* blood supply → from renal artrey , Fx → clean the body and get waste

Labs: blood, urin , imaging , Biopseis

blood → CBC : RBC , WBC , Pt

Chemistry : Complete metabolic Panel

① ↳ blood sugars → Fasting blood glucose < 100 mg/dL

② electrolytes → Na = 135 – 145 (التركيز بالدم)

Ka → 3.5 – 5.3   CL → 90 – 110   Ca 8.4 – 10.2

mg < 2 , Po4 < 3

③ KFT → A → createnin : .6 – 1.2

blood urea nitrogen < 20

* CK → createn kinase (↳ enzyme) → Po4 بيشيل او bs   Fx : creatine phosphate (every molecule in muscle)

لا يشبه ATP وهو أقوى منه

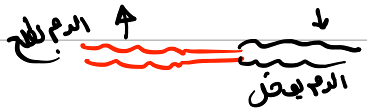
→ creatine → creatinin (waste product) → blood → kidney → excread into urine

muscle in body . يعبر عن الـ نشاط الـ عضل أقل

Bun , urea Produced b Liver from (Protene degradation) → excretead by urin (لعم)

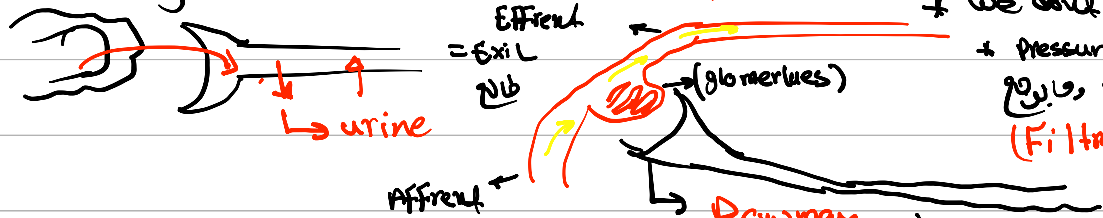
From gi through Portal circulation

* every kidney has 1/2 million nephron



= capillary , tubes → Filtration of plasma.

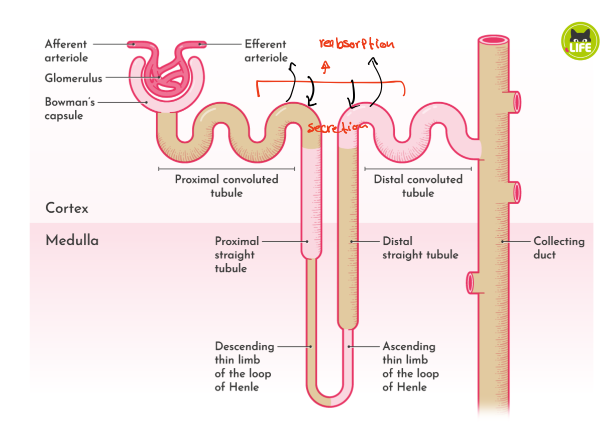
* we don't have venule
* Pressur high at ALL
عشان هيرد يطلع ويرجع
(Filtration)



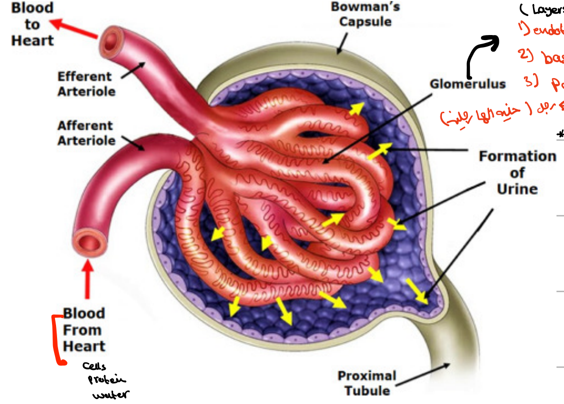
Bowman capsule (Filtration بسمي الـ)

↳ Proximal convulated tubule ↓ ملفوف

+ filtration → reabsorption → secretion → excretion



(AKI)



(Layers)
1) endothelium
2) basement layer } → filter
3) Podo cyte
(خلايا عليها رجول)

* blood from glomerulus to bowman capsule through this filter
↳ allow to plasma
prevent cells, proteins

cells, protein, water

glomular Filtration rate (GFR)

* CO : 5 L/min → 1/4 to kidneys (renal blood flow) 1.2 → Filter (90–120 mL/min) . كل 5 دقائق

GFR = 90 – 120 mL/min / 1.73 m² BSA   * GFR بزيادة وزن ما بزيد

* Body surface area

(safe) مؤثر   poor (substance C)
بنعمل تحليل → give the Pt a substance (Inulin) → into the blood

need 24 urin collection not practicle ما بنستخدمها الا equation

the filtrate to urin * without absorption or secretion (تطلع كما هي)

(produced from muscle)
* in hospital → rely on (createnine) →*

الجسم مش عم يطلعها

if kidney Disease → ↓ GFR ↑ createnin → لما يكون فيه نقص بالـ GFR

* equation → to (estimate) تقدير GFR

Cockcroft-Gault Formula for Estimating Creatinine Clearance

$$CrCl\ (mL/min) = \frac{(140 - age) \times \text{Lean Body Weight (kg)}}{\text{Serum Creatinine (mg/dL)} \times 72} \ (\times\ 0.85 \text{ if female})$$

(140 – 40) × 70
1 × 72
→ 7000 / 72 →
تقريبا أقل من 100 normal

Defention of acute kidney injury

* MDRD   if GFR ↓ → Cr ↑, BuN ↑

CKD-EPi   (0.5 mL/hr/kg) ← ↓ urin out put

60 k → 30 mL /hr → 24 × 30 mL = (لازم كم يطلع باليوم)

AKI = ↓ GFR acutly within days (1–7)

Diagnosed if creatine ↑ in 48 hours

urin output ↓ in 6 hours

* 40 Female → for elective cholecystectomy → cr : 1 mg/dL (mL)
1 week later → cr = 1.7
if ≥ 1.5 times or ↑ ≥ 0.3 ↗   (AKI) هاد يعني يصير 1.3 – 1.5 بعد يعتبر عالي خلال يومين هاد

base line → 0.5 ——→ 0.8 / 0.75 دوا لها بنصير الحل AKI ما

Causes of AKI

Pre renal → ↓ renal blood flow (1.2 L/m)

renal → kidney Disease

Post renal → obstruction of urin out flow

Pre renal → ↓ RBF

*1) ↓ CO (HF) → cardio renal syndrome

2) ↓ effectiv volume of blood
↳ liver cirrhosis ↳ nephrotic syndrome

3) renal vascular disease
* obstruct renal vessel :
Drugs → constrict ↓
1) NSAIDs ; constrict → afferent arterioles
2) ACEI/ARB → ↑ K + AKI → constrict efferent
3) Tacrolemus / cyclosporin : immuno suppresent in Transplant Pt

4) shock : ↓ BP → ↓ RBF
↳ Hypovolmeic : hemorrage
↳ septic : infection
↳ cardiogenic : from sever HF
↳ nurogenic

Hx : cardiac disease : edema / dyspnea (HF)

* تناخذ مسكنات ؟ شو ادوية الضغط ؟

Liver DxP
↗ ↓ BP → shock
PE → vitals , volum status

hypo →
dry mucos membran
conjectiva / lips
↓ BP ↑ HR Prolonged capillary refill
↓ loc

normo

hyper
edema
LL Ascites, Plural effusion, Periorbibal
Triad of edema
1) HF 2) cirrhoses
3) nephrotic

From PE

* How to assess volume status better?

1) I/O best way
* hyper volemia
2) body weight : * 65 → 75 → water retention
↳ (objective way)   week
← Dehydration

Labs :

1) cr ↑ BuN ↑
when ↓ RBF → the kidney try to compensate through
2 hormone : Aldosteron / ADH → increase blood volume

* Aldosteron → ↑ Na reabsorption
ADH → ↑ water, urea ↓

* Pre renal

↑ Bun > ↑ cr → لذا ال ADH يرفع ال urea

Bun / cr → 20 / في صحيح ال 40 بيتكون عادي عند 20

urin, Na ↓ < 10 / Fractional Excretion Na NL 2%

(FENA) النسبة التي يطلعها بتعرف قليل
في AKI لذا أمن بخلص إنه يتعاملها ما بطلعه

* urin concentration ↳ لذا بيتكون عادي لأن الجسم بجي احتفاظ الماء

↓ Na ↓ urea ↓ H_2O

PRE Renal

Bun / cr	↑	> 20	ADH
U Na	↓	< 10	Aldo
FENa	↓	< 1%	Aldo
osmarllity	↑	> 350	both mainly (ADH)

RENal

كل الامور عكس

↓ ↑ (>20) ↑ (>2%) ↓

bc2 the kidney dont respond to ADH / Aldosteron

* causes :

1) glomerula 2) tubular 3) interstial between tubules.

1) glomerular :

↳ Fx : Filter the blood : remove cells and Protiens

Dys fx : * Proteinuria (Protein) → لذا في glomeral ضعيف
* hematuria (cells) معيوبة فيه

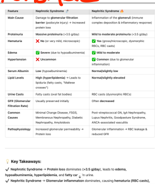
AKI → glomerulonephritis : acute infection
((GN) (nephritic syndrom)) (NS)
Proteinuria

* Syndrom → HEHO

(dysmorphic RBC)
Cola / urin

* hematuria : appear Like RBC cast (NS) مش موجود في
عدم وجودها لا يعني نفي ال NS بس وجودها يؤكد.

* Edema → hyper volemia → LL / Periorbital
↳ ↓ GFR ↓ u/o ↑ Na + water retention



* HTN → hyper volemia

* oliguria → ↓ u/o (AKI)

* Protein uria →

Causes :

Pharyngitis / skin infection → the 2-4 weeks later can cause
group (A) → strep Pyogens (PS GN) 5-15 (female)

mcc in children : Post streptococal GN

Adults : IgA nephropathy

Case : 12 y girl, eye swelling * Cola urin
* Periorbital edema

Labs → ① urin anylasis → RBC cast

u/o → Adult → 0.5 - 1 mL / h / kg
children → 1 - 1.5 mL / h / kg

* Proteinuria → Dipstick

more accurate : 24 urin collection (الحجم)

NL : < 150 mg / day / > 150 → Proteinuri

* faster wey → urin Protein creatinine ratio.
↓ 24 urin Protine. بنعمل هاي Spot (بيجيبوها عادة) بنقيس هاي → spot ونقسمهم على بعض.

.6 → 600 mg / day NL < 0.15
.1 → 100 mg / day

estimate protine in just one sample.

PSGN Diagnosis :

Confirm GAS infection : by history if has Pharyngitis or skin infection (2-4) weeks

Labs → ① anti streptolycin O (Anti body)

② biopsy of the kidney (if we not sure)

Tx → fluids to ↑ u/o
Anti hypertensive
blood if enemia (due to hematuria)

nephritic criteria:

1) urin analysis → 1) cast / dysmorphic (hematuria)

2) Edema / HTN / oliguria / Proteinuria
best test: upcr

Adult → IGA nephropathy → occure After URTi , gastro enterites within (2-4) Days , They already have Phyrigites.

* Bx → IGA deposition

Glomular disease

(RRT) ← renal replacement Therapy . غسيل/زراعة
days
GN
→ Rapidaly Progressive GN (RPGN) normal → End stage renal Dx
→ nephitic syndrome → supportive (Few weeks self limiting) وضعه بيصير منيح لحاله

* RPGN → to 3 Types depend on (Biopsy)

Biopsy: (crescentic GN)
على حسب
* immune fluroscence / → Ab → ملون

1) (Linear) (GBM) Bm على طول
Antibodies against (Glomural base ment membrane)

2) (granular)
immune complex deposition

3) (nothing)
Pauci immune : no thing (no 1/2)

→ (Ab + Ag) in blood → انفش بالغلوميرولي
trigger to inflammation / تتجمع على الـ glomurloes ، فتعمل
→ (inflammation) / يصير جسم بيحط يعمل التهاب

2 immune complexes:
→ PsGN
→ SLE
مثال 1

→ immune complex disease.

PSGN: bacteria GAs → Pharygnitis / Skin infection
↓
تتغير rapidly
when WBC fight GAs → release Antigen catch by
بتصير benign
immunity → causes immune complex then filtered by kidney.
nephrogenetic Strain GAs
مش كل البكتيريا بتعمل هاد، فيه

* IgA nephropathy (rapidly)

URTi / gastro enteritis → Protein Ag + IgA (Viral)
(IC) ما بنتج immune complex

PsGN	AgN
bacteria	viral
2-4 weeks Post phyrangitis	2-4 days syn pharngitis
IgG - Ag	IgA - Ag
children	Adult

1 Anti GBM
→ GN alone → Hematuria / AKI
→ Lung involment → effect Alveol membrane
Hematuria / Hemoptysis
(goodpasture syndrome)

2 ICD
→ PsGN
→ SLE

3 Pauci → vasculitis (ANCA)
* affect lung * Hemoptysis * hematuria.

tubular → renal AKI

* acute tubular necrosis (ATN)

2 causes
→ mcc
1) ischemia ↓RBF 2) toxic material

* tubule The first Part necrosis → the hieghst demand →

↓ RBF → Pre renal (تأخر، علاجه سوائل) → 1-2 days → renal → waiting for healing

ATN: (proximal) أول اشي بتأثر

doing epithelial cast → muddy brown cast (تتجمع)
(ATN بتشخص)

2) toxic materials: Drugs / toxins

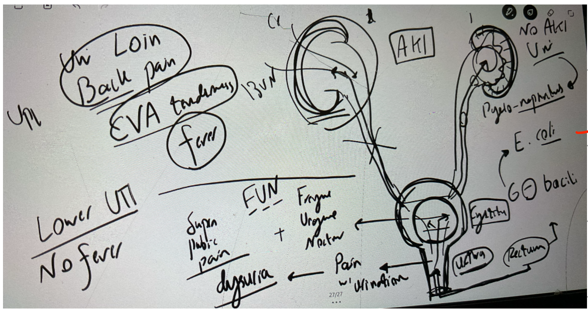
Abx: Aminoglycoside → genta mycin / Vanco mycin
Loop diauretics : Lasix : Furosmide
chemo: cis platine / Carbo Plein
Anti fungal : ampho tericin B
PPI → omeprazole.
Contrast material / NSAIDs

* heavy metals
* Proteins: multiple myleoma
* Hb
myoglobin: rabdomylosis

* interstitial nephritis: التهاب بكل التعليقة

1) bacterial → Pyleonephritis → Pelvis → urin البكتيريا بتوصل عن طريق الـ (ascending inf) pelvis لل

2) allergic interstial nephritis (AIN)
both Affected → ↑ of AKI
bi lateral
Fever / Loin Pain / CVA tenderness bi/uni ⊕ Articarial Rash. منقطة ودائرية حمراء + itchy



* Costo – Vertibre Angle Tenderness
renal Angle بنص الظهر
ابتعاد UTI

Labs	(kidney) Pyleo	AIN
–	↓ AKI	↑ AKI
WBC	neutrophils	Esinophil > 4%
ESR, CRP	↑	↑

لانه عم ما دخل يصير بالعلاج

1–2 (hours)

UA → detect gram [–ve] [+ve] bacteria

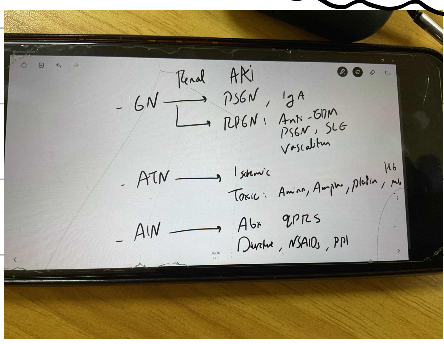
1) gross (color) → yellow (NL)
RBC الـ ← ممكن لما يكون فيه دم تجي حمراء (rabdomylosis) يصير فيه
enzyme produce by WBC infection inflammation

2) dipstick — not accurate
Protein → NL Absent, Heme, Hb, myoglobin, sugars
↓
UPCR
① Lukocyte estrase
② nitrite
sign of gram –ve bacteria (infection just)
<3 hPF

3) microscopy
→ cells: RBC, WBC, Epithelial
<5 hPF
↳ casts ↳ RBC, muddy
GN ATN
↳ crystals

[1–5 day]
urin culture → what the Type of bacteria

Abx mcc of AIN → P Q R S
→ quinolone
→ Rifampin
→ sulfonamide
↳ Pencillin + cephalosporine



Post renal AKI

* urinary tract diseases

* obstruction
↳ should affect both kidney to cause AKI
* stone / tumer, cervical cancer
↳ Benign Prostate hyper plasia

* reflux → when blood goes back → increas the Pressure inside the kidney causes destruction (scaring)

* anurea + AKI → Acute urin retention.
* Lower Abd Pain due to distention bladder
Tx: (cath)

Chronic kidney disease.

> 3 Months

Definition:
1) impired kidney structure
2) " " function
3 ↓ GFR (most common)

* structure: Poly cystic kidney disease with NL GFR + function
* Function: Proteinurea + NL GFR + NL structure → nephrotic
* GFR: 90–120 mL/min/1.73

Stages:
1 → GFR > 90 with have structural or functional impai
2 → 60–90
3 → 60–30
4 → 30–15
5 → < 15 ESRD.

* createnin, UO ↳ AKI
CKD → GFR

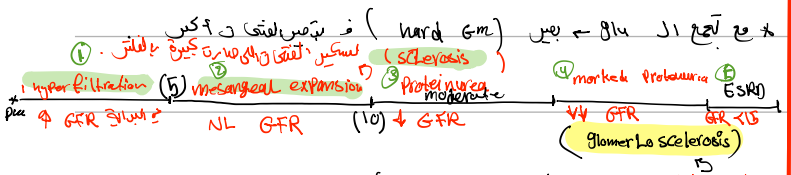
* what the causes.
most common cause → 1) (DM) 2) HTN
other causes → 1) glomurulopathy / 2) amylodosis.
3) Heredatry 4) Analgesic nephro pathy 5) congenital
6– (sickl cell)

Diabetec nephro pathy

due to micro vascular complication of (DM)
affect small vessel → diabetic
retino pathy *
nuropathy *
* nephropathy → in glomulai

have ↑ blood glucose → react with GBM → glomuler damage
due to hyperglycemia

* Stages → depend how much duration → nephropathy



(mesangial cell) glomerules ← thickning

From 3 cause HTN / Proteinurea / CKD.

DM → CKD → Proteinuria → (toxic for tubule)
CKD ↔ HTN → vascular damage

so if we treat the DM CKD keep progress to reach ESRD

Tx → DM → life style modefecation → ↓ glucose (metformin / insulin)
target < 130/80.

(Proteinurea / HTN → ACEI / ARBS) ↓ Protein intake (<2g / day)

Diagnosis of DM nephropathy → (through stages)

1) hyperfiltration → 0-5 years

2) mesangial expansion
(NL <30) (30-300 mg/dL)
micro Albuminuria -ve in DIPSTICK / +ve Albumin creatinine ratio

3) moderate Proteinuria → from here we diagnose (not before)

4) Sever " (>300 mg/dL) Frank Proteinuria

5) ESRD

screening → 1) Albumin creatinine Ratio >30 micro albuminuria
when ? After (5) years of developing (not diagnosis)

in type 1) Pt complains (DKA) → After 5y.

Tx → we don't know → so immediately doing screening

Complication of CKD

1) HTN → ↓ GFR
↳ ACEI / ARBS
HTN → CKD
CKD ↔ HTN

2) Anemia of kidney disease → ↓ EPO (80% From kidney) normocytic Anemia
↳ Target → 11 / start Tx if <9 Tx ↳ replace EPO → (Im) (Epoten Alpha)
↳ once reach we stop EPO → bc EPO cause HTN and bleeding.

3) IDA → due to blood less / ↓ intake Tx: Iron

4) renal Bone disease

5) uremia

nephrotic syndrome → glomuriopathy → damage to filtration membrane.

GFR can be normal

* NO inflammation * No hematuria , NO HTN , No oliguria

main problem is ① Proteinurea → dipstick → Part of urin analysis. Use less
↳ 24 hour urin collection / UPCR → NL <150 mg/day

nephrotic range // = 7/3.5 gl day = 3500 mg/day

② Edema → ↓ Albumine → ↓ oncotic Pressure

③ Hypo Albuminuria <3.5 g/dL

④ ↓ immunoglobulins (Anti body) → immuno defiency → infection

⑤ ↓ anti coagulation → like Anti thrombin III → Thrombosis (Hypercoagulable)
DUT, PE, MI

⑥ → Lipiduria → Hyperlipidemia → (MI → Death)

Lipids → Cholestrol, Triglecryde → not water soluble

Can't move in blood alone → to transfer the Proteine we

Should use Protein around it (APO LIPO protein) + lipids = LIPOprotein (from liver)

like LDL / HDL → so Loss of Lipo protein → cause ⑥ lipiduria

Lipoproteinemia → so liver compensate and ↑ Production of LipoProteine → so increases more than normal

Causes →

Children → minimal change disease

Adult → membranous Glomerulopathy (Caucasian)

African Adult → focal segmental glomerulosclerosis

minimal change disease.

Called this due to Bx on Light microscope there is minimal change.
Light → NL
IF → normal
Em → damage (Loss of Podocyte Process)

1 old boy → Puffy eyes.

Hx = infection / edema /

blood test → Lipid ↑, ↓ Albumin <3.5 (3.5-5) / cr >Bun (normal 1/1)

↓ Ig, coagulation

urin test → UPCR >3.5 / Lipid

Tx → steroids → due to auto immune

Causes 1) Primary

2) secondary → 1) infection. 2) Drugs (NSAIDs) 3) Cancer Hodgkin lymphoma

membranous →

Bx → Lm → thick basement

membrane

* Em → spike + dome → due to Anti body – Antigen immune complexes

IF → granular Appearence

causes → 1) Primary 2) secondary → * infections → Hepatitis, syphilis

* drug → Pencillamen, gold * Auto immune → SLE (immuno complex)

* cancers solid (Lung, kidney, breast)

Tx → depend on cause.

diffuse lesion → affect part of glomureles → thick base ment membrane

focal segmental glomerosclerosis

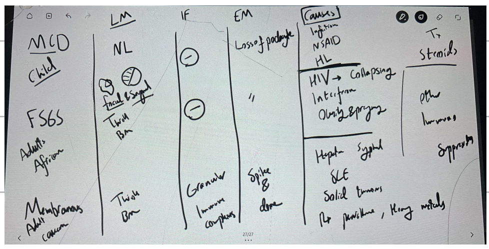
↳ Affect some glomurli not all (global) lesion

Lm → thick Bm / IF –ve / Em loss of foot Process.

Primary / secondary → 1) infection → HIV → collapsing glomuropathy (bad)

2) obesity, Pregnancy 3) drugs → interferone

Tx → ① steroids → Poor respone ② other immune suppressant



* membrano proliferative Glomuropathy MP GN
combination between (nephrotic + nephritic)

membarnous + prolifrative
thick Bm → nephrotic
PSGN → nephritic

(* Protenurea / AKI) [illegible]

causes → infection → hepatitis C

* auto immune → cryo globunemia

---

* Polycystic kidney disease (structural impaired) **ADPCK**

inherted. – multible cyst in both kidney – Appear like Abdominal masses

2 types.

infant: Autosomal reccesive, at birth + congental abnormal

Adult: Autosomal Dominant, 20–30, fx history, → HTN at early age, manifestation → kidney mass (cyst) → ct scan

cyst → Rupture (hemmorage) → cause pain / infection → obsess
↳ ↑ uTi + stones.

1) Liver cyst – Pancreatic cyst — Berry aneurysm → sub arachnoid hemmorage → cause Death

2) diverticulosis 3) mitral valve Prolapse → murmre

* Alport syndrome

defect in Collagen Type 4 which in GBM → glomeropathy → Lead to hematurea, Proteneuria (nephrotic) ESRD (↓ GFR) → RRT.

② Ear → sensi neural hearing Loss (Later to life)

3) Eye: Lens → visual Problems (على عمر معين) (سمع نظر بول hematurie)

mc → X Linked (male) through mother → so we ask about maternal family hx like uncle (الأخوال مش العم والأعمام)

Em: basket weave (متشقق مثل سلة القش.)

CKD manifestation

① Na + water retention → hyper volemia → edema due ↓ u/o
↳ give diuretics.

② electrolyte distrbences → ↑ hyper kalemia

3) Acid base disorders → metabolic Acidosis ليش ما يتطلع Acid
→ ESRD

4) uremia → BUN From Liver through protein use

* the wast Product From protein. / kidney cant get from body

urea it self not very toxin / but urea toxin very worse

(uremia → signs and symptoms not Labs)

↑ BUN → Azotemia (Azo → nitrogen) this is the lab

uremic syndrome → * accumulation of urea toxin lead to ESRD

gi → anorexia, nausea, vomiting → due to intake protein → تزيد هذه الاعراض فبقلل يعمهم. *

skin → hyper Pigmentation Earthy color زي لون التراب

Hematological: Platlet dys function Plt count is NL, but bleeding time Prolonged

* brusing and gi bleeding → IDA

④ CNS → ↓ concentration, letharyy, uremic encephalopathy = ↓ Loc, Peripheral nephropathy.

⑤ CVS → uremic Pericarditis

indication of dialysis

A acidosis resistance

O overload hyper volemia

U uremic → CNS / CVS

E electrolyte → ↑ K^+

I intoxication

Anemia of kidney disease

① ↓ EPO → EPO injection ② Anemia of chronic Dx

③ IDA

* **renal Bone Dx** – nephrogenic osteodystrophy

CKD → Bone PTH, Vit D

* the bone = collagen + minerals → Ca^{+2}, PO_4^-

vit D responsible of absorption Ca + Poy From GI and the go to bone by blood

PTH → activate osteoclast → releasing Ca + Poy from bone to the blood

↳ kidney → secret Poy & reabsob Ca^+

Vit D → ↑ Ca, Poy From GI

↳ cholecalciferol

From where? 1) From sun to skin 2) Food

Vit D —liver→ 25 OH Vit D (↑ half life)

25-hydroxylase

storage in liver

go to the kidney —1 alpha hydroxylase→ 1,25 di hydroxy (active form, work on GI)

when Poy → Ca^+ increase both ↓ Poy bind Ca^+ → bone

PTH → ↑ Ca – ↓ Poy → bones

Target of PTH maintain Ca^+ in blood (Target Place: kidney)

" " Vit D " " " bone (GIT)

The main problem in CKD is the Poy can excrete →

Hyper Phosphatemia

↳ ↑ Poy (in blood) – ① ↓ Ca^+

The kidney should Activate Vit D → ↓ vit D Activity (1-25 OH Vit D)

↓ Ca → due to 1) ↑ Poy 2) ↓ 1,25 vit D (in blood)

so the Tx → remove Poy – supply the Active form (1 alpha 25 vit D)

GI Through Poy binders

and surgery → give Ca

Poy binders Prevent Absorption of Poy → excrete in feces like severe uremic remove Poy by GI instead by kidney

↑ Poy → ↓ vit D → by hyper Para Hyroidism → bone resorption → ↑ Ca (Poy) → ↓ Ca

↳ osteomalacia, osteoporosis, fractures.

Tx → severe uremic, Ca, 1 & 25 OH vit D → (hyper Ca^+)

Acid–base disorders

Change in PH of blood – Normal PH → 7.35 – 7.45 (Acidosis / Alkalosis)

respiration = التنفس → burning nutrients (glucose) = CO_2 (acid) + H_2O → H_2CO_3 (base) — blood

H_2CO_3 → H^+ (Acid) + HCO_3^- (Base)

O_2 → (CO_2) ← acid

↳ gas → remove through expiration by lung

↳ Liquid " " urin by kidney

(if ↑ → PH ↑ Alkalin)

PH

* acidosis damage all enzymes → Death.

↓ expiration → respiratory Acidosis ↑ CO_2 ↓ PH

↓ urin → metabolic acid ↑ H^+ (↓ HCO_3^-, ↓ PH)

2 ways to maintain blood Acide

① excrete acide CO_2 & H^+ ② Produce base HCO_3^- by kidney

secretion of Acid

Production of base

Acidosis

① R.A → hypo ventilation

② MetA → (many) ① Kidney X ② ↑ acid production (Endogenous)

③ exogenous, toxication → 1) Aspirin 2) Alcohol.

* Acid → can donate H^+, AH^+ → H^+ + (A^-) Anion (acid)

Hydrochloride other acids.

① HCl ② AH

Cl^- chloride Anion

if we Add HCl → ↓ PH → acidosis

↑ Cl

↑ AH → ↓ PH → acidosis

– Cl^-

↑ anion

electrolyte

Cations $^+$ → Na^+ 140 (135–145)

Anions$^-$ → Cl^- → 110 , HCO_3^- → 22–26 , (24) Avg] 134
we have (6–12) un mesured anions.
* blood nutrient (+ =−)

if Pt has Na 135 CL 102 HCO_3^- 23 (135 − (102+23)) = 10 (anion gap)

PH → 7.31 → metabolic Acidosis → (نقص) ↓ HCO_3^- → ↓PH

طب لما عند المريض Na 140 واذا قلت HCO_3^- اذا لازم يرتفع اما CL او anion gap شي تاني فاذا زاد
so if the sorce of Acidosis is HCL → so CL ↑↑ , if HA → A gap ↑↑
Normal Anion gap metabolic Acidosis (NAGMA) لو زادت ال CL بسميها
hyperchloremic أو بعرفه باسم
لو زادت ال A gap بسميه (hyper Anion gap)

X5 male → Abd pain + blood per rectum → Acute
mesentric ischemia , PH = 7.21 (acidosis) CO_2 → 30 HCO_3^- 15 Low metabolic
Na 140 CL^- 100
(anino gap → 25 hyper.) (NL 12) * why ischemia cause acidosis →
bcz ↓O_2 in tissue / glycolysis → pyruvate → to mitochondria → by O_2 → ATP
if no O_2 → Lactic Acid.
Lactate + H^+ (anion) → so when ↑ cause (hyper AG)
ischemia اللي صار بسبب ال

HAGMA Causes

1) Lactic Acidosis → ischemia
2) keto acidosis → DM , starvation (keto diet) just fat *Alcohol
ketone bodies ← [* aceto Acetic Acid / B hydroxy butaric Acid / Aseton → not acid] bcz The Acidosis seizure → epilepsy
بزيد ال كلاح بيدخل مرض → ↑ ↑

3) intoxication → C Alcohol , Aspirin / Iron teblet / Isoniazed → (TB)
cause keto acidosis / lactic acidosis / acid (نضيف نسب)

* Alcohol → 1) Ethanole → Acetic Acid
acidosis ←
2) methanole → Formic // → toxic to optic nerve blinding
3) Ethylene glycole → (oxalic acid) oxalate stone toxic to kidney
4) Propylene glycol

4) uremia → ↑ urea → (CKD)

M → methanole
U → uremia
D → DKA
P → pro pylene glycol
I → iron / Isoniazid
L → Lactic Acidosis
E → Ethanol / Ethylen glycol
S → selycilate Acid (Aspirin)

normal Ag mA → (NAgmA)
1- renal tubular acidosis → RTA function of collecting system
* 1) Problem in excretion in (Acids) (collecting system) / reabsorption of HCO_3^- (PCT (convuluted))

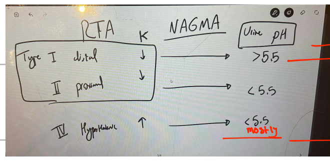
* Collecting system 2 Types of cell
1) Principle cell ↳ Na & water reabsorption ↳ K^+ excretion
2) α intercalated cell ↳ K^+ reabsorption, H^+ excretion

There is hormone activate 2 cell → Aldosteron → From cortex Adrenal gland.

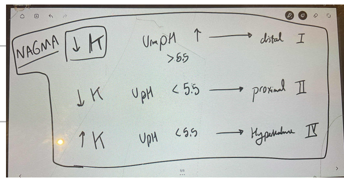
↑ Na ↓ K ↓ H^+ → doing Alklosis

* when there is α intercalated cell disease causes → (Acidosis Type I) distal (↓K) لو المريض عنده
[↓K]

* disease in Aldosterone → Acidosis Type 4 ↑K → (hyper kalemic RTA)
// in PCT → RTA Type II (↓K) (Proximal)



→ الجسم ما بطلع ال urine
→ Cant Absorbe → HCO_3^- → still in urin so ↑ PH
but Late ↓ HCO_3^- there is no more HCO_3^- out to urine
→ in more sever cause alkalin urin → لانه ساكت ↓↓ بحدوث ال α intercalated. ال Aldo نقص وتأخر بحيث



2) GIT → stomach → HCL Pancrease → base (HCO_3^-)

diarrhea → Loss HCO_3^- → Acidosis NAGMA + Low volume.

vomiting → Loss HCL → metabolic Alklosis.

metabolic Alkalosis.

↑PH , ↑HCO_3 , ↑ Aldosterone ← اهم سبب → α intercalated → ↓H^+ ; Principle → ↑Na , ↓K^+ → to ↑ blood volume (dehydration) (saline response)
↳ due to ↓ blood volume (volume contraction) → ↑RAAS → ↑ Na + H_2O → ↑ volume / ↓K^+ hypokalemia / ↓H^+ alklosis (contraction)
* disease giving pt NL (saline (fluid) (+K)

* Causes ↓ volume → 1) sweating 2) vomiting 3) excessive diuretics (over dose)
4) bleeding → but ischemia → cause lactic Acidosis (here pt has hemmorgic shock.

* Abnormal ↑ Aldosteron due to ↑ Renin (From kidney) → which depend on
RBF → when ↓RBF → ↑ renin → ↑ Na & H_2O → ↑ volume → ↑(HTN) (↓K , ↓H^+)
if happen renal artey stenosis Although (volume NL) * saline unreponsive
NL كان ↑ بس برضو

not saline responsive.

* RAS → ↑ renin

* Conn syndrome → tumer in Adrenal gland secrete Aldosteron

met. Acidosis

NAGMA
- Renal → RTA
- GIT → diarrhea

HAGMA

MUD PILES

met Alkalosis

- saline responsive
  - ↓ volume
    - sweating
    - vomiting
    - Hemmorage
    - ↑ diuresis
- saline unresponsive
  - ↑ Aldo
    - RAS
    - Con syndrome.

اللهم ارحم الدكتور جهاد الجبور واغفر له واعفُ وتجاوز عنه واجعله من أهل الفردوس الأعلى يا رب العالمين