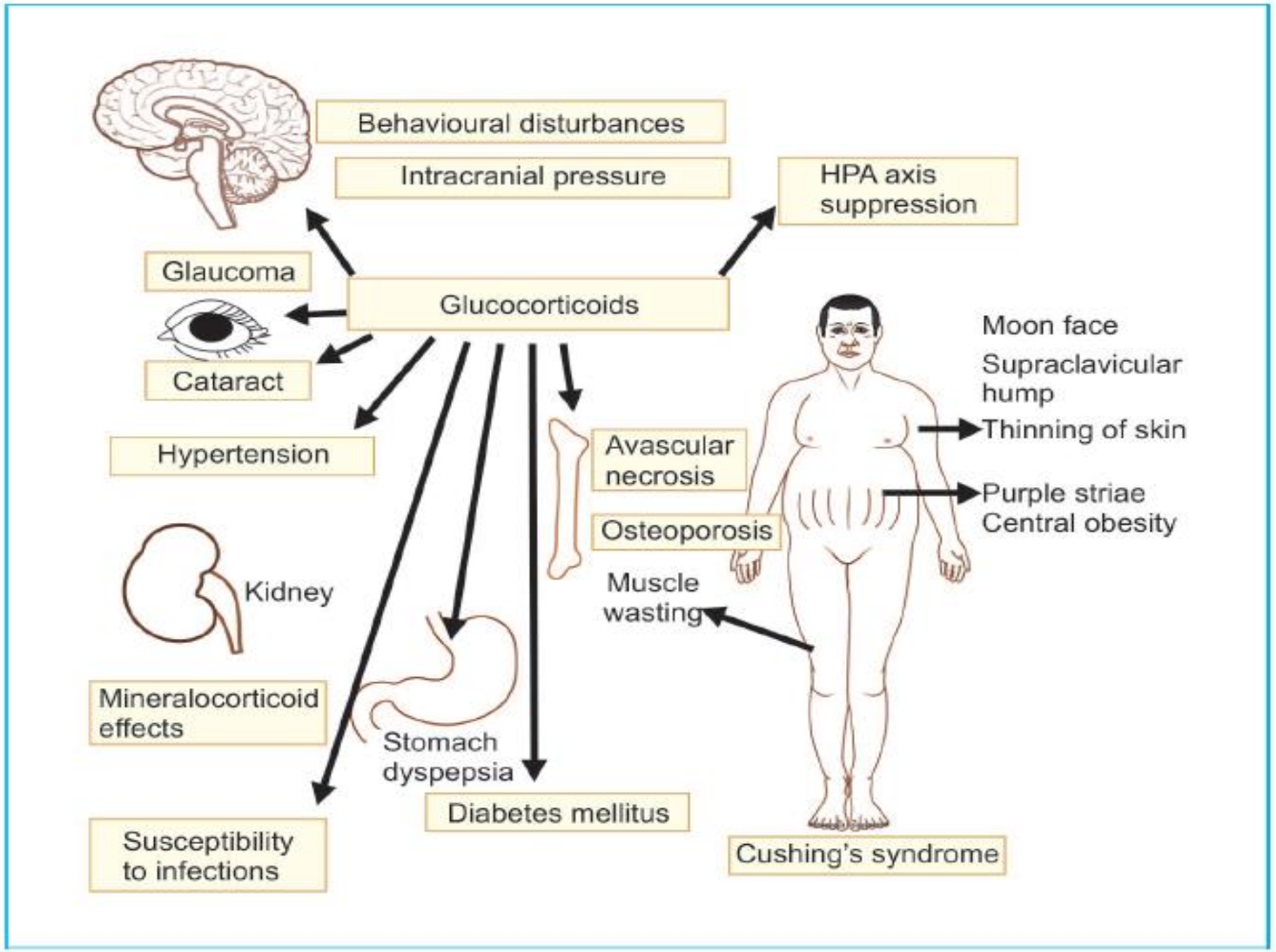
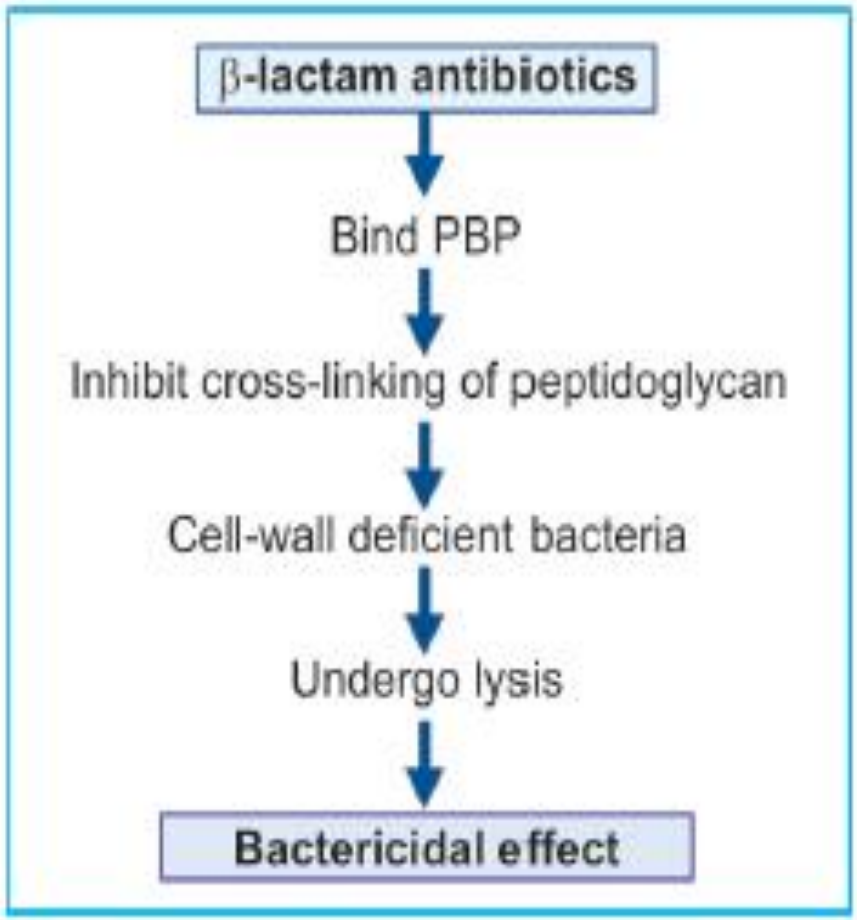


Fig. 38.5: Adverse effects of glucocorticoids

- Benzylpenicillin /penicillinG
- Phenoxymathylpenicillin /penicillinV

- Benzylpenicillin /penicillinG-Acid labile-can’t be given orally always given parentally. Long duration of action.
- Phenoxymethyl penicillin(**Penicillin V)-** is acid stable and can be given orally. Short duration of action.
- Narrow spectrum penicillin.
- **bactericidal and act on actively multiplying** bacteria.
- Bactericidal activity of penicillin is **more against Gram positive**
- They are highly safe with a high therapeutic index,


β-lactam antibiotics
Bind PBP
Inhibit cross-linking of peptidoglycan
Cell-wall deficient bacteria
Undergo lysis
Bactericidal effect

Adverse effect

- The main hazard is allergic reaction- skin rashes,urticaria, pruritus, fever, bronchospasm,

serum sickness and rarely, exfoliative dermatitis and anaphylaxis.

Glomerulonephritis

Medicine

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GLOMERULONEPHRITIS

- **While glomerulonephritis literally means 'inflammation of glomeruli', the term is often used more broadly to describe all types of glomerular disease,even though some of these (e.g. minimal change nephropathy) are not associated with inflammation.**
- **There are many causes of glomerular damage, including immunological injury, inherited diseases such as Alport syndrome , metabolic diseases such as diabetes mellitus and deposition of abnormal proteins such as amyloid in the glomeruli.**
- **Most types of glomerulonephritis are immunologically mediated and several respond to immunosuppressive drugs. Deposition of antibody occurs in many types of glomerulonephritis and testing for circulating or glomerulardeposition of antibodies may aid diagnosis.**
- **In small-vessel vasculitis, no glomerular antibody deposition is observed (pauci-immune), but the antibodies may be indirectly pathogenic by activating neutrophils to promote endothelial injury.**
- **Glomerulonephritis is generally classified in terms of the histopathological appearances,**

Mechanism

Nephritic Mechanism

- Inflammation
- Reactive cell proliferation
- Breaks in GBM
- Crescent formation

Clinical features

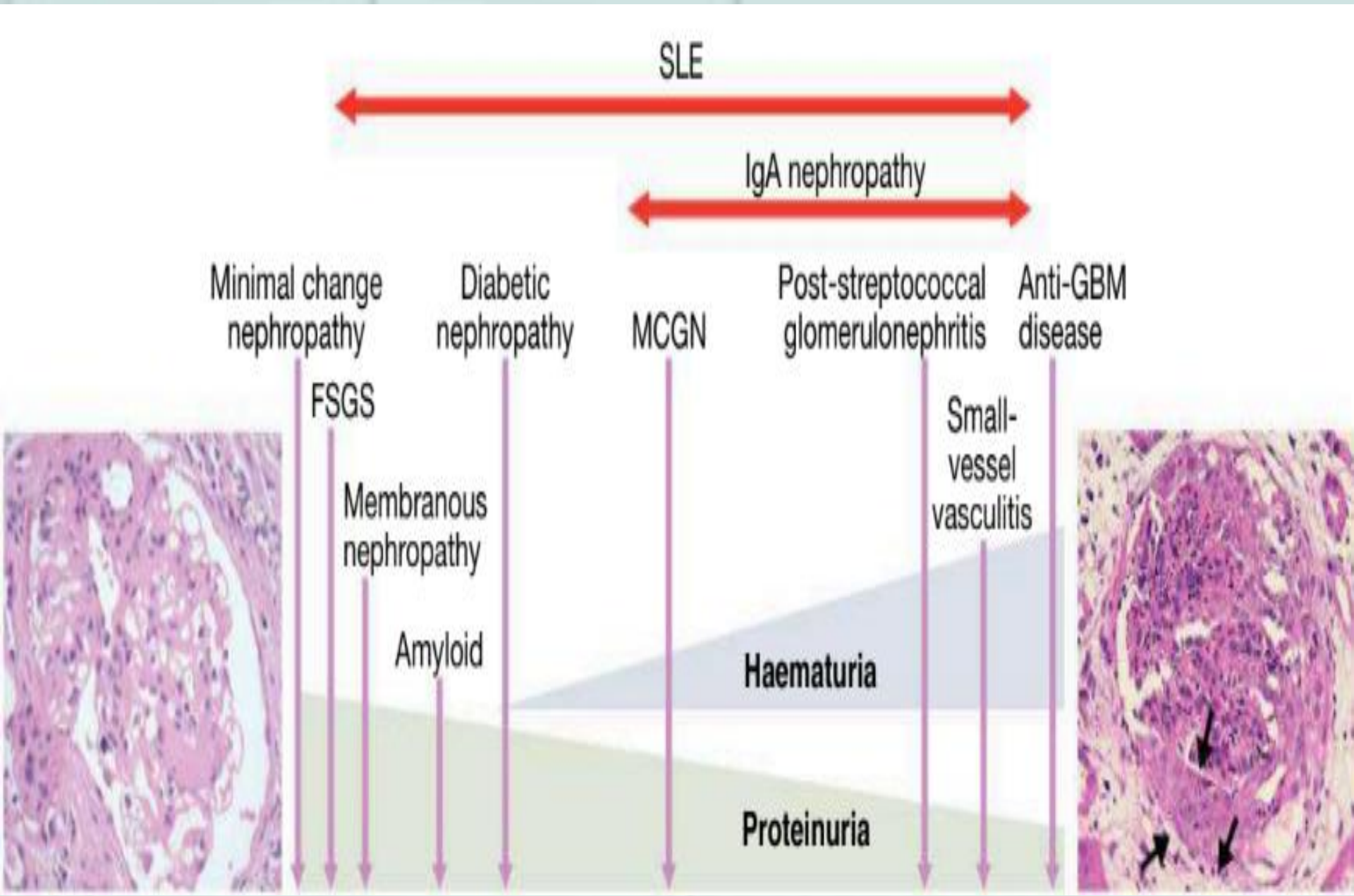
- Haematuria (red or brown urine)
- Oedema and generalised fluid retention
- Hypertension
- Oliguria
- Reduced renal function

Nephrotic Mechanism

- Injury to podocytes
- Changed architecture
  - Scarring
  - Deposition of matrix or other elements

Clinical features

- Overt proteinuria: usually > 3.5 g/24 hrs
(urine may be frothy)
- Hypoalbuminaemia < 30 g/L
- Oedema and generalised fluid retention
- Possible intravascular volume depletion with hypotension, or intravascular expansion with hypertension


SLE
IgA nephropathy
Minimal change nephropathy
FSGS
Membranous nephropathy
Amyloid
Diabetic nephropathy
MCGN
Post-streptococcal glomerulonephritis
Small-vessel vasculitis
Anti-GBM disease
Haematuria
Proteinuria

INVESTIGATION OF NEPHRITIC SYNDROME

Cause	Investigations
Rapidly progressive glomerulonephritis (RPGN)	
• Post-infectious glomerulonephritis	ASOT, C3, C4
• Anti-GBM disease	Anti-GBM antibody
• Small-vessel vasculitis	p-ANCA, c-ANCA
• Lupus nephritis	ANA, dsDNA, C3, C4
Mild glomerulonephritic presentation	
• IgA nephropathy	Serum IgA (polyclonal rise in 50% of patients)
• Mesangioproliferative glomerulonephritis	C3, C4, hepatitis B, C + HIV serology, ANA, dsDNA, immunoglobulins, PPE
• Alport syndrome*	Genetic screening, hearing test

INVESTIGATION OF NEPHROTIC SYNDROME

Cause	Typical age group	Investigations
Minimal change disease	Children, young adults, occasionally seen in older patients	None specific
Primary focal segmental glomerulosclerosis	**Young adults**	**None specific**
Membranous nephropathy	**Middle-aged to older patients**	**Anti-phospholipase A2 receptor antibody (primary disease) Hepatitis B, C + HIV serology, ANA, dsDNA**
Amyloid	**Older patients**	**Immunoglobulins, PPE, Bence Jones protein, serum free light chains**
Diabetic nephropathy	**Any age, but rarely <10 years from diagnosis of type 1 diabetes**	**Glucose, glycosylated haemoglobin**

Light microscopy

- Focal : affecting some but not all glomeruli
- Diffuse : affecting >50% of glomeruli
- Segmental : affecting a portion of a glomerulus
- Global : affecting all of the glomerulus
- Necrotising : severe injury leading to an area of necrosis, usually associated with vasculitis
- Crescentic : a crescent-shaped area of inflammatory cells responding to severe glomerular injury

Electron microscopy

- **Subendothelial immune deposits : found between the endothelial cell and the GBM – often found in nephritic presentations**
- **Intramembranous immune deposits: found within the GBM – found in the dense deposit variant of mesangiocapillary glomerulonephritis**
- **Subepithelial immune deposits: found between the epithelial cell and the GBM –often found in nephrotic presentations, including membranous presentation of lupus**

Presentation of diseases

- **Diseases typically presenting with nephrotic syndrome**
  - **Minimal change nephropathy**
  - **Focal segmental glomerulosclerosis**
  - **Membranous nephropathy**

- **Diseases typically presenting with mild nephritic Syndrome**
  - **IgA nephropathy**
  - **Henoch–Schönlein purpura**
  - **Mesangiocapillary glomerulonephritis**

- **Diseases typically presenting with rapidly progressive glomerulonephritis**
  - **Anti-glomerular basement membrane disease**
  - **Infection-related glomerulonephritis**

POOR PROGNOSTIC INDICATORS IN GLOMERULAR DISEASE

- Male sex
- Hypertension
- Persistent and severe proteinuria
- Elevated creatinine at time of presentation
- Rapid rate of decline in renal function
- Tubulo-interstitial fibrosis observed on renal biopsy

Glomerulonephritis

Paediatric Nephrology

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Acute glomerulonephritis (AGN)

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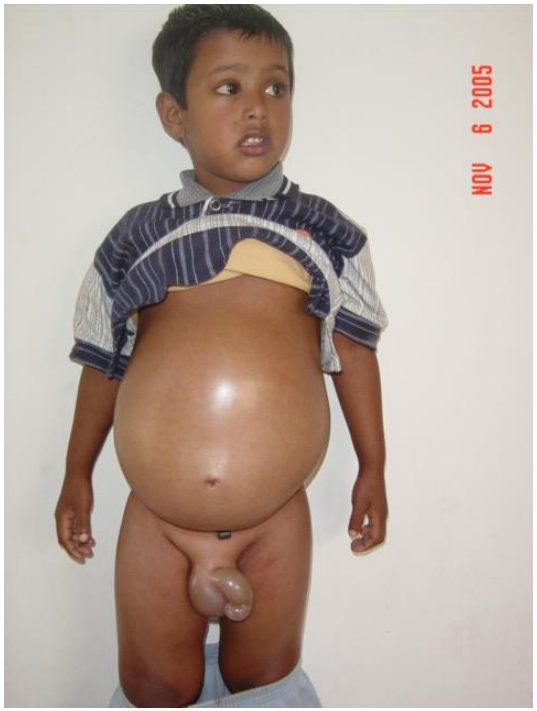
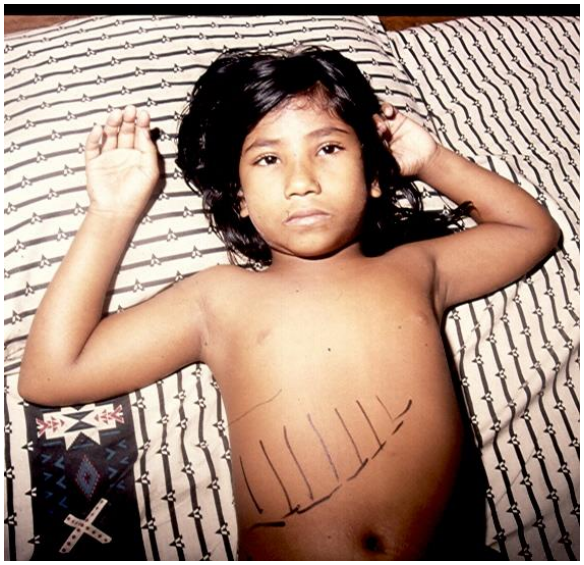
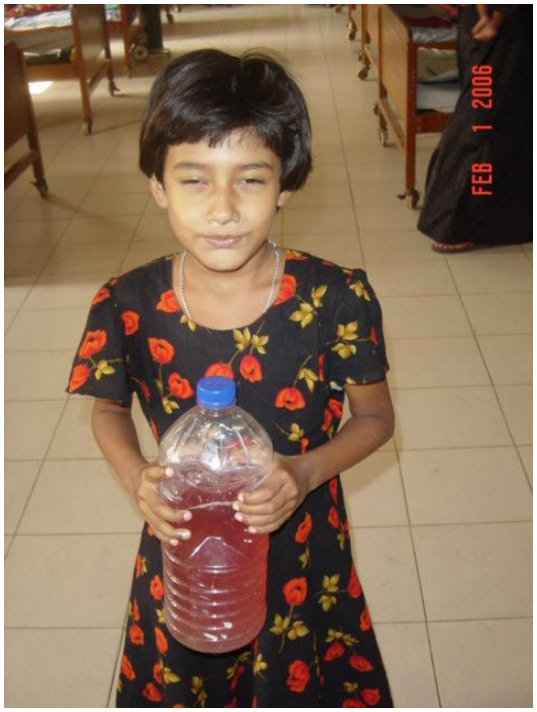
Scanty urine and swollen body



Nephrotic syndrome
(NS)



Acute
glomerulonephritis
(AGN)



Acute renal failure
(ARF)

AGN: Definition

- AGN is a renal disorder characterized by hematuria, oliguria, hypertension and volume overload following inflammation and proliferation of the glomerular capillary tuft.

AGN: Fatema 7 yrs

Swollen body with scanty high colored urine

AGN: Epidemiology

- AGN is the second most common renal disorder next to NS
- Acute post streptococcal gnomerulonephritis (APSGN) is the most common AGN affecting children between 5-15 yrs with the peak of 7-8 yrs in the developing countries

AGN: Types

Postinfectious
•Streptococcal, staphylococcal, Salmonella typhi, Treponema pallidium •Plasmodium malariae •Hepatitis B and C, CMV, parvovirus, Epstein Barr Virus •Associated with shunt infection, bacterial endocarditis
Systemic vasculitis
•Henoch-Schonlein purpura (HSP) •Systemic lupus erythematosus (SLE)
Others
•Membranoproliferative GN •IgA nephropathy •Hereditary nephropathy •Acute interstitial nephritis

Pathophysiology

- APSGN is basically an immune complex-mediated glomerulonephritis.
- APSGN occurs following pharyngitis, impetigo or rarely otitis media caused by group A β-hemolytic nephritogenic streptococci.
- Glomerular injury results from deposition of immune complexes in the glomerular capillaries.
- Nephritogenic antigens derived from streptococci may bind directly to subepithelial glomerular sites.
- Antibodies formed against these antigens combine and result in an inflammatory response, leading to activation of complement, infiltration of neutrophils, proliferation of glomerular cells and expansion of mesangial matrix.

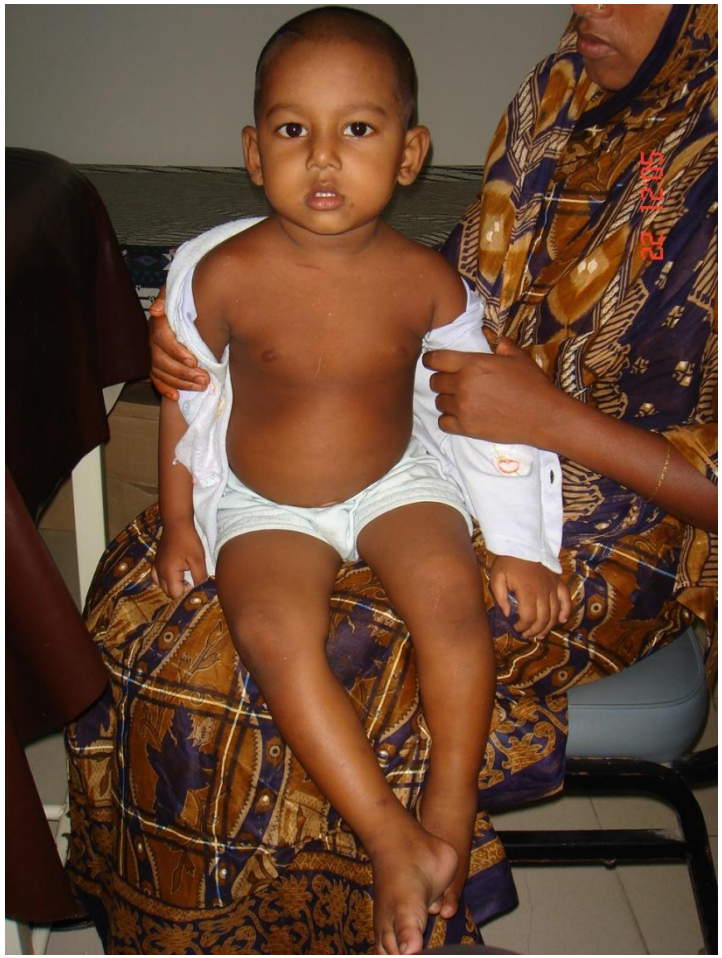
Pathophysiology cont'd

- Immunofluorescence examination shows granular deposits of IgG and C3 along the capillary walls and in the mesangium.
- Electron microscopy shows electron-dense subepithelial deposits or 'humps'.
- Resulting consequences are hematuria, hypertension, oliguria along with diminished GFR, salt and fluid retention and circulatory congestion.
- If filtration of solute is very much reduced, azotemia, acidemia, hyperkalemia and hyperphosphetemia occur. Anemia occurs due to volume expansion and is of normocytic and normochromic variety

AGN: Symptoms

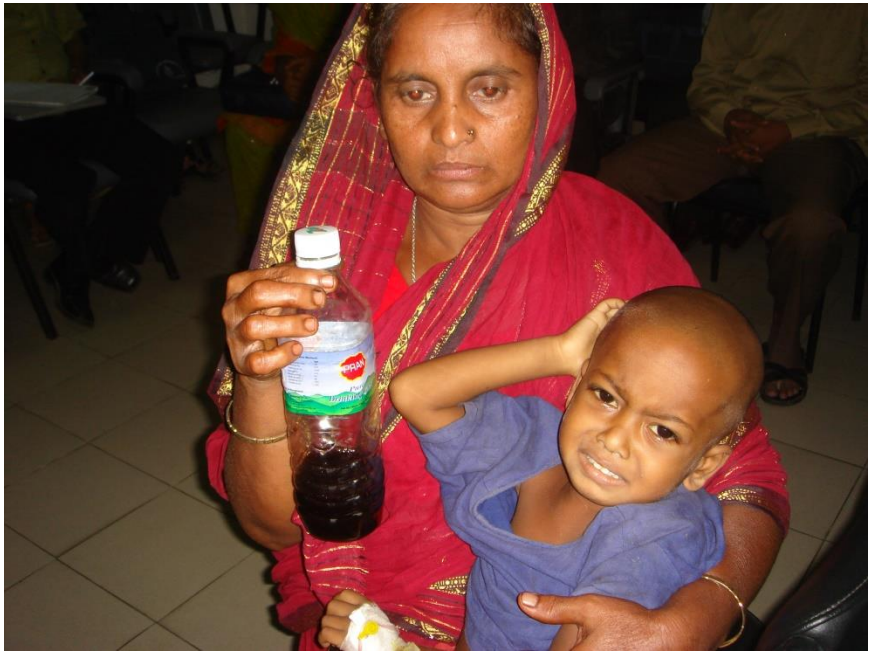
- Age: 5-15 years
- H/O sore throat/ pyoderma (2-4 weeks earlier)
- Scantly high colored urine
- Puffiness of face
- Suppression of urine
- Swelling of body: abdomen/ legs/ whole body
- Headache/ blurring of vision/ convulsion/ impaired consciousness
- Anorexia/ vomiting/ abdominal pain/ loose motion
- Respiratory distress/ cough/ hiccough
- Fever/ weakness
- Antecedent infection: scabies, infected scabies/ sore throat (in 7-21 days prior to kidney problem

AGN with swollen body



-3 years old child
-Puffy face
-Leg edema (turgid)

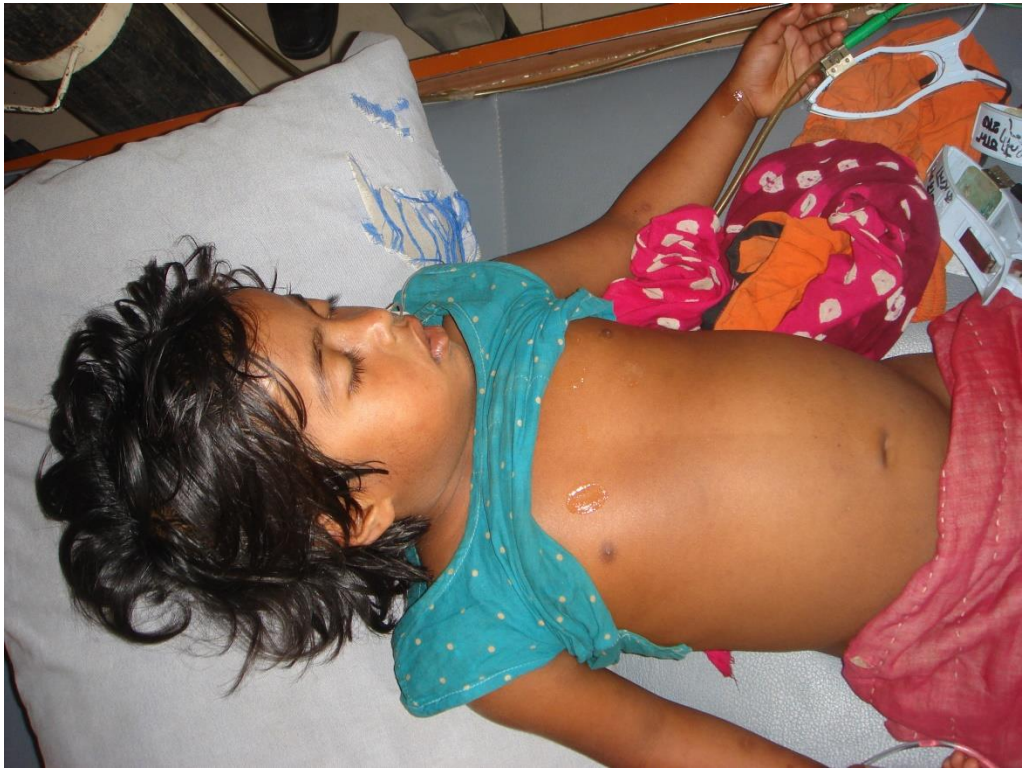
AGN with high colored urine



Coca cola urine

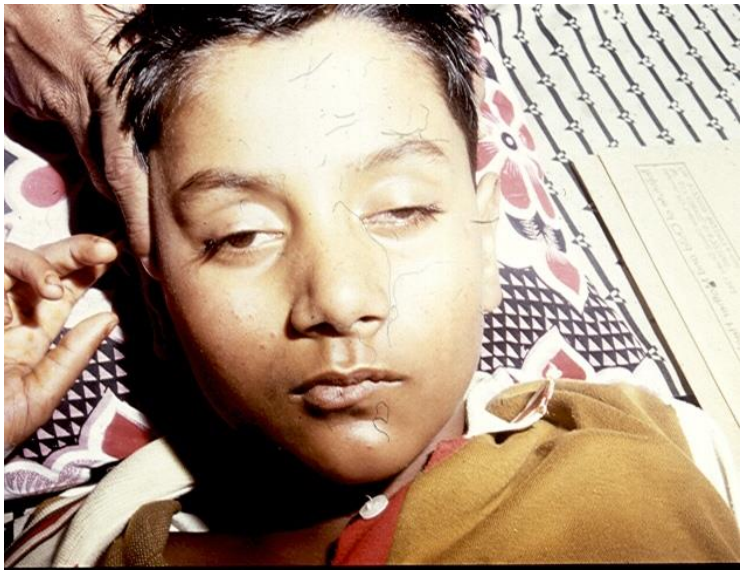
red urine

AGN with heart failure



6 yrs old female : puffy face, respiratory
Distress, engorged neck vein, hepatomegaly and ascitis

AGN with encephalopathy



Faruk 12 yrs: High BP, impaired consciousness

Faruk 12 yrs: AGN with multiple healed skin lesions

AGN : Signs

Appearance: Puffy face or eyelid swelling
Vital signs (HR/RR/BP/Temp.): Tachycardia/ tachypnoea/ ↑BP
Anthropometry (wt / ht - lh / OFC) : Age appropriate

General features
-Edema
-Skin infection: impetigo/ scabies/ scratch marks/ healed skin lesions

Systemic features
-Ascitis
-Heart failure/ gallop
-Enlarged tender liver
-Basal crepitation in lungs
-Hypertensive encephalopathy-impairment of consciousness

AGN: Investigations

Urine

-RE: smoky or coca cola color / albumin present 1+ / planty of RBC and RBC cast / a few pus cell

-24-hrs urine for proteinuria/ urine for protein creatinine ratio: normal

Blood

-CBC, PBF: non-specific

-urea/ creatinine: may be raised

-STP/ albumin: normal

-Serum electrolytes: hyperkalemia

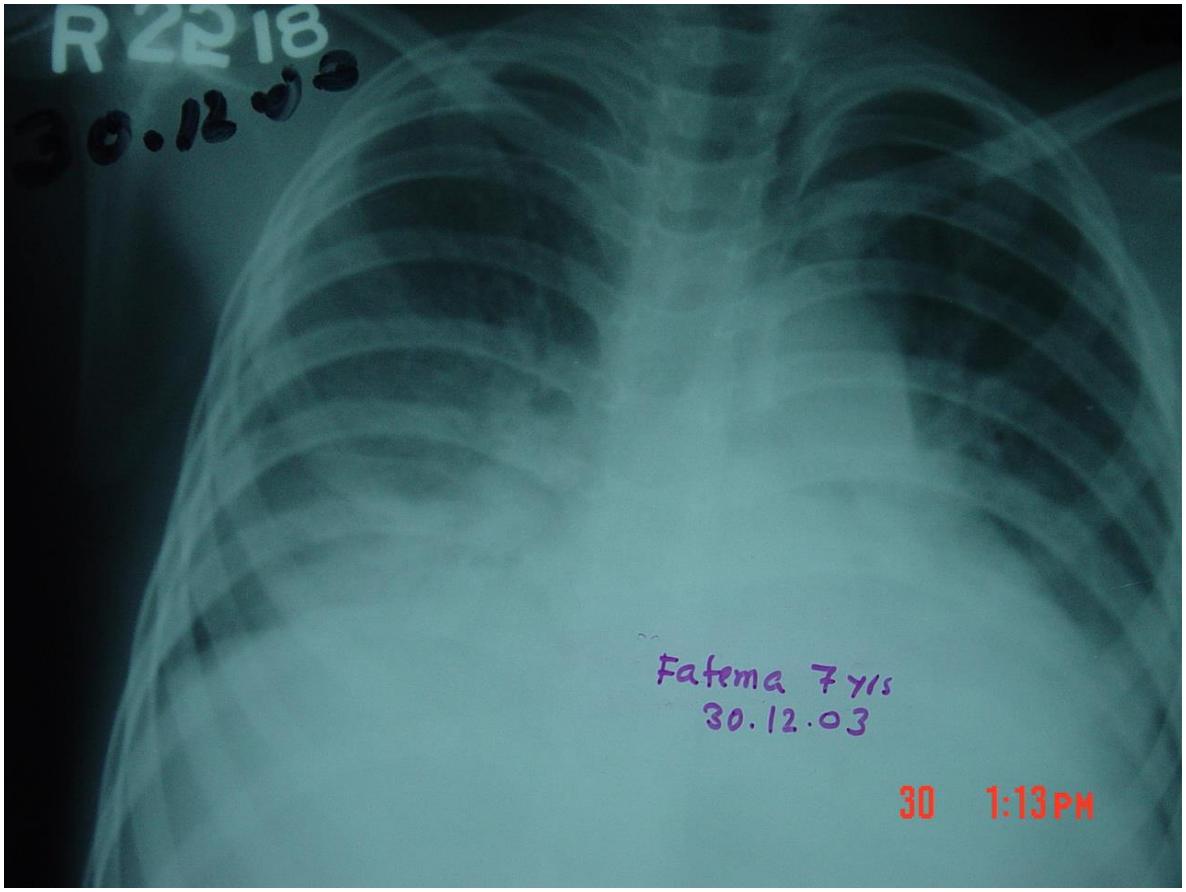
-ASO titre: may be raised

-Complement C3: may be reduced

X-ray chest: may have cardiomegaly/ pulmonary edema

ECG: peaked T wave, prolongation of PR interval, widen QRS complex (hyperkalemia)

CXR of Fatema having AGN



CXR: Enlarged heart shadow with pulmonary edema

AGN: Treatment

General	Complete bed rest Restriction of fluid 400 ml/m2 plus previous day's output Diet: salt restricted/ potassium and potassium containing food and fruits to be restricted -In severe cases ICU care may be needed -Hospitalisation in moderate to severe hypertension --Maintain chart for : GCS/ HR/ RR/ BP/ Wt/ Intake-output chart/ liver span

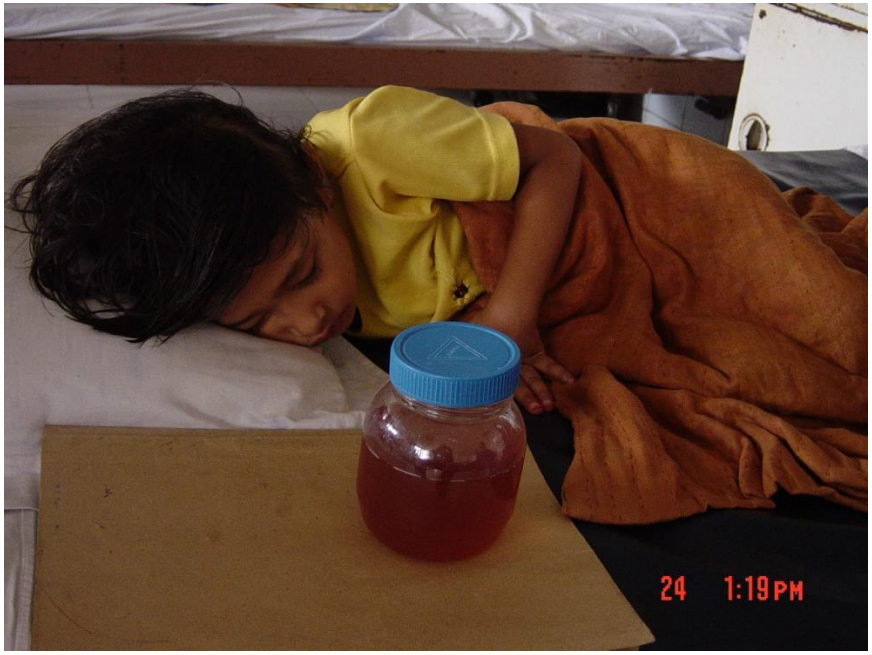
AGN: Treatment cont'd

Symptomatic	-Antihypertensives in case of hypertension (Frusemide, nifedipine) -Anticonvulsant for convulsion
Specific	Oral penicillin 250 per day 4 divided doses for 10 days
Complications	Heart failure: bed rest/ diuretics/ digoxin/ oxygen ARF: diuretics/ dialysis Hyperkalemia: IV sodibicarb/ glucose plus insulin/ calcium gluconate/ ion exchange resin/ dialysis Hypertensive encephalopathy: antihypertensive/ diuretics/ management for unconsciousness

AGN: Prognosis

- The long term prognosis of APSGN is excellent. Most of the patients (90%) recover. Recovery is complete even after serious condition

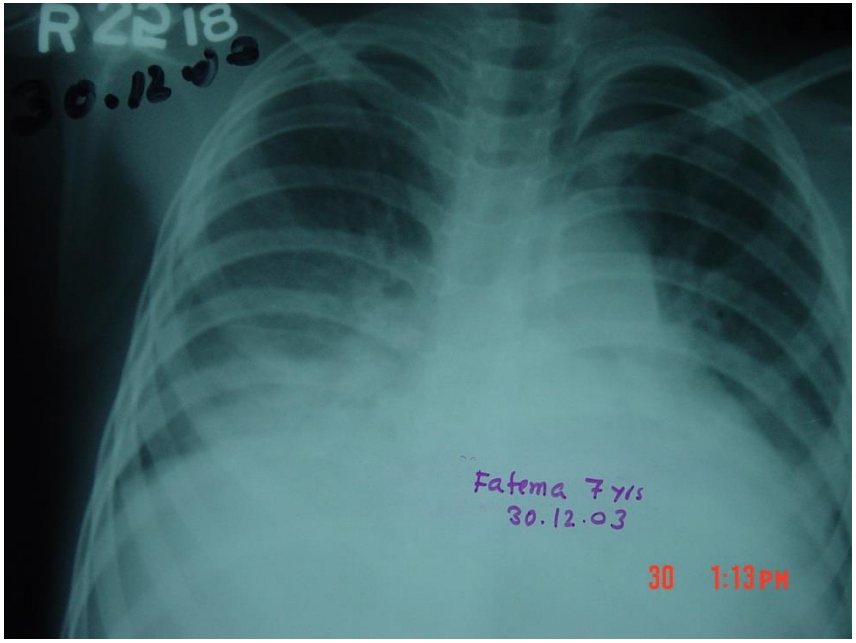
Fatema after improvement



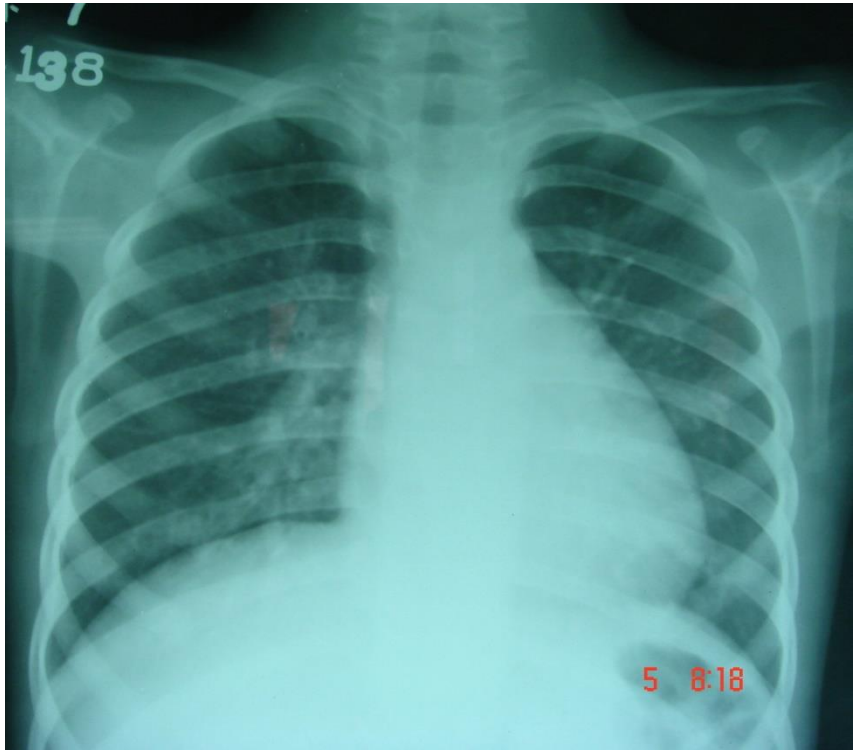
7 years old child with AGN

After improvement

Pulmonary edema

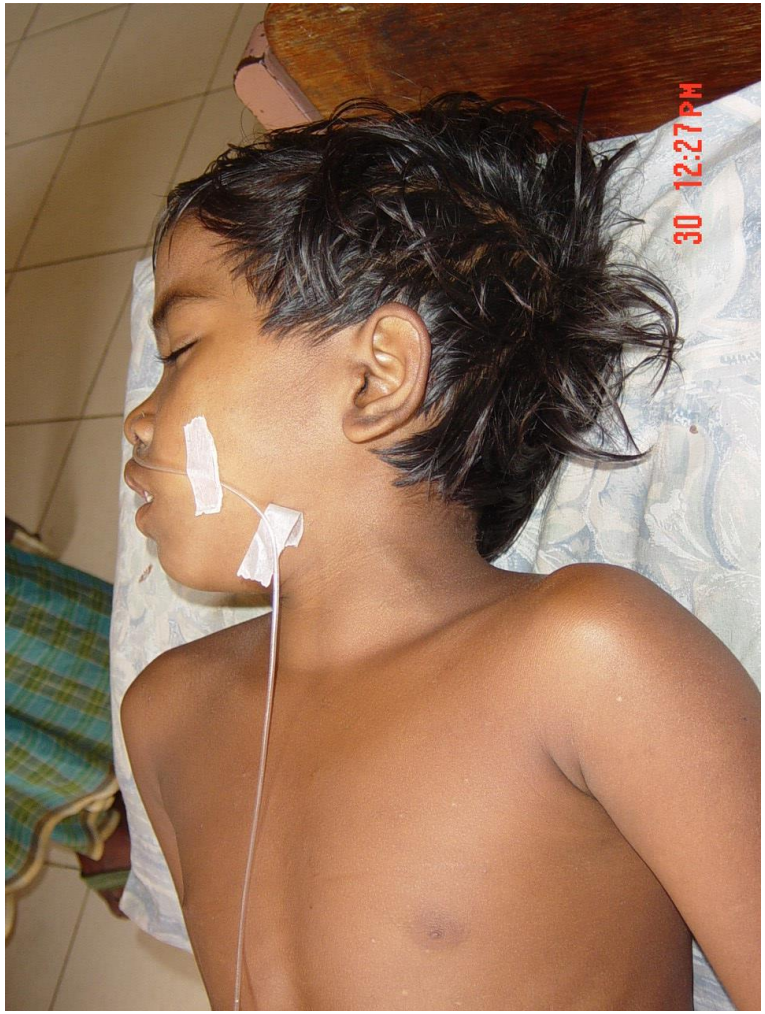


CXR: Pulmonary edema



CXR: Pulmonary edema improved

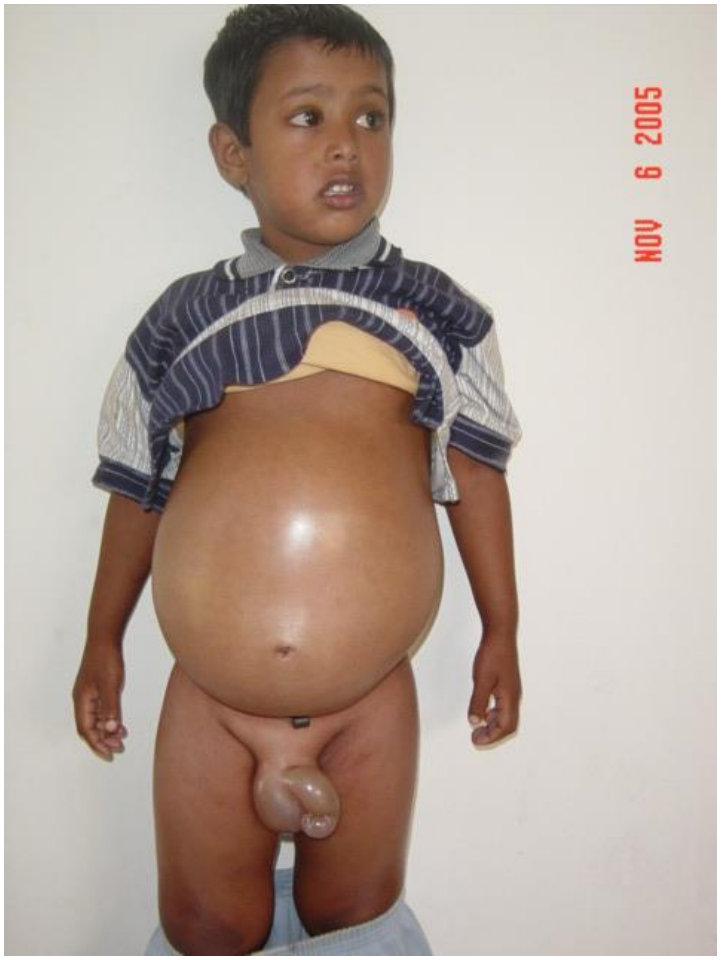
AGN who died



-7 yrs old female with AGN
-Hypertensive encephalopathy
-Died soon after admission

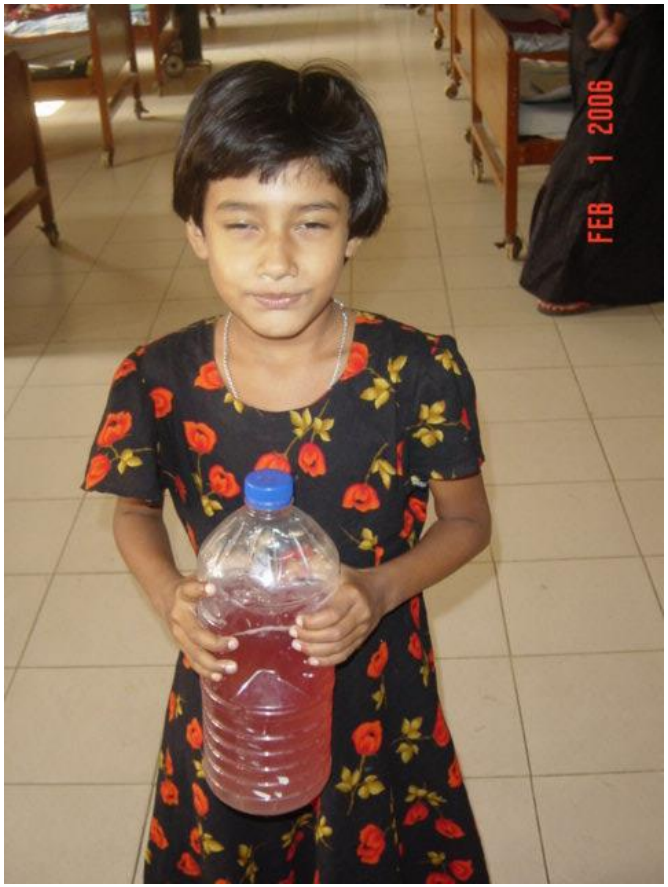
Differential diagnosis

Nephrotic syndrome



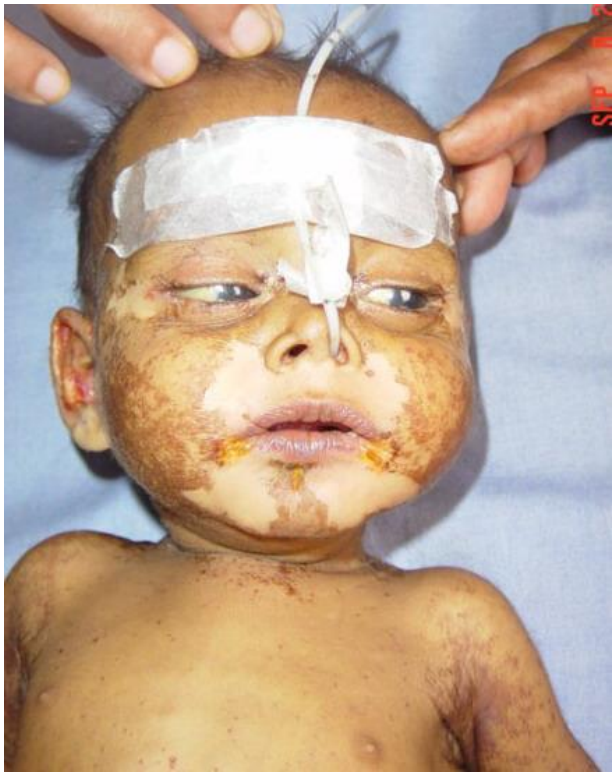
-4 yrs old male
-Scanty urine
-Swelling of whole body (anasarca)
-Massive proteinuria
-Hypoalbuminaemia
-Hypercholesterolemia

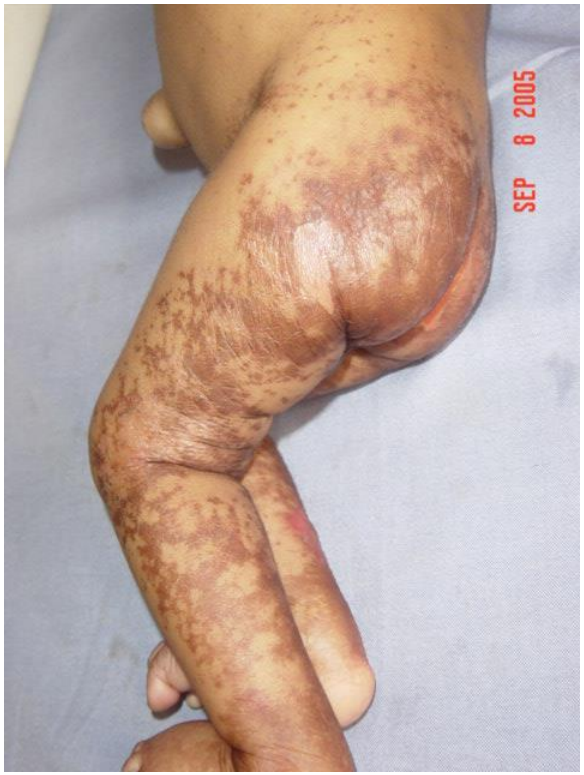
Acute Renal Failure



-7 yrs old female
-Scanty high colored urine
-Swelling of the body
-Hematuria/ albuminuria
-Urea 200 mg/dl
-Creatinine 3.0 mg/dl

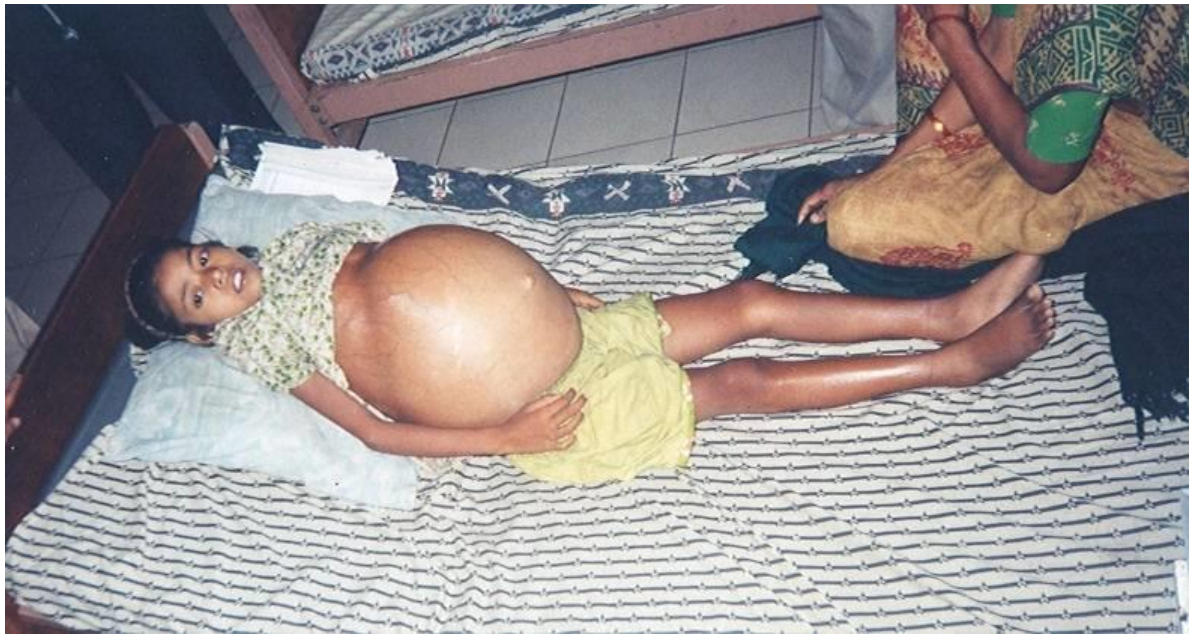
NS DD: Kwashiorkor





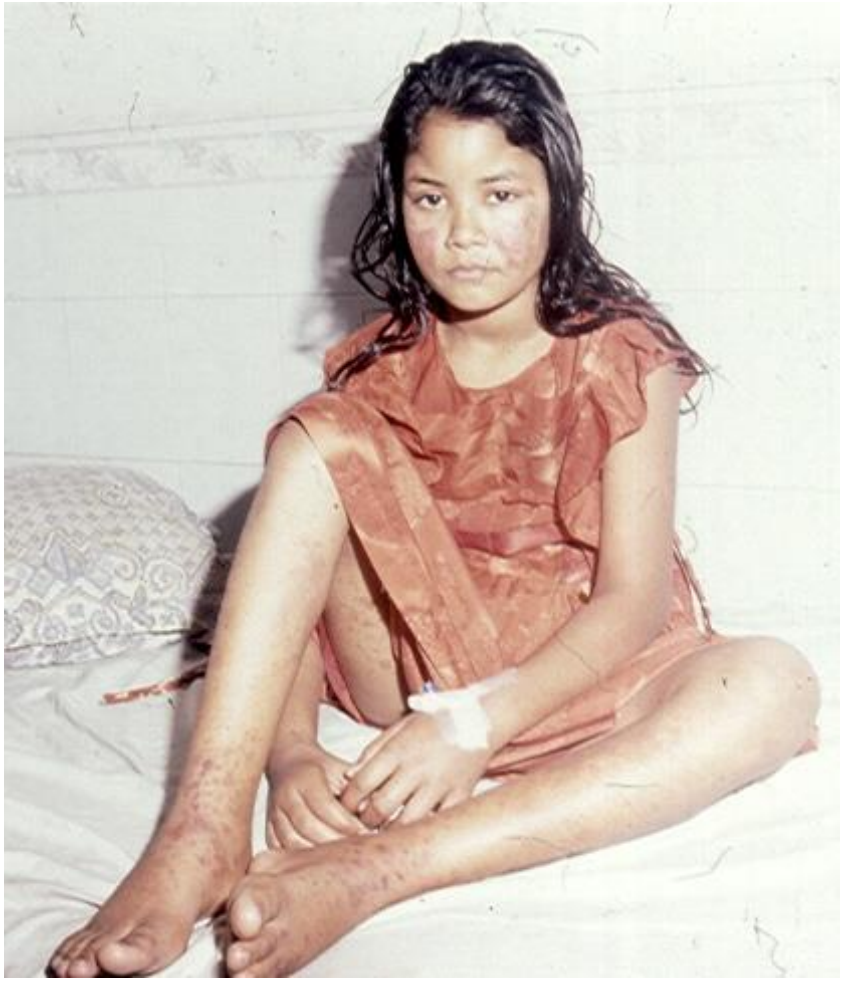
-One year old child
-Father rickshaw puller
-Edema
-Sparse hair
-Xerophthalmia
-Dermatosis

NS DD: Cirrhosis of liver



-10 yrs old female child
-H/O jaundice
-Ascitis/ edema
-Hepatosplenomegaly
-Hypoalbuminaemia
-HBsAg +ve

Henoch-Schonlein Purpura



-10 yrs old female
-Purpuric rash on the extensor surface of Limbs
-Abdominal pain
-Joint pain
-Hematuria

AGN: SAQ

Q1. Write down three important causes of scanty urine along with swelling of the body *(NS/ AGN/ ARF)*

Q2. A 7 year-old male child presented with scanty urine and puffy face. What is the most likely diagnosis? *(AGN)*

Q3. Why there occurs edema in AGN? *(reduced GFR due to glomerular injury because of immune complex deposition)*

AGN: SAQ cont'd

Q4. Name three important clinical features of AGN *(scanty high colored urine/ swelling of the body/ high BP)*

Q5. Name three important urinary features of AGN *(scanty urine/ hematuria/ urinary cast)*

Q6. Name three investigations to diagnose AGN *(Urine : hematuria, RBC cast in urine / CXR: enlarged heart shadow/ blood: azotemia, hyperkalemia)*

Q7. Name three complications of AGN *(Heart failure/ ARF/ hypertensive encephalopathy)*

AGN: SAQ

- Q8. What is the specific treatment of NS *(Penicillin either IV, IM or oral)*
- Q9. How will you counsel parents of a child having NS *(Problem in the kidney, the blood purifying and urine producing organ, the filtration function of which is at fault resulting in loss of red cells in urine, water logging and salt retention in the intravascular compartment. As a consequence, hypertension, heart failure, renal failure may occur and function of brain may be impaired)*
- Q10. What is the prognosis of AGN? *(Prognosis is excellent in children with complete recovery in most (90%) of the cases.*

Integrated Teaching on Glomerulonephritis

Q. 1) Master Alam a 7 years old boy came with H/O swelling of whole body starting from face and high colored scanty micturition for 5 days. What is your provisional diagnosis? How will you confirm your diagnosis?

Q. 2) Master Motin an 8 years old boy with swelling of whole body starting from face and high colored scanty micturition for 4 days. He has H/O skin lesion for 15 days. His classmates were suffering from same type of skin disease? What is your clinical diagnosis? What is the pathogenesis of underlying condition?

Q. 3) Master Sujit an 8 years old boy came with H/O swelling of whole body starting from face and high colored scanty micturition for 5 days. What is the clinical diagnosis? Describe the clinical features of the disease?

Q.4) Tania a 6 years old girl came with H/O severe headache with respiratory distress. She was suffering from scanty micturition for 7 days and swelling of whole body starting from face what is your clinical diagnosis? How will you manage the patient?

Q.5) Samira a 5 years old girl came with high colored scanty micturition for 4 days with H/O swelling of whole body starting from face. She is suffering from skin disease for 3 weeks. What is your provisional diagnosis? How will you differentiate the disease the nephritic syndrome?

Thanks