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Clinical Cases in PEDIATRICS

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CASE 4 Renal System

DIFFERENTIATION BETWEEN NEPHRITIS AND NEPHROTIC SYNDROME

Nephritist	*Nephrotic syndrome (Fig. 4.1)*
1. Incidence:	
School age (6–12 years)	Preschool (2–6 years)
2. H/O Infection	
H/O Sore throat	
H/O Skin infections	
3. **Clinical features:**	
Acute onset	Insidious onset
Mild to moderate edema	Massive
Hematuria +	-
Oliguria +	Oliguria +
Hypertension +	-
4. **Investigations:**	
Urine	
i. RBC's and RBC Casts	Hyaline casts
ii. Spot urine: $\frac{\text{Protein}}{\text{Creatinine}}$ 0.3 – 3	>3
iii. 24 hr urine	
1+	3+
2+	4+
iv. Albumin: Trace	50 mg/kg/day
	40 mg/m^2/ h
Blood:	
1. Total serum protein: Normal	Decreased(<5g/ dL)
2. Serum albumin: Normal	(<2.5 g/dL)
3. A:G ratio: Normal	Reversal
4. Cholesterol: Normal	>220 mg/dL
5 Electrophoresis: Normal	Decreased albumin ; α_2-globulin
X-ray chest:	
• Cardiomegaly	Hydrothorax
• Congested lung fields	(Transudate)
USG Abdomen:	
Renal enlargement	Kidney not enlarged

Contd...

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Contd...

Nephritist	*Nephrotic syndrome (Fig. 4.1)*
Treatment: • Salt restricted diet and rest • Penicillin: 10 days • Treat hypertension: Calcium-channel blockers • Diuretics **Complications:** • Acute renal failure • Hypertension • Electrolyte disturbance (Increased K^+ and PO_4^- Decreased Ca^{2+}) • Acidosis • Seizures	**Treatment:** • Rest/fluid/salt restricted diet • Diuretics • Steroids—prednisolone for 4–6 weeks • Cyclophosphamide (Immunosuppressants) **Complications:** • Infections (Spontaneous peritonitis) • Arterial/venous thrombosis • Ascites/pleural effusion

HISTORY TAKING

- Age : School going children (5–8 years—AGN)
- Young children (3–8 years—Nephrotic)
- Sex: More among boys
- Place
- Informant: Mother
- Reliability: Good

C/O

1. Puffiness and face
2. Oliguria
3. High-colored urine
4. Abdomen distention.



Figure 4.1: Nephrotic syndrome

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DD: EDEMA

1. Renal causes:

   Edema starts in face: more in morning
   1. AGN
   2. Nephrotic syndrome
   3. Acute/chronic renal failure
   4. Urinary tract infection
2. Cardiac causes:

   Edema ***–more in feet and sacrum (dependent***)
   1. CCF
   2. Pericarditis
   3. Pericardial effusion
3. Hepatic causes:
   1. Ascites
   2. Cirrhosis with PHT
4. Nutritional edema:
   - Kwashiorkor
   - Beriberi
5. Endocrine cause:
   - Cushing syndrome
   - Hypothyroidism
6. Collagen vascular disorders:
   - SLE
7. Superior mediastinal syndrome:
8. Angioneurotic edema:

   Ask H/O to rule out all causes:
   - H/O Pharyngitis, skin infection
   - H/O Oliguria
   - H/O Red/cola colored urine
   - H/O Breathlessness
   - H/O Chest pain, palpitation, orthopnea, paroxysmal nocturnal dyspnea (CCF)
   - H/O Abdomen pain, loss of appetite, jaundice –liver causes
   - H/O blurring vision, nausea, vomiting, convulsions (hypertension/hypertension encephalopathy)
   - H/O Recurrent RTI
   - H/O Dysuria,UTI
   - H/O Fever, rash, abdomen pain, arthritis (connective tissue disorders)
   - H/O Jaundice (hepatic encephalopathy)
   - H/O Ear discharge (secondary to streptococcal pharyngitis)
   - H/O Deafness (Alport's syndrome—deafness with hematuria).

PAST HISTORY

- H/O Similar illness before (nephrotic syndrome has 2–3 episodes before but rare in AGN)
- H/O Steroid therapy (nephrotic syndrome)
- H/O Skin infections—common in summer
- H/O Sore throat (post-streptococcal/glomerulonephritis)—common in winter
- H/O TB

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- H/O Ear discharge
- H/O Exanthematous fever/measles—IgA nephropathy
- **Antenatal/natal/postnatal-history:**
- **Developmental history**: In PEM
- **Diet history**: To r/o PEM producing edema (kwashiorkor)
- **Family history**: Similar illness: among family members social economic states
- **H/o Siblings with infection**: Surrounding area of living and over crow cling
- **Immunization history**: BCG vaccine
- **Contact history**: Tuberculosis, sore throat.

GENERAL EXAMINATION

- Conscious
- Cooperative
- Nourishment
- General Appearance: Puffy face with periodical edema (Fig. 4.2)
- Anemia, other vitamin deficiency
- Jaundice, clubbing, cyanosis
- Lymph adenopathy (Jugulo-digastric node)
- Pedal edema
  - Minimal edema- AGN
  - Massive (anasarca)—nephrotic/PEM

Look for – **previous impetiginous scar**: Cause for acute glomerulonephritis.
Examine—throat, tonsil, ear, scalp.

Vital Signs

- Pulse
- BP (sometimes labile hypertension in AGN)
- Temperature
- Respiratory rate.



Figure 4.2: Periorbital puffiness

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Anthropometry

- Height
- Weight
- Head circumference
- Chest circumference.

SYSTEMIC EXAMINATION

Abdomen- R/o Hepatosplenomegaly
Palpate for kidney
Look for renal angle tenderness
Suprapubic tenderness (cystitis)
Free fluid

CVS
RS – Pleural effusion (nephrotic syndrome)
CNS

DIAGNOSIS

- **AGN**
- **Nephrotic syndrome**

Viva Questions

Urine in AGN is cola colored. Why? (Fig. 4.3)

'**Acid hematin'** is produces the color.

Urine color in Nephrotic Syndrome:

1. White/straw color
2. Gets frothy in the container (due to increased protein)
3. Decreased/normal output

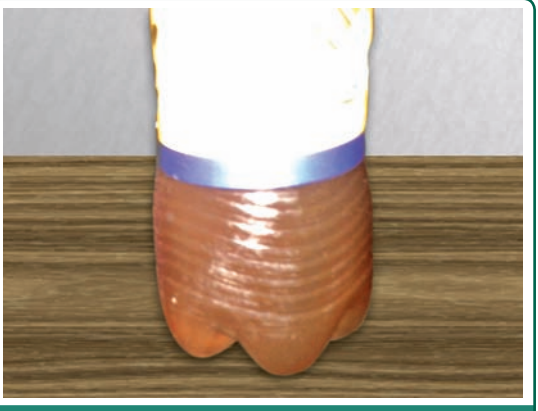

Figure 4.3: Cola color urine

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Differences between edema in

Acute glomerulonephritis	*Nephrotic syndrome*
• Turgid	• Soft
• Does not pit easily	• Pits readily

ACUTE GLOMERULONEPHRITIS (AGN)

- Rapid onset
- Minimal edema (puffiness of face periorbital edema)
- Oliguria
- H/O Preceding sore throat (or) skin infections (or) scabies
- Increased BP
- Hematuria
- No history of similar episodes
- Anemia.

Investigations

- Hematuria
- Renal function test/urine C/S
- To prove AGN/to exclude nephrotic syndrome

1. **Urine:** Macroscopic : Less quantity/high-colored urine(cola colored)
   Microscopic: RBC casts + Granular hyaline casts + Bacteria (UTI)
   Urinary protein: Not of much rise (1+)
2. **Serum**: Urea/creatinine levels: Increased depending upon renal function alteration
3. **Chest X-ray**: Hypervolemia can lead to pulmonary congestion, cardiomegaly
4. **Blood**: Protein, TC, DC, ESR, serum cholesterol
5. **ASO tirte:** Antihyaluronidase, AntiDNAse units increased
6. **Throat culture**
7. **C3 level decreased** : By 6 weeks, it will be normal if not by 6 months—R/O systems conditions; polyarteritis nodosa or other form of glomerulonephritis.

Management of AGN

1. **Bed rest**: Reduces edema/hypertension
2. **Penicillin**: 50,000–1lac units IM injection/orally
   (or) amoxicillin/ampicillin/erythromycin (for 10 days)
3. **Diet**:
   - No added salt
   - Fluids and salt restriction (sodium: 0.5 g/day—amount adding salt in separator)
   - Solids can be taken unlimited
   - Fruit juices/tender coconuts to be avoided
   - Sweet can be given (payasam, sweet pongal)
4. **Input-output maintenance**:
   - **1st day Input**: 20 mL/kg/day

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Insensible water loss = 400 mL

- **2nd day Input**: Calculate the total urine output on day I; add 400 mL with this (output + 400 mL) amount.

Example: 800 mL is output on day 1

Input 2nd day: 800 + 400 = 1200 mL fluid today

Replaced:

- Insensible loss by
  10% Dextrose
- **Urine output by**
  50%—normal saline
  50%—dextrose 10%

5. **Control of blood pressure:**

   >90 mm Hg diastolic, give
   - Sublingual nifedipine—0.01 mg/kg/day
   - Monitor ½ hourly for first 2 hours then 8th hourly (or) twice daily
   - BP ≤90 mm Hg: Give oral—nifedipine/hydralazine/atenolol

   Ask mother to keep head low position(since hypotension is frequent)

6. **Hypertensive crisis:**

   1st alpha methyldopa: 5–10 mg/kg IV drip

   Give Na^+—**Nitroprusside** = 0.5–8 µg/kg IV infusion

   Diazoxide = 5 mg/kg

   Nifedipine sublingual 300–500 mg/kg.

Monitor weight regularly: Loss should be an 0.5% of actual body weight every day

- Diuretics: Frusemide: 2 mg/kg/BW
- Indication:
  - Severe edema
  - Pulmonary congestion
  - Left ventricular failure.

Prognosis: Excellent in AGN

- Resolve in 2 weeks
- If not, renal biopsy to review the diagnosis
- Recurrence is rare
  - Macroscopic hematuria disappears by 2–3 weeks
  - Microscopic hematuria disappears by 6 months–1 year.

Complications

1. Hypertensive encephalopathy
2. Left ventricular failure; pulmonary edema
3. Electrolyte disturbances
4. RPGN—very rarely needs dialysis
5. Chronic renal failure.

Why renal edema is over face first?

1. Loose areola tissue
2. Patients are ambulant

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What is cause of anemia?

1. Dilutional
2. Hematuria

Causes of palpable kidney?

1. Hydronephrosis
2. Wilm's tumor
3. Polycystic kidney disease

What should be monitored before giving Frusemide?

- Blood urea
- Serum creatinine
- In case of increased blood urea when diuretics given because of uremia leads to encephalopathy, DIC

NEPHROTIC SYNDROME

- Young children (2–6 years)
- Insidious onset
- Massive edema/ascites; pleural effusion and hydrocele
- H/O Previous similar episodes (recurrence)
- H/O Steroid therapy
- H/O Normal/high-colored urine with normal quantity
- No H/O hematuria
- No hypertension
- In examination also, BP normal.

Investigations

- To diagnose protein in urine/ to include hematuria
- To diagnose blood protein level
- To diagnose cholesterol.

1. Urine Examination: Macroscopic—normal quantity/normal or high-colored
   Microscopic—no hematuria (hyaline, waxy casts)
   24 hour urine protein—>0.1 g/kg/h—heavy proteinuria

   Tests
   1. Heat coagulation test
   2. Sulfosalicylic acid test
   3. Albustix test
   4. Esbach Albuminometer

   WBC cast—pyelonephritis
   RBC morphology—altered when seen in phase contrast

   To diagnose hypoproteinemia:
   1. Serum albumin (N: 3.5–5 g/dL)
   2. A:G ratio (normal globulin: 1.5–3.5 g/dL)
   3. Serum: Protein/creatinine ratio (N: <0.3)

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4. Blood urea (N:20–40 mg/dL)
5. Serum creatinine (N: 0.1 to 0.5 mg/dL)
6. Electrophoresis mobility pattern

2. Serum: Total protein decreased
   Albumin level <2.5 g/dL
   Abumin: Globulin ratio reversed
3. Serum: Creatinine, protein ratio
   Normal: 0.7–1.5 mg/100 mL
   More than 3.5 = Nephrotic syndrome
4. Plasma urea, creatinine, elevated
5. Serum cholesterol: Elevated >200–220 mg%
   Proteinuria: >50mg/kg/day
6. Chest X-ray/mantoux: To R/O TB; because TB will flare up on steroid therapy
7. Serum electrolytes
8. Electrophoretic motility pattern: Albumin decreased, decreased α_2-globulin; AG reversal

Management (Figs 4.4 and 4.5)

Bed Rest

Furosemide: 1 mg/kg BW (tds or BD) to relieve edema with sodium, potassium and calcium supplements.

1. Monitor electrolytes
2. Diet/fluid correction: Refer AGN
3. Give protein such diet: Milk/white of egg
4. Treat other infections:
   - Pneumococcal pneumonia
   - Pneumococcal pneumonitis
5. Treat the TB
6. Steroid therapy: 2 mg/kg BW/day—for 6 weeks
   If there is nil proteinuria for 3 days after therapy, Taper the dose



Figure 4.4: Peritoneal dialysis

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Figure 4.5: Hemodialysis

- 1.5 mg/kg—next 4 weeks
- 1 mg/kg—next 4 weeks
- 0.5 mg/kg—next 4 weeks and stop drug

7. Prophylactic antibiotics
8. Relapse: Common—

   Steroids – 2 mg/kg/day for 10–14 weeks

   Taper the dose if there is nil proteinuria for 3 days successively.

   1.5/1/0.5 mg/kg for 4 weeks successively.

   Frequent relapses:
   - Give above treat with above 0.5 mg/kg—alternate days for 1 year
   - More than 4 episodes/1 year
9. **Steroid resistant cases:**
   - Review by renal biopsy

   Treatment:
   - Cyclophosphamide
   - Cyclosporin
   - Methotrexate.

Complications of Nephrotic Syndrome

- Electrolyte imbalance (due to edema)
- Peritonitis
- Pneumonitis
- Cellulitis
- Meningitis
- Thromboembolic manifests
- Acute renal failure.

Prognosis

- Child will have no residual dysfunction after 2nd decade up to which, relapses may occur
- Child may become fertile (except when cyclophosphamide or chlorambucil—sterile).

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PROTEINURIA

Normal: 150 mg/24 h
Mild: 0.5 g/24 h
Moderate: 0.5–2 g/24 h
Massive: >2 g/24 h

- Hypoproteinemia is due to proteinuria >2g/24h
- Hematuria: (h/o)
  Proximal—Urethritis(RBC morphology not altered)
  Terminal—Cystitis
  - Schistosomiasis
  - Calculi

  Throughout – AGN (altered RBC morphology)
- DD—red urine:
  1. Hematuria
  2. Hemoglobinopathies
  3. Rifampicin
  4. Beet root
  5. Coloring dyes
  6. Porphyrias
  7. Newborn.

Causes of Hematuria

- AGN
- Nephrotic syndrome (H/O fever, infection)
- Rent calculi (H/O abdominal pain)
- Bleeding diathesis (H/O purpuric spots)
- Cystitis, UTI, hemangiomas
- **Drugs**: Sulfonamides, aspirin, cyclophosphamide.

Macroscopic Hematuria

Coca or coffee-colored urine
Frank blood-extraglomerular.

Microscopic Hematuria

>2 RBC/HPF – uncentrifuged urine
>5 RBC/ HP – centrifuged urine.

Tests to Detect Proteinuria

1+ : 30 mg/dL
2+ : 200 mg/dL
3+ : 300 mg/dL
4+ : 2000 mg/dL

Heat Coagulation Test

- Fill 2/3 of test tube with the sample
- Heat the upper part, opalescence is formed

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- Add a few drops of (acetic acid-glacial)
  - Disappears: Phosphate +
  - Persists: Protein.

Sulfosalicylic Acid Test

- To 5 mL sample, add 5 drops of 10% sulfosalicylic acid (white precipitate formed).

ACUTE STREPTOCOCCAL GLOMERULONEPHRITIS

Most common cause of hematuria: IgA nephropathy

1. Characterized by: Sudden onset of
   1. Acute hematuria
   2. Edema
   3. Hypertension
   4. Final

Etiology and Epidemiology

Follows infection of throat/skin
With certain nephritogenic strains of Group A beta-hemolytic streptococci

- Cold weather – AGN follow streptococcal pharyngitis
- Warm weather – AGN follows skin infections and pyoderma
- Epidemics of nephritis have been associated with serotype 12 (throat) also serotype 4 and serotype 49 (skin)

AGN follows:
Respiratory infection in 2–3 weeks
Skin infection in 4–6 weeks

Pathology:

- kidneys symmetrically enlarged
- Light microscopy – all glomeruli enlarged
  - Relatively blood less
  - Diffuse mesangial cell proliferation
  - Increase in mesangial matrix
- Polymorphonuclear leukocytes are common (early stages)
- Crescents (severe cases).

Immunofluorescence

- 'Lumpy–bumpy deposits' of immunoglobin
- Deposits of immunoglobulin and complex on GBM and mesangium.

Electron Microscopy

Electron dense deposits (HUMPS) on the epithelial side of GBM (Glomerular basement membrane).

Pathogenesis

- Decreased serum complement (C3) level
- Suggests immune complex-mediated.

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