- **Fluids, electrolytes and dehydration:**
  - **The most common cause of acute fluid and electrolyte disturbance is acute diarrhea with dehydration.**
  - **Management of dehydration can be with:**
    - ✓ Oral Rehydration Solutions (ORS): if it is mild-moderate.
    - ✓ IV fluids: if it is moderate-severe.
  - **Total fluid needed by the body is the sum of** = maintenance + deficit + replacement of ongoing losses.
  - **Maintenance fluid**: it is the normal amount of fluid needed by the body to maintain its metabolic functions. It is calculated according to the following:

1st 10 kg of weight	100 ml/kg
2nd 10 kg of weight	50 ml/kg
Rest of weight	20 ml/kg

    - ✓ Maintenance of Na = 2-3 mEq/kg/day.
    - ✓ Maintenance of K = 2-3 mEq/kg/day.
  - **Fluid deficit**: losses caused by diarrhea or vomiting. Calculated according to severity of dehydration:

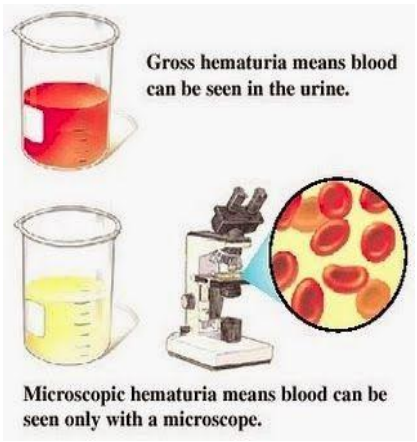
Severe dehydration (15% loss of weight)	150 ml/kg
Moderate dehydration (10% loss of weight)	100 ml/kg
Mild dehydration (5% loss of weight)	50 ml/kg

    - ✓ Notice that dehydration can also be classified according to concentration of Na:

Hyponatremic dehydration	< 130 mmol/L
Isonatremic dehydration	130-150 mmol/L
Hypernatremic dehydration	> 150 mmol/L

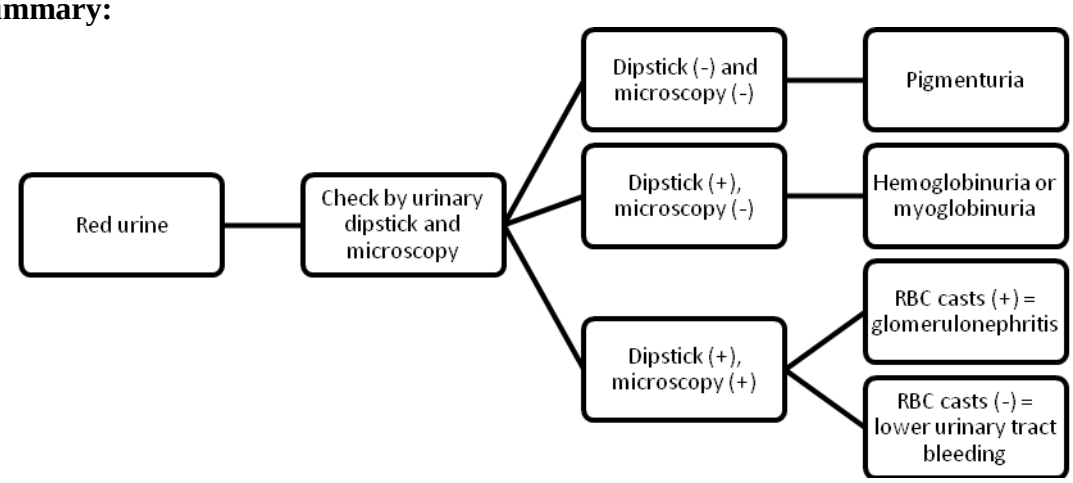
  - **How to administer IV fluids?**
    - ✓ If patient presents with severe dehydration and disturbance in hemodynamic status (e.g. hypotension and tachycardia) → a bolus of normal saline must be given (20 ml/kg).
    - ✓ Fluid maintenance is given according to the following:
      - ❖ *In the first 8 hours* = 1/3 maintenance + 1/2 deficit.
      - ❖ *In the following 18 hours* = 2/3 maintenance + 1/2 deficit.
    - ✓ Na correction must be gradual and slowly (especially in hypernatremic dehydration) to prevent the occurrence of cerebral edema.

- **Hematuria**:
  - **It is defined as the presence of blood in urine which can be:**
    - ✓ Gross: seen by the naked eye.
    - ✓ Microscopic: ≥ 6 RBCs/HPF in ≥ 3 consecutive samples of urine.
      - ❖ Urinary dipstick might also be used which detects the presence of hemoglobin or myoglobin in urine. False-negative results occur with large ingestions of vitamin C.



- ❖ *Dysmorphic RBCs and RBC casts* = glomerular bleeding.
- ❖ *Normal biconcave RBCs* = lower urinary tract bleeding.

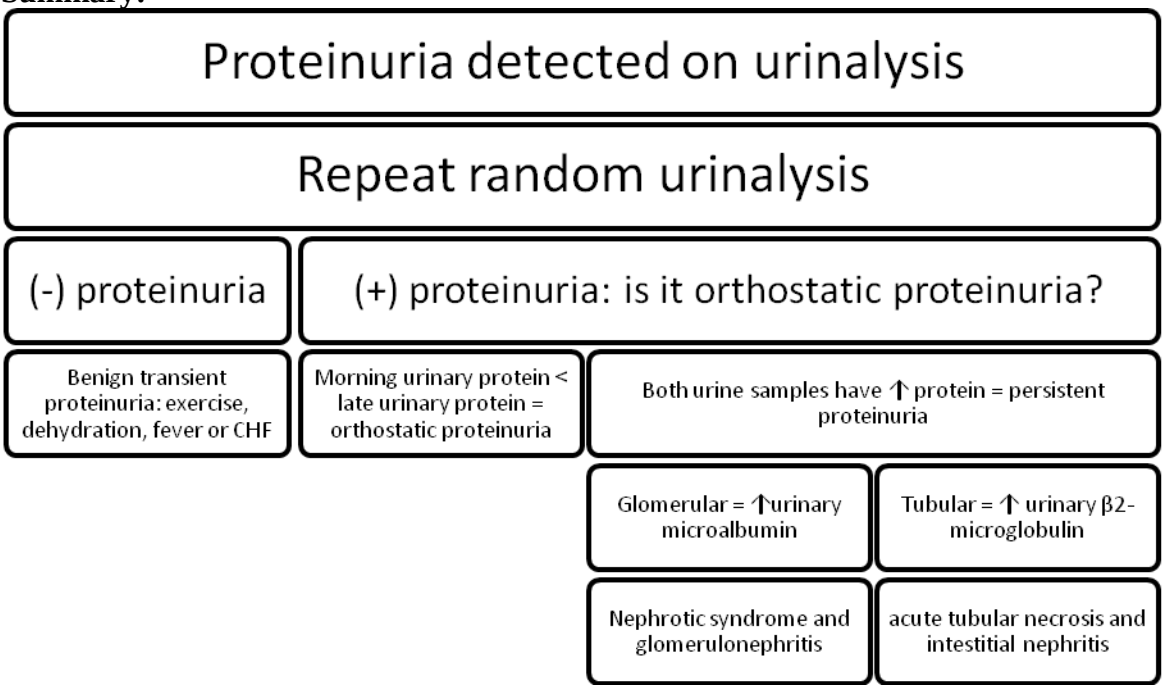
- **Clinical significance**: hematuria indicates a lot of conditions for example glomerulonephritis, presence of a tumor or a stone or a trauma.
- **Summary:**



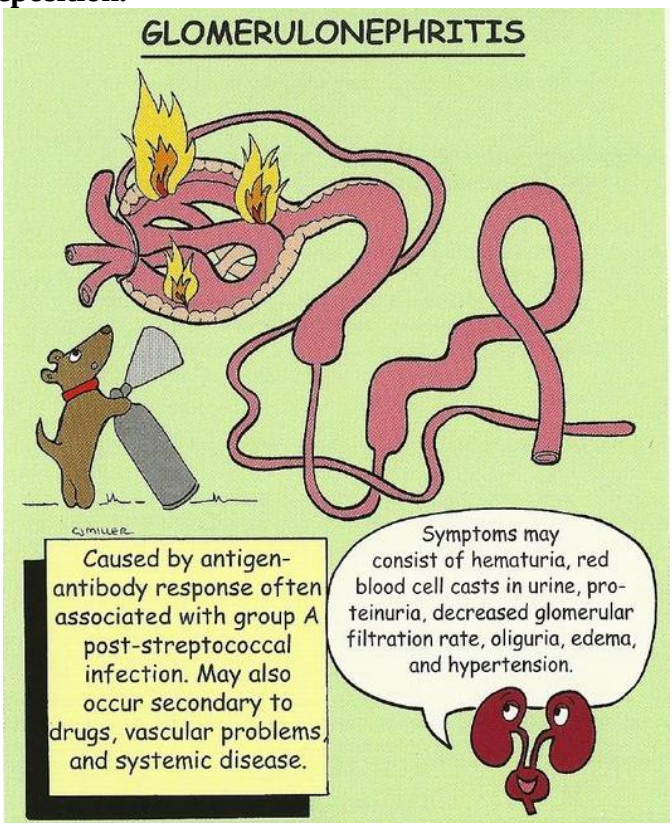
- **Proteinuria:**

- **It is defined as the presence of protein in urine > 100mg/m^2/day. It can be detected by:**
  - ✓ Urinary dipstick (most commonly used method):
    - ❖ *False-positive*: concentrated urine; alkaline urine (pH ≥7) or certain medications (e.g. aspirin and penicillin).
    - ❖ *False-negative*: diluted urine.
  - ✓ 24-hour urinary protein collection (most accurate method): but it is difficult to be done in children thus replaced with total protein-to-creatinine ratio (TP/CR):
    - ❖ *Normal TP/CR ratio in infants 6-24 months < 0.5*
    - ❖ *Normal TP/CR ratio in children > 2 years <0.2*

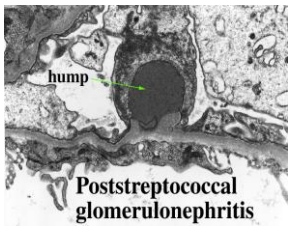
- **Summary:**



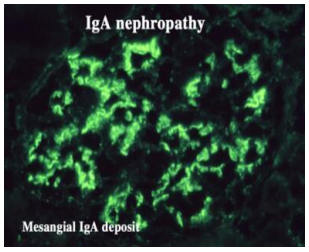
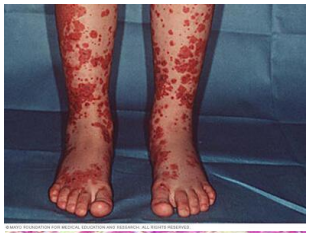
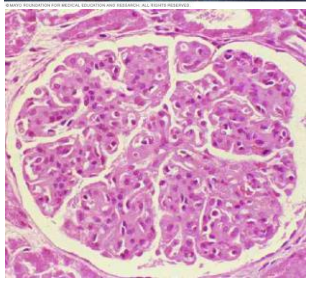
- **Glomerulonephritis:**
  - **It is defined as inflammatory changes within glomeruli caused by immune complex deposition.**



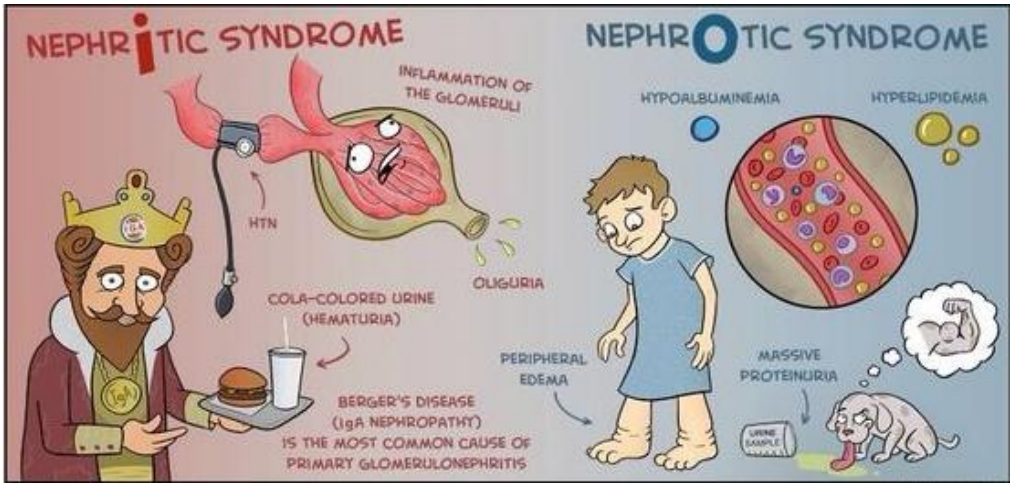
  - **Classification:**
    - ✓ Primary: disease process limited only to the kidney.
    - ✓ Secondary: there is a systemic disease (such as SLE).
  - **Clinical presentation is that of nephritic syndrome which is characterized by:**
    - ✓ Gross hematuria.
    - ✓ Hypertension.
    - ✓ Signs of fluid overload from renal insufficiency (edema).
  - **Laboratory investigations to be done when glomerulonephritis is suspected:**
    - ✓ ↑blood pressure.
    - ✓ Urinalysis (to check the presence of RBC casts and morphology of RBCs).
    - ✓ Urinary TP/CR ratio (to check for proteinuria).
    - ✓ Serum complement components.
    - ✓ ANA (maybe SLE is caused); ASO (maybe it is post-streptococcal glomerulonephritis).
    - ✓ Serum IgA level is checked when IgA nephropathy is suspected.
  - **Common types of glomerulonephritis in children:**



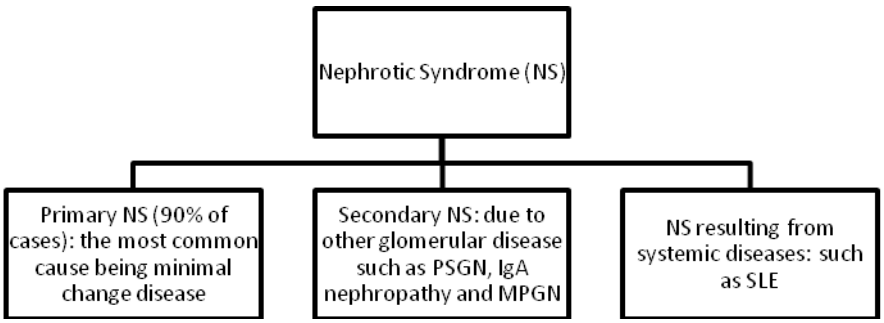
Post-Streptococcal Glomerulonephritis (PSGN: most common type of acute glomerulonephritis)	• Occurring 2 weeks after an infection to the skin or pharynx with nephrogenic strain group A β-hemolytic streptococcus. • **Clinical presentation**: cola-colored urine (hematuria), hypertension and edema (features of nephritis syndrome). • **Lab investigations**: ↓serum C3, ↑ASO (with streptococcal pharyngitis)/ ↑ADB (with streptococcal skin infection) and urinaylsis will show hematuria and proteinuria • **Management**: supportive (anti-hypertensive drugs and restriction of fluids and proteins). Prognosis is excellent with complete recovery

	**IgA nephropathy (Berger's disease: most common type of chronic glomerulonephritis)**	• **Cause**: abnormal formation/clearance of IgA immune complexes. • **Clinical presentation**: recurrent episodes of gross hematuria associated with respiratory infections. • **Diagnosis**: ↑serum IgA level (50% of patients); mesangial proliferation and increased mesangial matrix (LM); mesangial deposition of IgA (IF). • **Management**: supportive with 20-40% of patients progressing to ESRD.
	**Henoch-Schonlein Purpura (HSP) nephritis**	• **It is an IgA-mediated vasculitis characterized by**: palpable non-thrombocytopenic purpura on buttocks and lower extremities, arthritis/arthralgia, abdominal pain and gorss/microscopic hematuria. • Renal features of HSP are self-limited with complete recovery within 3 months.
	**Membranoproliferative Glomerulonephritis (MPGN)**	• **It is characterized by:** ✓ Thickening of glomerular basement membrane. ✓ Lobular mesangial hypercellularity. • **Clinical features**: combination of both nephrotic and nephritic syndromes. • **Management**: no definitive treatment but some patients might respond to corticosteroids and ACE-inhibitors slow the disease progression. • **Prognosis**: most patient progress into ESRD.

- **Nephrotic syndrome:**
  - **It is characterized by the following:**
    - ✓ Proteinuria (> 50mg/kg/day).
    - ✓ Hypoalbuminemia.
    - ✓ Hypercholesterolemia.
    - ✓ Edema.



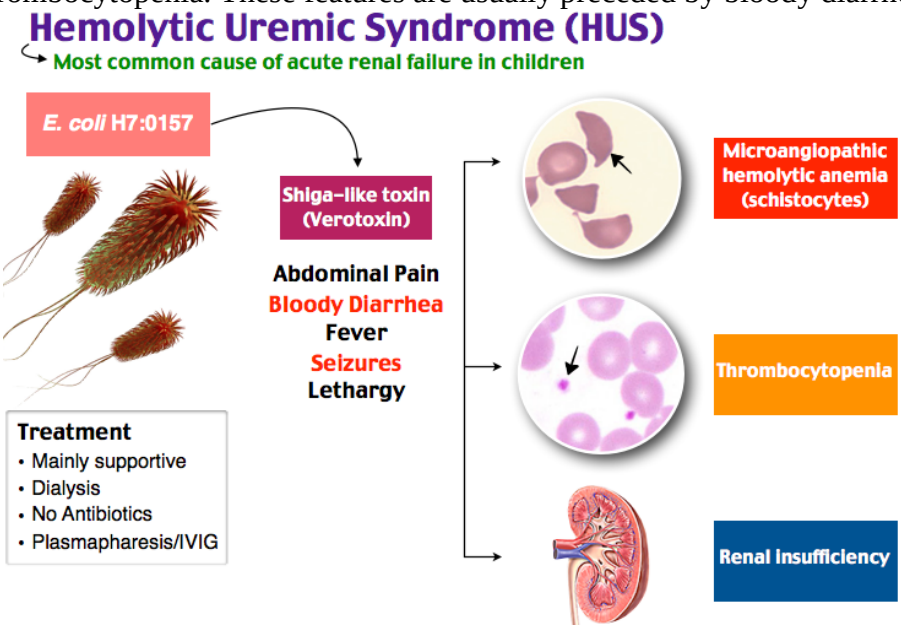
  - **It presents before 5 years of age (in 2/3 of cases) and it is more common in boys.**
  - **Classification:**



- **Clinical features:**
  - ✓ Edema (most common presentation): it can be periorbital, scrotal/labial or widespread (if very severe).
  - ✓ Increased risk of thrombosis due to hypercoagulable state caused by loss of anti-thrombin III in the urine. This might result in stoke, renal vein thrombosis or DVT.
  - ✓ Increased risk for infection with encapsulated organisms (such as S.pneumoniae).
- **Laboratory investigations:**
  - ✓ CBC: ↑Hct (because hypoalbuminemia will result in escape of fluids to interstitial tissues and subsequent hemoconcentration).
  - ✓ Urinalysis: 3^{+}-4^{+} proteinuria by urinary dipstick.
  - ✓ ↓serum albumin.
  - ✓ ↑serum cholesterol.
- **Management**: corticosteroids (prednisolone). If mortality occur, it is usually due to thrombosis or infection.

- **Hemolytic Uremic Syndrome:**

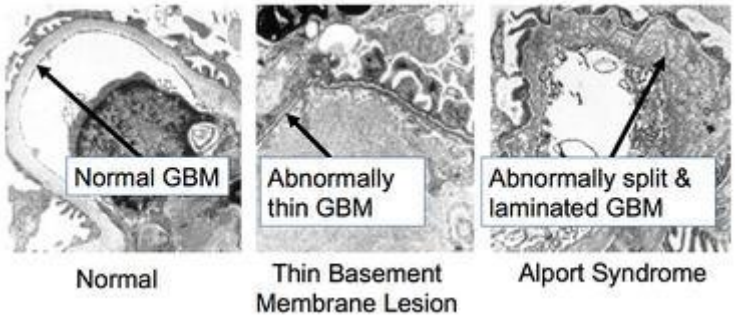
- **It is characterized by**: acute renal failure, microangiopathic hemolytic anemia and thrombocytopenia. These features are usually preceded by bloody diarrhea.



- **Classification:**

Shiga toxin-associated HUS	Atypical HUS
• It is the most common and caused by E.coli O157:H7 strain which is acquired from undercooked beef, unpasteurized milk or contaminated fruit juices. • The toxin will cause vascular endothelial injury especially in kidneys resulting in thrombi formation and ischemia. • Management: supportive. Notice that antibiotics are NOT indicated! • Poor prognostic signs: ↑WBCs on admission and prolonged oliguria.	• It is less common. • Caused by: ✓ Drugs: oral contraceptives, tacrolimus, cyclosporine and OKT3. ✓ Inherited: AD or AR pattern • Clinical features: similar to those of Shiga toxin-associated HUS but without diarrhea. • Management: supportive. If there is any medication causing the condition it must be stopped. • Prognosis: higher risk for ESRD.

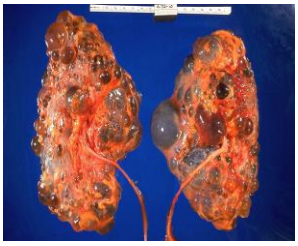
- **Hereditary renal diseases:**
  - **Alport's syndrome:**
    - ✓ **It is an X-linked dominant disease which is characterized by deficiency of type-IV collagen within glomerular basement membrane.**



    - ✓ **Clinical features:**

Renal	Hypertension, hematuria and ESRD (most common in males)
Hearing loss	50% of adult patients will have some loss of hearing
Ocular abnormalities	25-40$ of patients

    - ✓ **Management**: ACE-inhibitors to slow the progression of the disease but eventually patients will need renal transplantation.
  - **Polycystic Kidney Disease:**



Infantile (ARPKD)	Adult (ADPKD)
• **Uncommon** • **Clinical features**: enlarged cystic kidneys, maternal history of oligohydramnios, pulmonary hypoplasia, severe hypertension and cirrhosis with portal hypertension • **Prognosis**: renal transplantation is needed	• **Common** • **Clinical features**: flank masses, abdominal pain, hypertension, gross/microscopic hematuria and UTIs • **Prognosis**: renal transplantation is needed

- **Hypertension (HTN):**
  - **Normal blood pressures during childhood depends on the child's age.**
  - **Classification of HTN:**

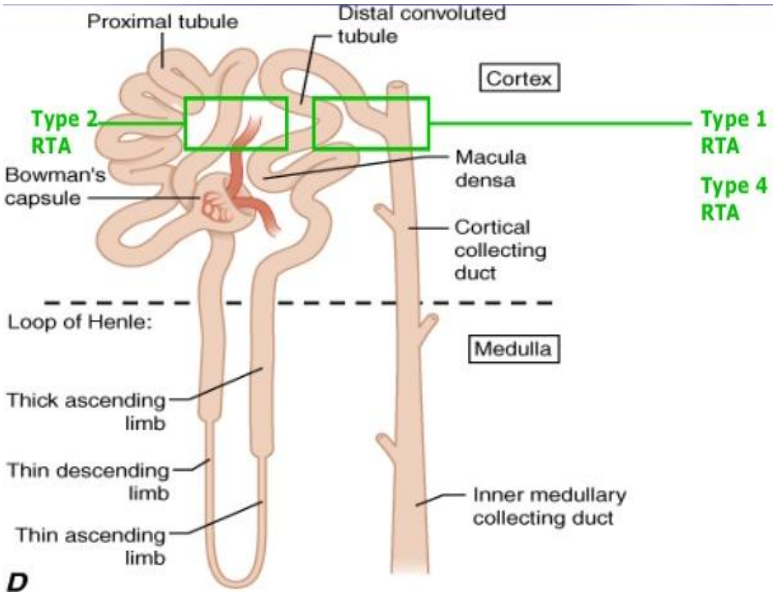
Significant HTN	> 95th percentile for age
Severe HTN	> 99th percentile for age
Malignant HTN	Evidence of end-organ damage (retinal hemorrhage, papilledema, seizures or coronary artery disease in adults)
Essential HTN	HTN with unknown cause
Secondary HTN	• **HTN with a recognizable cause.** • **Most common type of HTN in childhood.** • **Causes:** ✓ Neonates/infants: renal artery embolus, renal artery stenosis, renal disease or coarctation of aorta. ✓ 1-10 years: renal diseases or coarctation of aorta. ✓ Adolescents: renal diseases or essential HTN.

  - **Physical examination of HTN in childhood**: blood pressure must be measured in all four limbs to rule-out coarctation of aorta. In coarctation, there is hypertension in the right arm with lower blood pressures in the legs.
  - **Management:**
    - ✓ If there is an underlying cause of HTN → treat it.
    - ✓ Essential HTN → conservative management essentially → if it fails → anti-hypertensive medications.

- ✓ Hypertensive emergencies (e.g. severe headache, stroke, seizure CHF) → immediate therapy with IV anti-hypertensives.

- **Renal Tubular Acidosis (RTA):**
  - **It is defined as the inability of kidneys to maintain normal acid-base balance due to abnormality is reabsorption of HCO_3 or excretion of H^+**
  - **RTA can be:**
    - ✓ Congenital: caused by mutations in transporters in proximal or distal tubular cells.
    - ✓ Acquired: due to nephrotoxic drugs (such as amphotericin) or systemic autoimmune diseases (such as SLE).
  - **Clinical features**: growth failure, vomiting and normal anion gap hyperchloremic metabolic acidosis (with exception to type-I RTA in which anion gap is positive).
  - **Classification:**

Type	Feature	Cause	Presentation	Treatment
Distal RTA (type-I)	Inability to excrete H^+ by distal tubular cells	Congenital; drugs (amphotericin); associated with nephrotic syndrome	Growth failure; vomiting; acidosis	Small doses of oral alkali
Proximal RTA (type-II)	Inability to reabsorb HCO_3 by proximal tubular cells	Congenital; drugs (gentamicin); intoxication with heavy metals	Growth failure; vomiting; acidosis	Large doses of oral alkali
Type-III RTA	Subtype of type-I + inability to reabsorb HCO_3	-	-	Large doses of oral alkali
Type-IV RTA	Transient acidosis with hyperkalemia	-	-	Furosemide and oral alkali



- **Renal failure:**
  - **Acute renal failure:**
    - ✓ It is defined as a sudden decrease in the ability of kidney to excrete nitrogenous wastes.
    - ✓ Classification:

Type	Cause	Examples	Lab findings
Pre-renal	↓renal perfusion resulting in ↓GFR	CHF, hemorrhage or dehydration	↑urea/creatinine ratio > 20; ↑urine osmolality > 500

Renal parenchymal	Damage to glomerulus	PSGN, HUS, lupus nephritis	Hematuria and proteinuria
	Damage to renal tubules (acute tubular necrosis)	Renal hypoperfusion	↑urinary β_2-microglobulin
	Damage to interstitium (interstitial nephritis)	Drugs (semisynthetic penicillins)	Eosinophilia and eosinophiluria; ↑urinary β_2-microglobulin
Post-renal	Obstruction of urine flow from a single kidney, both kidneys or urethra	Stones, tumors or posterior urethral valve in males	Dilation of renal collecting system on renal ultrasound
Vascular	↓perfusion to kidneys	Renal artery embolus or renal vein thrombosis	↓renal blood flow on nuclear renal scan

- ✓ Clinical features: lethargy, nausea and vomiting, respiratory distress, hypertension and seizures. Notice that oliguria in children is defined as urine output < 1ml/kg/hour.
- ✓ Evaluation:

Laboratory investigations	Serum electrolytes, urea, creatinine and urinalysis
Imaging studies	Renal/pelvic ultrasound; nuclear renal scan

- ✓ Management:
  - ❖ Treatment of the underlying cause.
  - ❖ If there is hypovolemia, intravascular volume should be restored first and then total fluid intake will be restricted to patient's insensible losses and urine/stool replacement.
  - ❖ Restriction or protein intake.
  - ❖ Dialysis (peritoneal or hemodialysis) when conservative management fails.

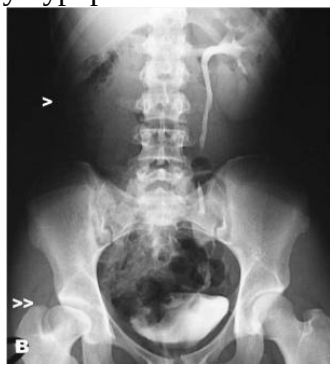
- **Chronic renal insufficiency and End-Stage Renal Disease (ESRD):**
  - ✓ The most common causes include the following: glomerular diseases, congenital renal disease, reflux nephropathy, HUS, cystic kidney disease.
  - ✓ Clinical features: lethargy, anemia, rickets, polyuria/polydipsia, short stature and FTT.
  - ✓ Management:

Medical	• Restriction of Na, K and proteins. • Monitoring serum electrolytes, urea and creatinine. • Vitamin D analogs, iron and recombinant erythropoietin. • Blood pressure monitoring and management.
Dialysis (when GFR = 5-10%)	• Peritoneal dialysis is preferred in infants and children because hemodialysis requires vascular access via arteriovenous fistula. • Kidney transplantation is the preferred treatment for children with ESRD but it requires life-long immunosuppression which predisposes the patient to increased risk of infections. • Most common causes of transplantation loss: acute and chronic rejection, non-compliance with medications, technical problems during surgery or recurrent disease

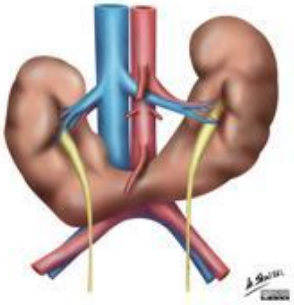
- **Structural and urologic abnormalities:**
  - **Congenital obstruction**: it is sub-classified into 3 types
    - ✓ Ureteropelvic obstruction: kinks, fibrous bands or overlying aberrant blood vessel.
    - ✓ Ureterovesicle obstruction: abnormal insertion of ureter into urinary bladder wall or ureterocele.

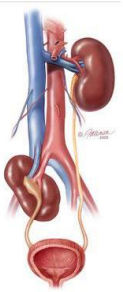
- ✓ Bladder outlet obstruction: posterior urethral valve in males or polyps.
  Notice that bilateral lesions causing severe impairment of renal function will result in maternal history of oligohydramnios which leads to pulmonary hypoplasia in the baby that can be lethal!

- **Acquired obstruction**: they occur due to tumors or stones.
- **Renal abnormalities:**
  - ✓ Renal agenesis: there is no formation of kidney due to failure in development of mesonephric duct.
    - ❖ *Unilateral agenesis*: 0.1-0.2% of children.
    - ❖ *Bilateral agenesis*: very rare! It usually results in infant death due to pulmonary hypoplasia.



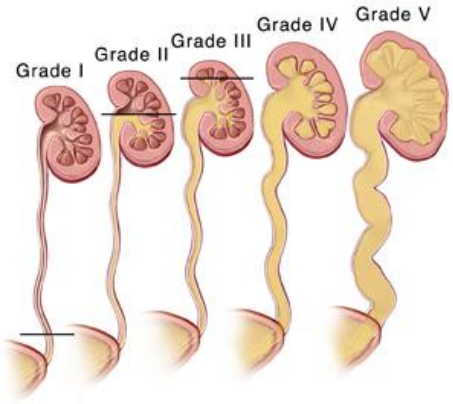
  - ✓ Renal dysplasia: more common than renal agenesis.
    - ❖ *Pathological*: abnormal structure of the kidney.
    - ❖ *Functional*: concentrating defects, renal tubular acidosis or varying degrees of renal insufficiency.
      Notice that the most common abdominal mass discovered in neonates is multicystic dysplastic kidney which is usually associated with atretic ureter and if it is severe it will cause death due to pulmonary hypoplasia.
  - ✓ Horseshoe kidney: fusion of lower poles of both kidneys and when this kidney ascends from the pelvis it will be stopped by the inferior mesenteric artery.
  - ✓ Renal ectopia: kidney located outside the renal fossa (such as in the pelvis).



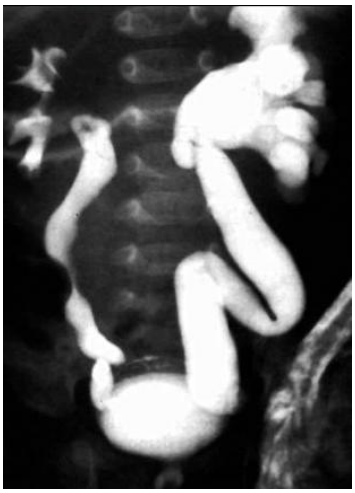


- **Vesicoureteral reflux (VUR):**
  - ✓ It is defined as retrograde flow of urine from urinary bladder back into the ureter and renal collecting system thus causing hydronephrosis and predisposing to infections (pyelonephritis).
  - ✓ Cause: the most common cause being abnormal insertion of ureter into urinary bladder wall. Notice that VUR can also be inherited in an AD fashion.
  - ✓ Classification: there are 5 grades of VSR

Grade-I	Reflux into distal ureter
Grade-II	Reflux extends to renal pelvis and calyces WITHOUT dilation
Grade-III	Reflux extends to renal pelvis and calyces WITH dilation
Grade-IV	More dilation with clubbing of calyces
Grade-V	Severe clubbing of calyces with tortuosity of ureter

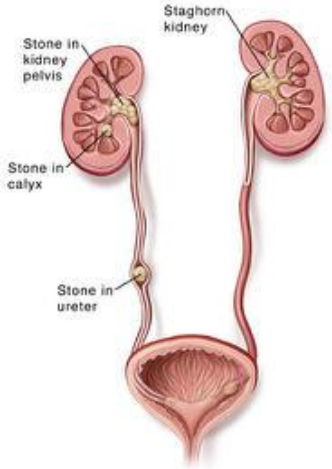


- ✓ Diagnosis: Voiding Cysto-Urethro-Gram (VCUG) → in which a dye will be introduced into the urinary bladder through a catheter and then there will be imaging under fluoroscopy during filling of bladder and voiding to watch if reflux or urine occurs or not.



- ✓ Management:
  - ❖ Prophylactic low-dose antibiotics to prevent UTIs.
  - ❖ *Low-grades VUR* → spontaneous resolution.
  - ❖ *High-grades VUR (IV and V)* → surgical re-implantation of ureters can be considered.

- **Renal stones:**



- **They are uncommon in children but the most common stones seen in childhood include the following:**
  - ✓ Calcium salts: cause by hypercalciuria, hyperoxaluria or hyperparathyroidism.
  - ✓ Uric acid: caused by hyperuricosuria.
  - ✓ Cysteine: caused by cyteinuria (AR).
  - ✓ Magnesium-ammonium-phosphate.
- **Clinical features**: Colicky abdominal/flank pain; nausea and vomiting; gross/microscopic hematuria.
- **Diagnosis:**

Laboratory investigations	Serum electrolytes, urea, creatinine, calcium, PTH, phosphorus, uric acid and urinalysis (to rule out presence of hematuria)
Imaging studies	KUB, renal ultrasound and non-contrast spiral CT-scan
Stone fragment analysis	If a fragment is collected

- **Management:**
  - ✓ Pain relief: by using NSAIDs or opioids.
  - ✓ Hydration with use of antibiotics if UTI is associated.

- ✓ Expulsion therapy: stimulating spontaneous expulsion of the stone by dilating ureters through α-adrenergic blockers.
- ✓ Lithotripsy: for stones located near the renal pelvis.
- ✓ Ureteroscopic surgery: for stones located in lower ureter.

- **Urinary Tract Infection (UTI):**
  - **It is one of the most common bacterial infections in children.**
  - **Incidence:**

< 6 months of age	More common in uncircumcised males
> 6 months of age	More common in females

  - **Causative organisms**: E.coli (most common). **Others include**: Klebsiella, Proteus and Pseudomonas. They enter the urinary tract by ascending through urethra.
  - **Clinical features:**

Pyelonephritis	Fever and flank pain
Cystitis	Absent/low-grade fever and urinary symptoms (dysuria, urgency and frequency)

  - **Diagnosis**: through urinalysis (detecting leukocytes and leukocyte esterase) and urine culture (gold-standard). Criteria to diagnose with urine culture:

Suprapubic aspiration (in neonates and infants)	Any growth
Sterile urethral catheterization (in neonates and infants)	≥10,000 colonies
Clean-catch method (in older children)	≥50,000-100,000 colonies

  - **Management**: empiric antibiotic therapy.