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① Causes of AKI :- 1- Pre-renal 2- Renal 3- Post-renal.

Medstudy

* Pre-renal AKI is due to a decrease in effective renal blood flow. Causes of decreased flow are :-

1- Severe intravascular volume loss
2- Renal artery stenosis
3- Heart Failure
4- Cirrhosis
5- Nephrotic syndrome
6- Drugs - esp. diuretics, NSAIDs, ACEi, ILZ
7- Vascular problems (large blood vessels).

* Post-renal AKI is typically due to bladder outlet obstruction, bilateral urethral obstruction (v. rare).

* Renal Intrinsic or intrarenal AKI causes include :-

1- Acute tubular necrosis (ATN), which can be ischemic or nephrotoxic.
   * ATN is the most common cause of AKI.
2- Glomerular damage
3- Acute interstitial nephritis.

* ATN is caused by ↓ renal perfusion, eg. post surgical, trauma, sepsis, burns, Myoglobinuria, hemoglobinuria, Heavy metals, contrast dyes, Drugs.

Drugs :- Amphotericin B, Aminoglycosides (proximal tube damage)
Cisplastin (hypomagnesemia), Cyclosporine, IV contrast material,
↳ More likely to cause interstitial nephritis than glomerular damage.

~~② UTI investigations :-~~

② Atypical UTI :- ① Non-E. coli UTI ② Rising serum Cr. ③ Ill looking pt
④ Palpable abdominal mass.

③ UTI investigations:- (Urinanalysis, Urine culture, Radioimaging). ③

- Urinanalysis → Microscopy, Dipstick (leukocyte esterase test), Nitrite test.

- Urine culture → Supra-pubic sample if <6m,
  urine bag collection if infant.
  Catheter sample at any age
  Midstream sample in older children.

  → Suprapubic → Any growth is significant in a suprapubic sample.
  Catheter sample → $>10^3$/ml
  Midstream sample → $>10^5$/ml
  Bag sample $\geq 10^5$/ml (not v. reliable, ↑↑ false positive rate) →

- Radioimaging:- US, KUB, MCUG, DMSA

  ↳ Purpose of imaging is to detect anatomic abnormality, active renal involvement, assess renal function.

  ↳ Perform U/S if:- Young child <5-7 y/o, boy or girl,
  KUB us First UTI, Recurrent UTI, atypical UTI.

  ↳ MCUG is done to establish the presence; degree of VUR.

  ↳ DMSA scan for identifying areas of scars or ↓ uptake.

Note:- If abnormal U/S → do MCUG.

Note:- IVU is NOT done! (Intravenous urogram!)

Note:- Complications of UTI include:-
Renal scars, especially in young children.
FTT, Hypertension; CKD if Recurrent UTI
Renal stones (mixed stones, struvite stones).

④ Complications of CKD:-

↳ Main causes of CKD in children:- Obstructive uropathy, reflux nephropathy, hypoplastic/Dysplastic kidneys, PCKD, GN (FSGN).

1) Hypertension 2- Anemia 3- Poor growth 4- Bone-calcium metabolic derang- 7- ↑GH.
5- Nerve damage 6- Cognitive delays in young children.

Dialysis indications:-

1) Symptomatic uremia 2) Stage 5 CKD (GFR < 15 ml/min / 1.73 m²)
3) Progressive renal osteodystrophy despite optimal medical therapy

Hyperkalemia w/ ECG changes, Tumor lysis syndrome, hypo/hypernatremia, fluid overload: Pulmonary edema refractory to medical therapy.

Comp Nephrotic Syndrome.

1) Heavy proteinuria >1000 mg/m²/day or random urine: Prot:Cr >2
2) Hypoalbuminemia w/ serum albumin <3 mg/dL
3) Edema
4) Hypercholesterolemia

cellular casts are absent.

Complications of nephrotic syndrome:-

1) Infection → due to Ig deficiency → Primary peritonitis occurs in 2-6%, most infections are due to strep. pneumonia.
2) Thrombotic disease → due to hypercoagulable state from thrombocytosis & hemostatic abnormalities.
3) Edema and anasarca → Caused by hypoalbuminemia and primary sodium retention.
4) Renal insufficiency → from hypovolemia or AKI.
5) Hypovolemia → causes peripheral vasoconstriction ↓ GFR.
   ↳ occurs esp w/ MCD
6) Hyperlipidemia → children are at early risk of CVD.

If glucocorticoid therapy → monitor growth and cataract formation.

Nephrotic syndromes include:- Minimal change disease, Focal segmental Glomerulosclerosis, Membranous nephropathy; They have normal complement levels.

* Minimal Change Disease:- MCC of nephrotic syndrome in childhood.
  - Primary mostly!/Idiopathic
  - 2ndary causes include:- Hodgkin's lymphoma, NSAIDs, systemic immune-mediated diseases.
  - Early morning eyelid swelling.
  - Microscopic hematuria occurs in about 20% of pts (But)

Macroscopic hematuria is vvv rare!!

Complement levels are normal.

Renal biopsy is not indicated! Except if:- <1y/o, macroscopic hematuria, hypocomplementemia, steroid resistant, frequent relapses, renal failure not due to volume contraction.

Tt of MCD:- Supportive: symptomatic.

Prednisone 2mg/kg/day, max dose 80mg

Death in MCD is mostly due to infections.

70-80% have remission after 8-10y/o.

Note:- F SGS needs kidney biopsy to confirm diagnosis.

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Nephritic Syndrome

* Hematuria, variable proteinuria, edema, htn ...

(Hypocomplementemia frequently occurs in Post infectious glomerulonephritis, & chronic glomerulonephritis caused membranoproliferative GN.+

* Acute Postinfectious glomerulonephritis

  - to Strep pyogenes, (B-hemolytic strep).
  - Antibiotic therapy does not prevent acute GN! It prevents Rheumatic fever & the spread of the nephritogenic strain to others.
  - Usually afebrile, latency period btw pharyngitis & PIGN is about 1-2wks.
  - low C3.

IgA nephropathy → Berger disease → is the most common cause of gross hematuria, ∴ most common cause of ✱ primary chronic glomerulopathy

↳ Recurrent episodes of painless gross hematuria, usually during upper respiratory infections. ± microhematuria w/ mild proteinuria initially

It may develop into rapidly progressive GN or nephrotic syndrome.

- They have normal C3, unlike post streptococcal GN.
- Definitive dx requires kidney biopsy.

Kidney biopsy indications:- impaired renal function, hypertension, serologic abnormalities, multiple episodes of recurrent gross hematuria, or significant proteinuria >1g/24hr.

HSP (Henoch-Schönlein-Purpura) Nephritis:-

- Is a systemic vasculitis with 4 classic features:-
  1) Purpuric rash, esp. over the buttocks, abdomen ∴ lower extremities.
  2) Abdominal pain.
  3) Arthralgias
  4) GN w/ IgA deposition.

Membranoproliferative GN:-

- thickening of the glomerular basement membrane ∴ hypercellularity
- V. Low C3 Levels.

Rapidly progressive (crescentic) GN:-

- The acute presentation of a number of aggressive glomerular disorders.
- Majority progress to ESRD if not aggressively treated ∴ hospitalized.
  1) Anti-GBM disease 2) Immune complex nephritis 3) Pauci-immune
- Clinical features:- Gross hematuria, edema, anemia, hypertension.

Lupus nephritis :-

Immune complex mediated. Most children present w/ active nephritis instead of rash, joint complaints

Nephrotic / Nephritic → C3 is always normal in :-

1) Minimal change disease → loss of foot processes.
2) Focal sclerosis → sclerosis
3) Membranous nephropathy → Subepithelial deposits
4) Diabetic nephropathy
5) Amyloid nephropathy } large nodular hyaline masses.

Nephritic (occasionally causes nephrotic urine) :-

1) Post infectious GN → hypocomplimentemia.
2) Membranoproliferative GN → GBM changes + cell proliferation.
3) Rapidly progressive GN
4) Mesangial proliferative GN
5) IgA nephropathy

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HUS :- Triad of 1) Microangiopathic hemolytic anemia
2) Thrombocytopenia
3) Acute renal failure.

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Renal Tubular Acidosis.

Is a disease that occurs when the kidneys fail to excrete acids into the urine, which causes a persons blood to remain too acidic. (normal anion gap metabolic acidosis)

RTA type (1) → Distal renal tubular acidosis → inability of α intercalated cells to secrete $H^+$ → no new $HCO_3^-$ is generated → metabolic acidosis.

Urine pH > 5.5, Serum $K^+$ ↓, ↑ risk of $Ca_3PO_4$ stones.

RTA type 2 → Proximal tubular acidosis → defect in PCT $HCO_3^-$ reabsorption → ↑ excretion of $HCO_3^-$ in urine → metabolic acidosis.

Urine pH < 5.5, ↓ Serum $K^+$ (↑ risk of hypophosphatemic rickets)

RTA type 4 → Hypoaldosteronism or aldosterone resistance; hyperkalemia → ↓ $NH_3$ synthesis in PCT, ↓ $NH_4^+$ excretion

Urine pH < 5.5, $K^+$ ↑ !

so cause hypokalemia

autosomal recessive, hypokalemic metabolic alkalosis ± salt wasting w/o htn. (7)
Bartter syndrome → No changes in bp, ↑plasma renin, ↑aldosterone, ↑urine $Ca^{2+}$.

Diabetes Insipidus → Intense thirst with polyuria with inability to concentrate urine due to lack of ADH (central) or failure of response to circulating ADH (nephrogenic).

Central DI improves w/ administration of ADH analogue, unlike nephrogenic DI. Central DI is ttt w/ desmopressin.

SIADH → Characterized by excessive free water retention, euvolemic hyponatremia w/ continued urinary $Na^+$ excretion. Urine osmolality > serum osmolality

The most common cause of proteinuria → Benign orthostatic proteinuria.

Microalbuminuria is an indication of early glomerular injury including diabetic nephropathy. Random urine albumin : Cr.

Hyperkalemia:- ↑ cell turnover eg. tumor lysis syndrome, rhabdomyolysis, acute leukemia.
Trimethoprim
Renal failure (AKI ; CKD) due to poor urine flow.
Type 4 RTA.

If chronic hyperkalemia → most pts are asymptomatic.

C/P :- Significant weakness or paralysis, conduction abnormalities, arrhythmias :- ECG changes include :- (in sequence) if $K^+$ ↑ 7 meq/l
1) Peaked T wave : short QT interval
2) Progressive lengthening of PR & QRS intervals
3) Loss of P wave : QRS widening
4) Ventricular fibrillation.

Ttt of hyperkalemia:-
1) IV Ca gluconate 2) Insulin w/ glucose 3) So $NaCO_3$ if acidosis
4) Loop diuretics, 5) Dialysis 6) Resin binders but never as monotherapy!

(8)