بسم الله الرحمن الرحيم
Chronic Kidney Disease in Children

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Supervised by:-
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Chronic Kidney Disease (CKD)

is a slowly progressive loss of renal function over a period of months or years and defined as renal injury (proteinuria) and/or a glomerular filtration rate <60 mL/min/1.73 m² for >3 mo

The normal GFR in children is 90 - 130 ml/min/1.73m²
CKD is divided into 5 stages according to GFR:

<table>
<thead>
<tr>
<th>Stage</th>
<th>Description</th>
<th>GFR mL/min/1.73 m²</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Slight kidney damage with normal or increased filtration</td>
<td>More than 90</td>
</tr>
<tr>
<td>2</td>
<td>Mild decrease in kidney function</td>
<td>60-89</td>
</tr>
<tr>
<td>3</td>
<td>Moderate decrease in kidney function</td>
<td>30-59</td>
</tr>
<tr>
<td>4</td>
<td>Severe decrease in kidney function</td>
<td>15-29</td>
</tr>
<tr>
<td>5</td>
<td>Kidney failure; requiring dialysis or transplantation</td>
<td>Less than 15</td>
</tr>
</tbody>
</table>
Causes

The age of presentation of CKD correlates closely with the underlying cause:

- Congenital
- Acquired
- Metabolic

- $< 5$ years $\rightarrow$ congenital nephropathies and obstructive uropathy.

- $> 5$ years $\rightarrow$ acquired glomerular diseases (chronic glomerulonephritis, haemolytic uremic syndrome) or hereditary disorders (Alport's syndrome, Juvenile nephronophthiasis) are common causes.

- CKD related to metabolic disorders (cystinosis, hyperoxaluria) and certain inherited disorders (polycystic kidney disease) may present throughout the childhood years.
### Causes of CKD

<table>
<thead>
<tr>
<th>Table 3</th>
<th>Diagnosis distribution of North American Pediatric Renal Trials and Collaborative Studies (NAPRTCS) chronic renal insufficiency (CRI) patients [10]</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Distributions by diagnosis</strong></td>
<td><strong>Number</strong></td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>6,405</td>
</tr>
<tr>
<td><strong>Primary diagnosis</strong></td>
<td></td>
</tr>
<tr>
<td>Obstructive uropathy</td>
<td>1,385</td>
</tr>
<tr>
<td>Aplastic/hypoplastic/dysplastic kidney</td>
<td>1,125</td>
</tr>
<tr>
<td>Other</td>
<td>913</td>
</tr>
<tr>
<td>FSGS</td>
<td>557</td>
</tr>
<tr>
<td>Reflux nephropathy</td>
<td>536</td>
</tr>
<tr>
<td>Polycystic disease</td>
<td>257</td>
</tr>
<tr>
<td>Prune belly</td>
<td>185</td>
</tr>
<tr>
<td>Renal infarct</td>
<td>155</td>
</tr>
<tr>
<td>Unknown</td>
<td>168</td>
</tr>
<tr>
<td>HUS</td>
<td>134</td>
</tr>
<tr>
<td>SLE nephritis</td>
<td>96</td>
</tr>
<tr>
<td>Cystinosis</td>
<td>97</td>
</tr>
<tr>
<td>Familial nephritis</td>
<td>99</td>
</tr>
<tr>
<td>Pyelo/interstitial nephritis</td>
<td>87</td>
</tr>
<tr>
<td>Medullary cystic disease</td>
<td>82</td>
</tr>
<tr>
<td>Chronic GN</td>
<td>76</td>
</tr>
<tr>
<td>MPGN-type I</td>
<td>67</td>
</tr>
<tr>
<td>Berger's (IgA) nephritis</td>
<td>64</td>
</tr>
<tr>
<td>Congenital nephrotic syndrome</td>
<td>68</td>
</tr>
<tr>
<td>Idiopathic crescentic GN</td>
<td>46</td>
</tr>
<tr>
<td>Henoch-Schönlein nephritis</td>
<td>40</td>
</tr>
<tr>
<td>MPGN-type II</td>
<td>29</td>
</tr>
<tr>
<td>Membranous nephropathy</td>
<td>33</td>
</tr>
<tr>
<td>Other systemic immunologic disease</td>
<td>25</td>
</tr>
<tr>
<td>Wilms tumor</td>
<td>28</td>
</tr>
<tr>
<td>Wegener's granulomatosis</td>
<td>17</td>
</tr>
<tr>
<td>Sickle cell nephropathy</td>
<td>13</td>
</tr>
<tr>
<td>Diabetic GN</td>
<td>11</td>
</tr>
<tr>
<td>Oxalosis</td>
<td>6</td>
</tr>
<tr>
<td>Drash syndrome</td>
<td>6</td>
</tr>
</tbody>
</table>
Incidence

According to United States Renal Data System (USRDS):

- 10 – 12% of all renal cases
- 20 case per million population
Signs and Symptoms

Signs and symptoms of renal failure are due to overt metabolic derangements resulting from inability of failed kidneys to regulate electrolyte, fluid, and acid-base balance; they are also due to accumulation of toxic products of amino acid metabolism in the serum. Signs and symptoms include the following:

- Anorexia
- Vomiting
- Bone pain
- Headache
- Malaise
- High urine output or no urine output
- Recurrent urinary tract infections
- Urinary incontinence
- Pale skin
- Acidotic breathing
- Hearing deficit
- Detectable abdominal mass
- Edema
- Irritability
- Poor muscle tone
- Change in mental alertness
- Stunted growth
Pathogenesis

- progressive injury with ongoing structural or metabolic genetic diseases
- Hyperfiltration injury
- Proteinuria
- Hypertension
- Hyperphosphatemia
- Hyperlipidemia
## Pathophysiology

<table>
<thead>
<tr>
<th>MANIFESTATION</th>
<th>MECHANISMS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Accumulation of nitrogenous waste products</td>
<td>Decrease in glomerular filtration rate</td>
</tr>
<tr>
<td>Acidosis</td>
<td>Impaired bicarbonate reabsorption</td>
</tr>
<tr>
<td></td>
<td>Decreased net acid excretion</td>
</tr>
<tr>
<td>Sodium retention</td>
<td>Excessive renin production</td>
</tr>
<tr>
<td></td>
<td>Oliguria</td>
</tr>
<tr>
<td>Sodium wasting</td>
<td>Solute diuresis</td>
</tr>
<tr>
<td></td>
<td>Tubular damage</td>
</tr>
<tr>
<td>Urinary concentrating defect</td>
<td>Solute diuresis</td>
</tr>
<tr>
<td></td>
<td>Tubular damage</td>
</tr>
</tbody>
</table>
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<thead>
<tr>
<th>MANIFESTATION</th>
<th>MECHANISMS</th>
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<tbody>
<tr>
<td>Hyperkalemia</td>
<td>Decrease in glomerular filtration rate</td>
</tr>
<tr>
<td></td>
<td>Metabolic acidosis</td>
</tr>
<tr>
<td></td>
<td>Excessive potassium intake</td>
</tr>
<tr>
<td>Renal osteodystrophy</td>
<td>Impaired renal production of 1,25-dihydroxycholecalciferol</td>
</tr>
<tr>
<td></td>
<td>Hyperphosphatemia</td>
</tr>
<tr>
<td></td>
<td>Hypocalcemia</td>
</tr>
<tr>
<td></td>
<td>Secondary hyperparathyroidism</td>
</tr>
<tr>
<td>Growth retardation</td>
<td>Inadequate caloric intake</td>
</tr>
<tr>
<td></td>
<td>Renal osteodystrophy</td>
</tr>
<tr>
<td></td>
<td>Metabolic acidosis</td>
</tr>
<tr>
<td></td>
<td>Anemia</td>
</tr>
<tr>
<td></td>
<td>Growth hormone resistance</td>
</tr>
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<th>MECHANISMS</th>
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</thead>
<tbody>
<tr>
<td>Anemia</td>
<td>Decreased erythropoietin production, Iron deficiency, Folate deficiency, Vitamin B12 deficiency</td>
</tr>
<tr>
<td>Bleeding tendency</td>
<td>Defective platelet function</td>
</tr>
<tr>
<td>Infection</td>
<td>Defective granulocyte function, Impaired cellular immune functions, Indwelling dialysis catheters</td>
</tr>
<tr>
<td>Neurologic symptoms (fatigue, poor concentration, headache, drowsiness, memory loss, seizures, peripheral neuropathy)</td>
<td>Uremic factor(s), Hypertension</td>
</tr>
</tbody>
</table>
## Pathophysiology

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</thead>
</table>
| Gastrointestinal symptoms (feeding intolerance, abdominal pain) | Gastroesophageal reflux  
Decreased gastrointestinal motility |
| Hypertension                                       | Volume overload  
Excessive renin production |
| Hyperlipidemia                                     | Decreased plasma lipoprotein lipase activity |
| Pericarditis, cardiomyopathy                       | Uremic factor(s)  
Hypertension  
Fluid overload |
| Glucose intolerance                               | Tissue insulin resistance                      |
Laboratory Findings

- Blood urea nitrogen ↑↑
- Serum creatinine ↑↑
- S. K⁺ ↑↑
- S. Na⁺ ↓↓ (if volume overloaded)
- Arterial pH ↓ (acidosis)
- S. Ca²⁺ ↓↓
- S. Phosphorus ↑↑
- Patients with heavy proteinuria can have hypoalbuminemia
- CBC: normochromic, normocytic anemia
- Serum cholesterol and triglyceride levels may be elevated
- Urinalysis:
  - 🌻 GN → hematuria and proteinuria
Management

**GENERAL PRINCIPALS**

- Treat reversible renal dysfunction
- Prevent or slow the progression of renal disease
- Treat the complications of CKD
Slowing the progression of kidney dysfunction

1) Optimum Control of Hypertension
2) Control of proteinuria
3) Serum Phosphorus within normal range
4) Serum Calcium-Phosphorus Product < 5 mmol²/l²
5) Prompt treatment of infectious complications and dehydration
6) Correction of anemia
7) Avoidance of cigarette smoking
8) Minimization of regular use of NSAIDs
9) Control of hyperlipidemia
10) Dietary Protein restriction
COMPLICATIONS OF CKD

- Disorders of fluid and electrolytes
- Renal Osteodystrophy
- Anemia
- Hypertension
- Dyslipidemia
- Growth impairment
- Decreased clearance of renally excreted substances from the body (uremia).
Management of Health Problems Associated with Chronic Kidney Disease

- **Fluid and Electrolyte Management**

  - Patient with high blood pressure, oedema, or heart failure require sodium restriction and diuretic therapy.

  - Fluid restriction is rarely necessary until the development of ESRD.

  - Hyperkalemia is treated by restriction of potassium intake, administration of oral alkalinizing agents,
Figure 29  Hyperkalemia may cause cardiac arrest.
Hyperkalemia Treatment

- Calcium gluconate (carbonate)
- Sodium Bicarbonate
- Insulin/glucose
- Lasix
- Albuterol
- Hemodialysis
**Acidosis**

- Sodium bicarbonate is used to maintain the serum bicarbonate level above 22 mEq/L.

**Anemia**

- Anemia becomes manifested at a GFR less than 35 ml/min/1.73 m².

- Erythropoietin is usually initiated when the patient's Hb concentration falls below 10 g/dL, at a dose of 50 – 100 unit/kg S.C. one to three times weekly. The dose is adjusted to maintain the Hb concentration between 11 – 12 g/dL (not more than 13 g/dL)

- All patients receiving erythropoietin therapy should be provided with either oral or intravenous iron supplementation
Anemia:

Failure to respond to therapy

• Infection or inflammation
• Chronic blood loss
• Folate or vitamin B12 deficiency
• Malnutrition
• Hemoglobinopathies
• Hemolysis
Hypertension

- Children with CKD may sustained hypertension related to volume overload and/or excessive rennin production related to glomerular disease.

- Hypertensive children with suspected volume overload should follow a salt-restricted (2-3 gram/24 hr) and may benefit from diuretic therapy.

- ACE inhibitors are the antihypertensive medication of choice in all children with proteinuric renal disease.

- Angiotensin-II blockers have been shown to be effective in controlling BP and in slowing disease progression in patients with diabetic nephropathy.

- Ca channel blockers, centrally acting agents, and β- blockers may be useful in children whose BP cannot be controlled with the above agents.
**Renal osteodystrophy**

- The goals of treatment are to prevent bone deformity and normalize growth velocity using both dietary and pharmacologic interventions.

- Children and adolescents should follow a low phosphorus diet, and infants should be provided with a low phosphorous formula.

- Phosphate binders are used to enhance fecal phosphate excretion.

- Ca carbonate and Ca acetate are the most commonly used phosphate binder, although newer, non-calcium based binders such as sevelamer are increased in use, particularly in patients prone to hypercalcemia.

- The cornerstone of therapy for renal osteodystrophy is vitamin D administration.
– vitamin D therapy is indicated in patients with (1) renal osteodystrophy with PTH levels greater than three times the upper limit of normal or (2) persistent hypocalcemia despite reduction of the serum phosphorus level below 6 mg/dL.

– Therapy may be initiated with 0.01-0.05 µg/kg/24 hr of calcitriol.

– Phosphate binders and vitamin D should be adjusted to maintain the intact PTH level two to three times the upper limit of normal and the serum calcium and phosphorus levels within the normal range of age.
**Growth impairment:**

- Metabolic acidosis
- Decreased caloric intake
- Renal osteodystrophy
- Alteration on GH metabolism
**Nutrition**

– Sufficient energy should be provided in the diet or else the body tissues will be broken down to provide energy. This should be avoided as all tissues are mainly protein in nature and if broken down they increase the urea and creatinine levels.

– Proteins of high biological value or ‘good quality’ are preferred (are metabolized primarily to usable aminoacids rather than to nitrogenous wastes) and are found mainly in eggs, milk, meat, fish and poultry. Proteins of lower biological value are found in pulses, cereals, nuts, oilseeds and in some vegetables like greenpeas and dried beans. Normally 0.8-1.0gm/kg of body weight protein is effective.

– Dietary phosphorus, potassium, and sodium should be restricted according to the individual patient's laboratory studies and fluid balance.
...and this dish is totally potassium-free!
Vaccination

Children with CKD should receive all standard immunization according to the schedule used for healthy children, with the following exception:

- Live virus vaccines (MMR, varicella) must be avoided in immunosupressed patients (renal transplantation).

- All children with CKD should receive a yearly influenza vaccine, but it must be as Inactivated Influenza Vaccine (TIV) and not as Live Attenuated Influenza Vaccine (LAIV).

- Special care must be paid to viral hepatitis B vaccination, especially in pre-end stage renal failure.

- The efficacy of pneumococcal vaccination in CKD patients, including those on dialysis, may be considerably low, and so they may require repeat vaccination or an increased dose of vaccine.
H. Hemodialysis

Indications

A. Uremia - azotemia with symptoms and/or signs

B. Severe Hyperkalemia

C. Uremic encepalopathy

D. Volume Overload - usually with congestive heart failure (pulmonary edema)

E. Toxin Removal - ethylene glycol poisoning, theophylline overdose, etc.
Hemodialysis

- 3-4 times a week
- Takes 2-4 hours
- Machine filters blood and returns it to the body
Long-term Follow Up

close monitoring of a patient's clinical and laboratory status

- Blood studies to be followed routinely include:
  - serum electrolytes
  - blood urea nitrogen (BUN)
  - creatinine
  - calcium
  - phosphorus
  - albumin
  - alkaline phosphatase
  - Hb and Htc

- Periodic measurement of intact parathyroid hormone (PTH) levels and roentgenographic studies of bone may be of value in detecting early evidence of renal osteodystrophy

- Echocardiography should be performed periodically to identify left ventricular hypertrophy and cardiac dysfunction that can occur as a consequence of the complications of CKD
The preferred treatment in children with ESRD is transplantation, to allow the child to have the most possible normal growth and health. But transplantation is not always possible, and even sometimes the transplanted kidney may not work properly and still the child need dialysis despite transplantation.
Thank You