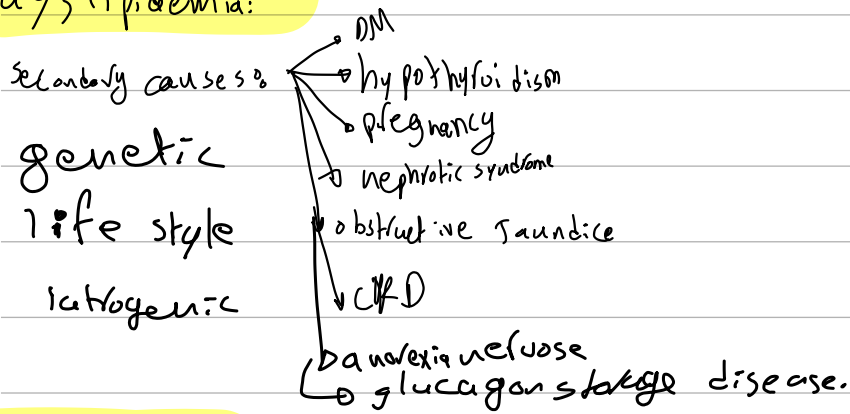


dyslipidemia:



hyperlipidemias:

↑ Total cholesterol + ↑ Triglyceride or ↑ LDL or both

hypercholesterolemia: ↑ total blood cholesterol

hyperbeta lipoproteinemia: ↑ LDL

Normal LDL < 100 Mg/dl

100 - 129 above optimal LDL

130 - 159 borderline high LDL

> 160 high LDL

Hypo alpha lipoproteinemia:

↓ HDL < 40

borderline low (40 - 49)

Normal → Male ≥ 40
Female ≥ 50

Hypertriglyceridemia: ↑ TG

Most common: hypothyroidism

Normal < 150

moderate 150 - 499

Moderate to severe 500 - 999

Severe > 1000 (acute pancreatitis)

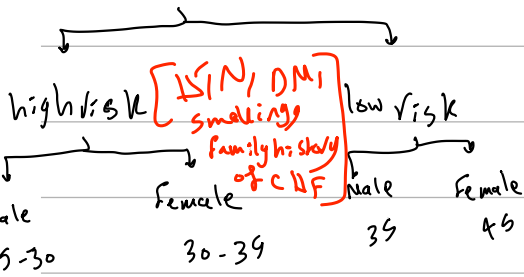
Metabolic Syndrome: 3 or more of the following:

	Male	Female
waist circumference	≥ 102	≥ 88
HDL	< 40	< 50
TAG	≥ 190	≥ 150
Bp	systolic ≥ 130 diastolic ≥ 85	systolic ≥ 130 diastolic ≥ 85
FBG	≥ 100	≥ 100

Screening

normal

elevated LDL, ↓ HDL, ↑ cholesterol



second lipid profile should be obtained (fasting)

exclude secondary causes:

Confirm diagnosis by fasting before starting

Thyroid

exclude secondary causes:

↑ Thyroid, renal, liver function test

statin Side effects

① Myalgia

② Rhabdomyolysis stop when $\geq 10 \text{ u/LN}$

③ ↑ liver enzymes stop when $\geq 30 \text{ u/LN}$

④ DM

⑤ GI symptoms



to increase HDL: stop smoking, exercise

hypothyroidism increase (TG)



high potency: atorvastatin (20-40)
(40-80)

Moderate: pravastatin

Fluvastatin

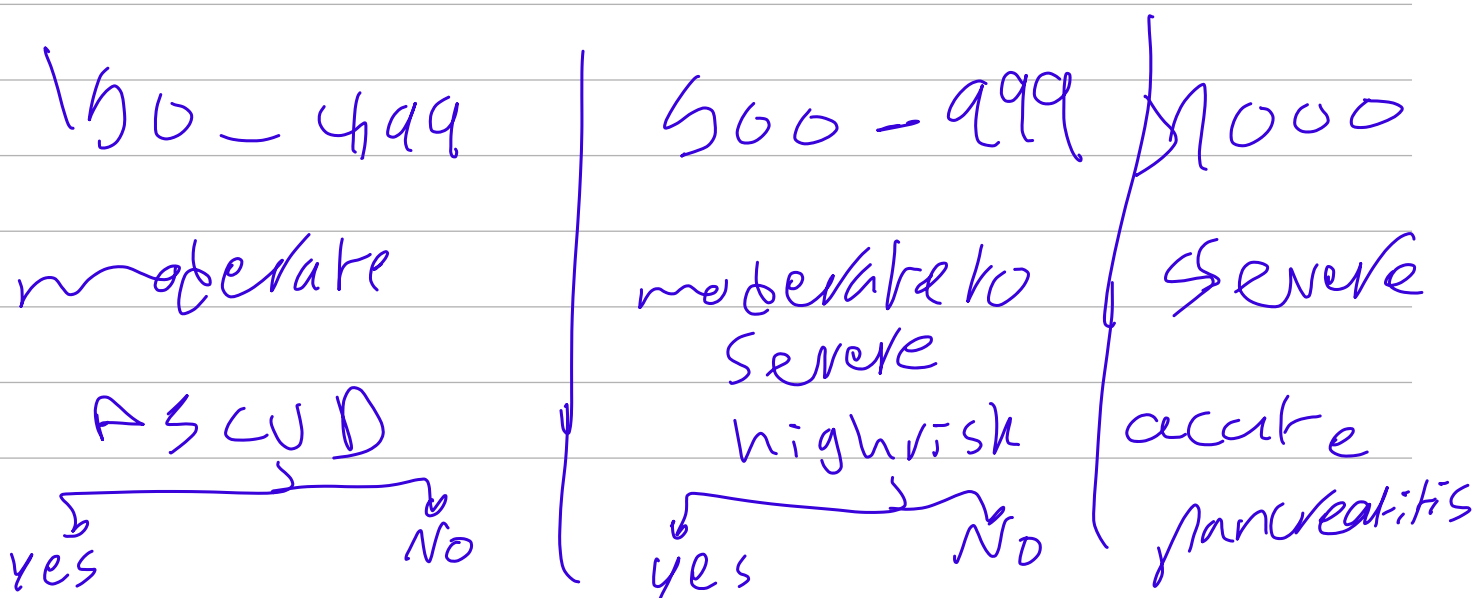
↳ > 20% high intensity statin
 ↳ 7.5 - 20 → moderate
 ↳ 5 - 7.5% moderate

if ASCD / is enhanced

- LDL ≥ 160
- FH of ASCVD
- metabolic syndrome
- inflammatory disease : psoriasis, Rheumatoid arthritis
- ethnicity
- CRP ≥ 2 , lipoprotein ≥ 50 apolipoprotein B ≥ 130 ABI < 0.9

except if moderate intensity statin

↳ < 5% ; life style modification



omega 3 fatty acid (IPE) ^{omega 3} ^{private}

private

لزم الهم

لزم الهم

والصفي القلب

لزم اعطيه

Fluid

TG < 2000

using TG lowering
Therapy

Secondary

As CVD $\Rightarrow$ high intensity statin ≤ 75

pathophysiology of statin

inhibit HMG CoA reductase enzyme

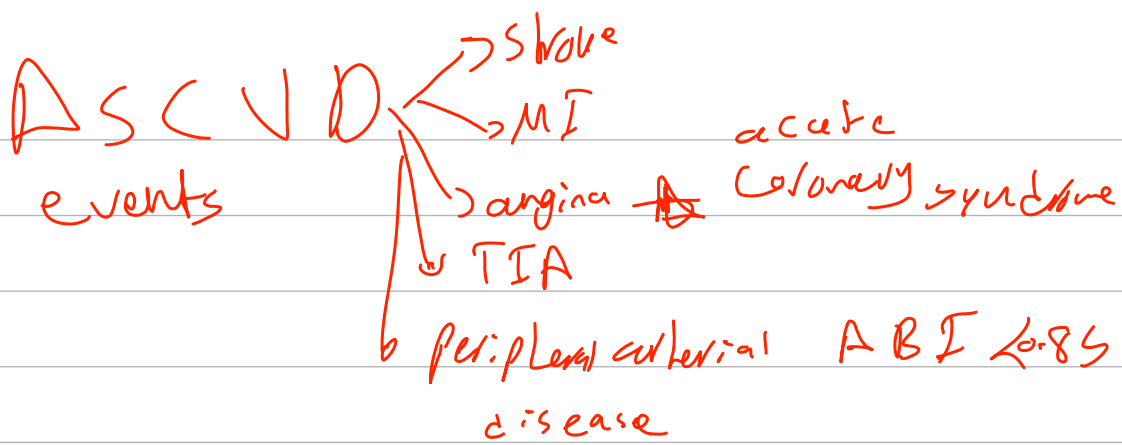
شرحها كخطوات فيزيولوجية:

1. الكبد يصنع الكوليسترول باستخدام HMG-CoA reductase
2. الـ Statins توقف هذا الإنزيم $\rightarrow$ $\downarrow$ كوليسترول
3. الكبد يشعر بنقص الكوليسترول $\rightarrow$ يعمل upregulation لمستقبلات LDL
4. هـاي المستقبلات تسحب LDL من الدم $\rightarrow$ $\downarrow$ LDL-C في البلازما
5. النتيجة: $\downarrow$ خطر ترسب الدهون على الشرايين $\rightarrow$ $\downarrow$ خطر تصلب الشرايين واحتشاء القلب

Competitive inhibition of HMG-CoA reductase renders this enzyme unable to convert HMG-CoA to mevalonate (the rate-limiting step of cholesterol synthesis) $\rightarrow$ reduced intrahepatic cholesterol biosynthesis $\rightarrow$ upregulation of expression of LDL receptor gene via sterol regulatory element-binding protein (SREBP) $\rightarrow$ increased LDL recycling and: [2]

$\downarrow \downarrow$ LDL cholesterol

$\uparrow$ HDL cholesterol



high risk

Age > 65

CABG

HTN

DM

CKD GFR $15-99$

smoking

persistently LDL > 100

history of CHD

heterozygous hypercholesterolemia

follow up

on statin repeat 4-12 weeks then every 3-12 months

high risk $\leftarrow$ desirable lipids every 1-2 years
borderline (6 months)

low risk ← 5 years
1 year

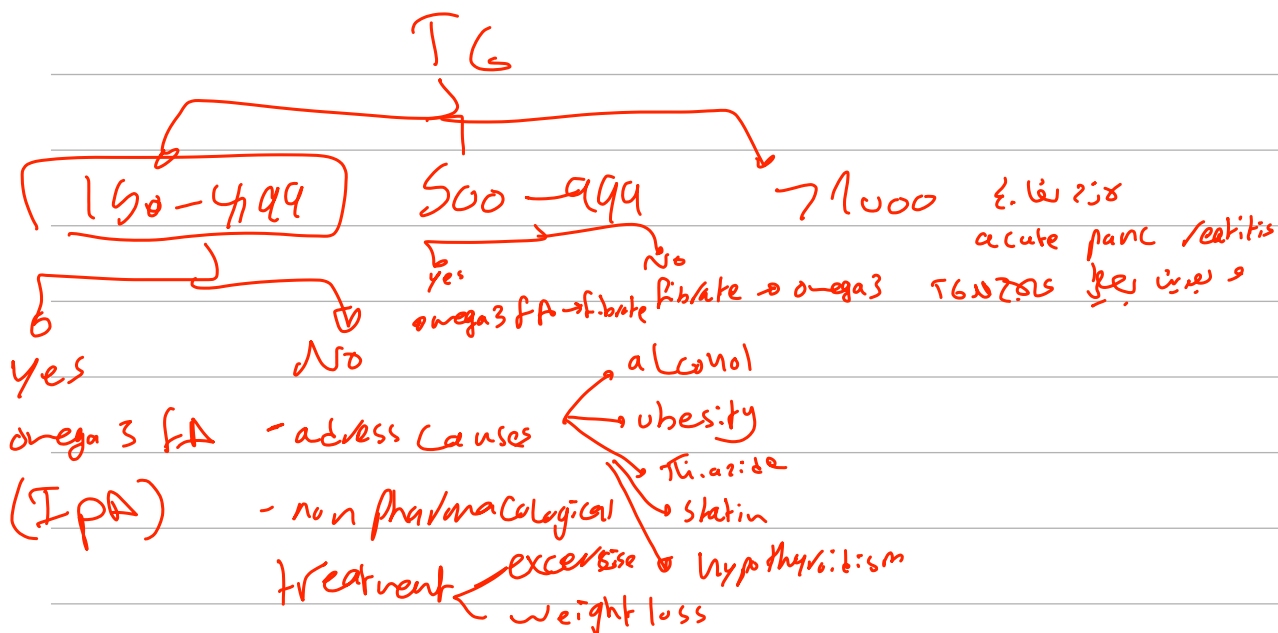
دو ساله

دو ساله

hypertriglyceridemia: A fasting TG

alcohol, obesity, steroid, uncontrolled DM,

hypothyroidism



- treat LDL-C

Vaginal discharge

speculum: device that facilitate inspection of the vaginal wall and ectocervix

- amount: varies by individual and by stage of menstrual cycle

- color: Profuse

malignant (fishy)

bloody, brown, green, yellow, gray

- pruritis & erythema tenderness

DD

MY HIGHLIGHTING

Vulvovaginitis > Infectious vulvovaginitis

Features	Bacterial vaginosis	Trichomoniasis	Vaginal yeast infection	Gonorrhea	Chlamydia infections
Pathogen	<i>Gardnerella vaginalis</i>	<i>Trichomonas vaginalis</i>	<i>Candida albicans</i>	<i>Neisseria gonorrhoeae</i>	<i>Chlamydia trachomatis</i> serotype D-K
Discharge	Gray/milky Fishy odor	Frothy, yellow-green Foul-smelling	White, clumpy, and thick (cottage cheese-like) Odorless	Purulent, creamy May be malodorous	Purulent, bloody
Vaginal inflammation	Typically absent	Present	Present	Absent	
Cervicitis	Typically absent	Often present	Absent	Present	
Vaginal pH	> 4.5		no/mal 4-4.5	Variable	
Microscopy findings	Clue cells epithelial cells surrounded by bacteria	Flagellated protozoa	Pseudohyphae budding yeast	Gram-negative, intracellular diplococci	Intracellular organisms that Gram stain poorly
Treatment	Metronidazole	Topical 0.75% Metronidazole Treat sexual partner(s)	Topical azoles Oral fluconazole in nonpregnant adults	Ceftriaxone IV/IM Treat sexual partner(s)	Azithromycin Doxycycline in nonpregnant patients Treat sexual partner(s)

500mg b/w 7 days
safe for pregnancy

risk factors smoking
multiple sexual partners/obesity/diet

strawberry cervix
red dots

DM / pregnancy
immunosuppressed, antibiotics, SGLT2 inhibitors

noninfectious

* atrophic vaginitis: in menopausal women
estrogen: 200g

* chemical irritant avoid douching

Contraception: prevention of pregnancy

hormonal Contraceptive

estrogen and/or progestin to prevent ovulation

non hormonal: Sterilization Copper IUD / barrier methods, behaviour methods

short duration ex: condom

long duration: IUD, progestin implant

Behavioral methods

① Fertility awareness - based methods: intercourse can be avoided in fertile window (5 days before ovulation and the day of ovulation)

② Lactational amenorrhea: breastfeeding induce amenorrhea and suppress ovulation

③ Coitus interruptus: penis is withdrawn from the vagina before ejaculation

Barrier methods: prevent fertilization by blocking the sperm from reaching an oocyte (Condom)

Hormonal Contraception

Combined

estrogen + progestin

highly effective, shortly acting
reversible

non contraceptive benefits:

- polycystic ovation syndrome
- endometriosis
- abnormal uterine bleeding, heavy menstrual bleeding
- primary dysmenorrhea

progestin only

benefits

dysmenorrhea
endometriosis

abnormal uterine bleeding

Types:

- oral POP
- injection DMPA: depot ^{IM, subcutaneous} medroxyprogesterone acetate
- IUD
- implant: upper arm ^{POP 3-5 years}

Types

① oral pills daily

② contraceptive patch: transdermal once a week for 3 weeks

③ vaginal ring for 3 weeks: flexible ring containing ethinyl estradiol and etonogestrel

(HC can be used for 3 weeks, then 7 days for placebo free week during this week, there may have withdrawal bleeding, but extended use increase incidence of breakthrough bleeding)

adverse effects of CHCs:

① irregular bleeding

② GI RD, nausea

③ mood swings

④ headache

⑤ breast tenderness

(regular/ emergent method for (3-12 months))

IUD: T shaped device inserted in uterus

non hormonal

hormonal (progestin)

Copper

benefits:-

↓ risk of endometrial & cervical cancer

↓ menstrual pain and bleeding

side effects:

prolonged and heavy menstrual bleeding especially the first 3-6 months of placement



MoA: create inflammation decrease sperm viability and function

suppress fertilization implantation

MoA:

decrease sperm viability
prevent sperm to reach oocyte

Sterilization: permanent infertility

① bilateral tubal ligation

② vasectomy

Complication

— hematoma

— surgical site infection

DM

hyperglycemia symptoms: polyuria, polydipsia, polyphagia, blurred vision, weight loss

hyperosmolar hyperglycemic state: type 2 DM, hyperglycemia, dehydration without ketoacidosis

25% of type 1 DM $\Rightarrow$ DNA

	Type 1	Type 2	Monogenic DM
age	< 25, may occur at any age	> 25	< 25
		overweight	general BP

Weight	Thin / obesity at diagnosis		
autoantibodies	✓	X	X
insulin dependent	✓	X	X
Insulin sensitivity	normal	↓	normal
Family history	5-10%	75-90%	≥ 3 generations
DNA	high	low	low

Diagnosis		
	asymptomatic non pregnant (Type 2 DM)	p/e Diabetes
Symptomatic	FBG ≥ 126 mg/dL	HbA1C (5.7-6.4)
Random ≥ 200	Two hour OGTT ≥ 200	FPG (100-125)
	HbA1C ≥ 6.5	OGTT (140-199)
	(at least 2 abnormal tests)	Repeat annually

Screening

Diabetes Mellitus type 1

urine microalbumin at 12 then
every 6-12 months

Eye examination within 5th year then
annually

Bp

lipid panel, HbA1C

Type 2

Same except
eye examination
urine microalbumin
at time of diagnosis
then annually

Management:

Goal $HbA_{1c} < 7$

comorbidities or a limited life expectancy.

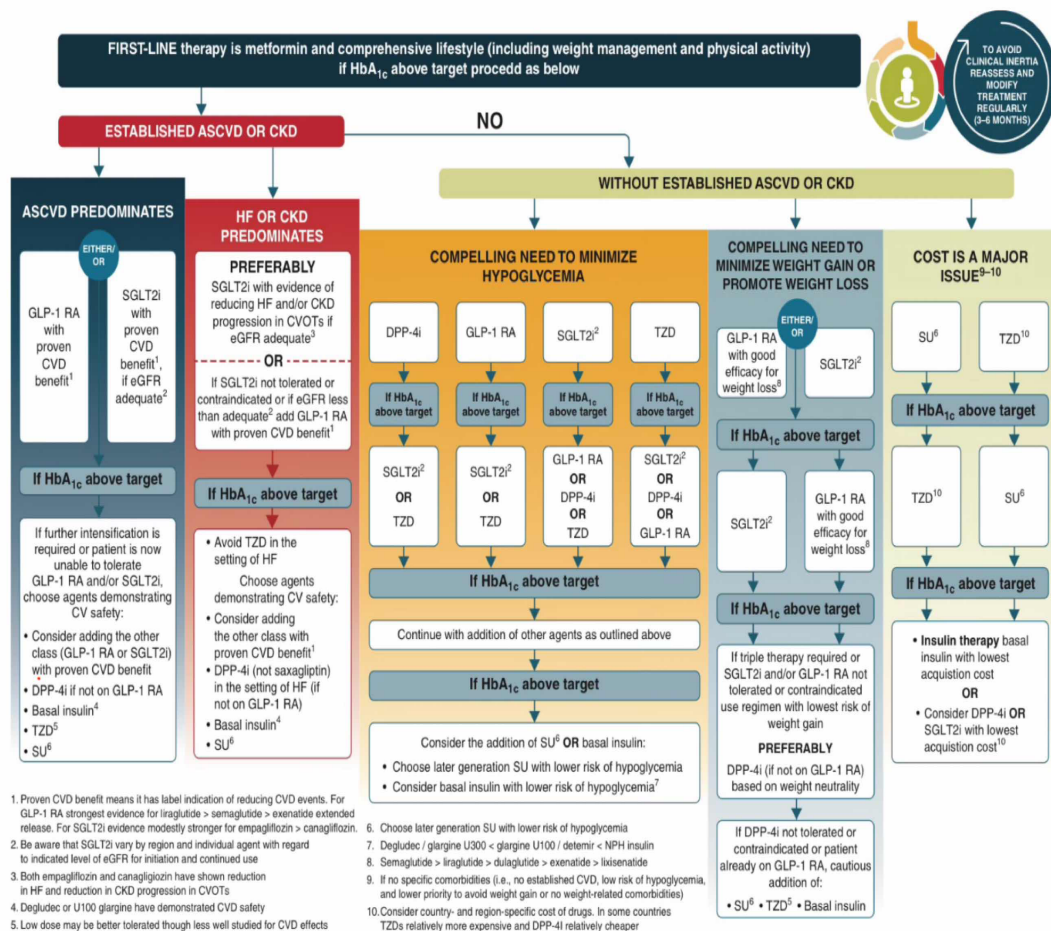


FIGURE 10-1. Recommendations for medical treatment of diabetes mellitus type 2. From American Diabetes Association. Standards of Medical Care in Diabetes 2019. *Diabetes Care*. 2019;42:S90-S102.

5. Monitoring

يعني:

- العلاج الأول دائماً هو الميتفورمين + تغيير نمط الحياة (نظام غذائي، رياضة).
- إذا HbA1c ظل فوق الهدف → تكمل حسب الخريطة التالية بناءً على حالة المريض.

الجهة اليسرى: 

● "Established ASCVD or CKD"

إذا المريض عنده تصلب شرايين قلبي وعائي (ASCVD) أو مرض كلوي مزمن (CKD) مثبت:

◆ 1. ASCVD Predominates (لو القلب هو المسيطر):

اختار: 

- GLP-1 RA: liraglutide, semaglutide
عندها إثبات أنها تقلل خطر الـ CVD

◆ 2. HF or CKD Predominate (إذا القلب الفشل أو الكلى هم الأهم):

الأفضل: 

- SGLT2i (مثل empagliflozin) إذا كان eGFR مناسب
لأنها تقلل progression CKD & HF hospitalizations

⚠ إذا SGLT2i ممنوعة أو eGFR نازل كثير:
● نستخدم GLP-1 RA المفيدة للقلب.

إذا HbA1c بعد هذا كله ما زال فوق الهدف:
● نضيف أدوية إضافية حسب الحالة (insulin, DPP4i, TZD)...

الجهة الوسطى: 

بدون ASCVD أو CKD، لكن مع أولوية علاجية خاصة

◆ 1. Need to minimize hypoglycemia (الهدف تجنب هبوط السكر):

استخدم أدوية لا تسبب hypoglycemia: 

- DPP4i, GLP-1 RA, SGLT2i, أو TZD

◆ 2. Need to minimize weight gain or promote weight loss (هدفنا نحافظ عالوزن أو نقلله):

الأفضل: 

- SGLT2i أو GLP-1 RA

SGLT2i = empagliflozin, dapagliflozin

GLP-1 RA = semaglutide, liraglutide

DPP4i = sitagliptin الجهة اليمنى: 

SU = sulfonylurea (مثل glimepiride)

Cost is a major issue (لو الكلفة عائق) TZD = pioglitazone

استخدم الأدوية الأرخص: 

- SU (Sulfonylureas) أو TZD

⚠ بس هدول ممكن يعملوا زيادة وزن أو هبوط سكر.

Gout: The disease of kings

hyperuricemia: Elevated serum uric acid > 6 women > 7 men

Causes

primary: diet (idiopathic)

Secondary:

decrease uric excretion

menopause, ketoacidosis, Chronic renal insufficiency

Increased uric acid production: obesity

OSA, respiratory acidosis

Nehan Syndrome

Combined: alcohol consumption

low temperature (1st metatarsophalangeal Joint) podagra

pathophysiology: Triggers urate crystal deposition

how to diagnose?? arthrocentesis and synovial fluid analysis is the gold standard

WBC = $7200 < 100,000$

① medium to large Toints

② specialist doctor

Clinical diagnosis:

Male (2)

HTN, angina, stroke, DVT (1.5)

history of

> 8 relatively high

4-8 moderate

≤ 4 low likely agent

Labs

* serum uric acid level (high/normal, low) during acute attack - **لا يزداد بعد ١٠-١٤ يومين**

* CBC, inflammatory markers $\uparrow$

Imaging:

US: signs of inflammation, erosion, uric acid crystals

X-ray: normal in acute attack

Chronic: punched out lytic bone lesion double contour sign: hyperechoic crystals above

Treatment

hypoechoic cartilage above hyperechoic bone

① life style modifications: organ meat, red meat, fish, weight loss, stop alcohol, ice on affected joint

1st line: NSAIDs, ^{systemic} glucocorticoids / ^{intra-articular} colchicine

2nd line: give LL inhibitor
max. 1000mg dose for 3-5 days in the 1st day

urate lowering therapies for chronic gout

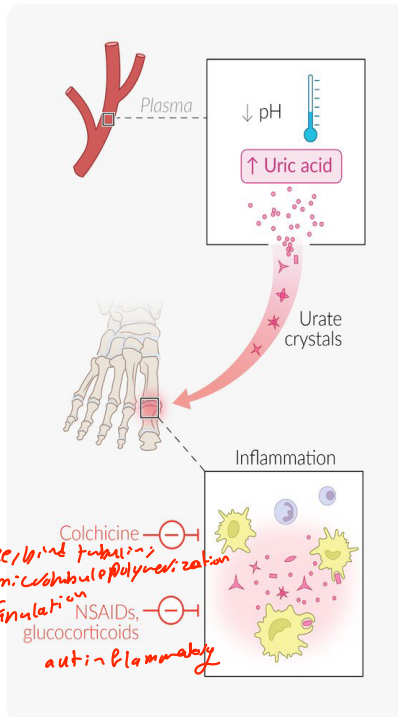
absolute indications:

① gout 2 or more times/year

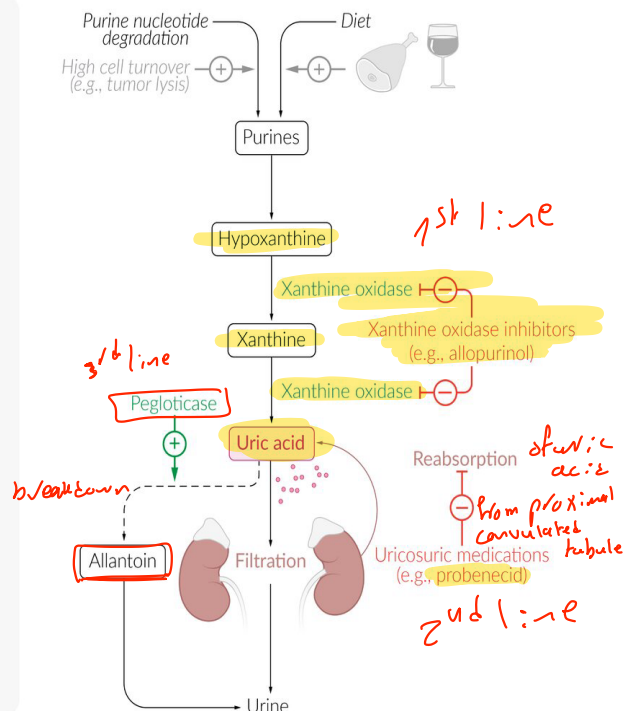
② damage on images

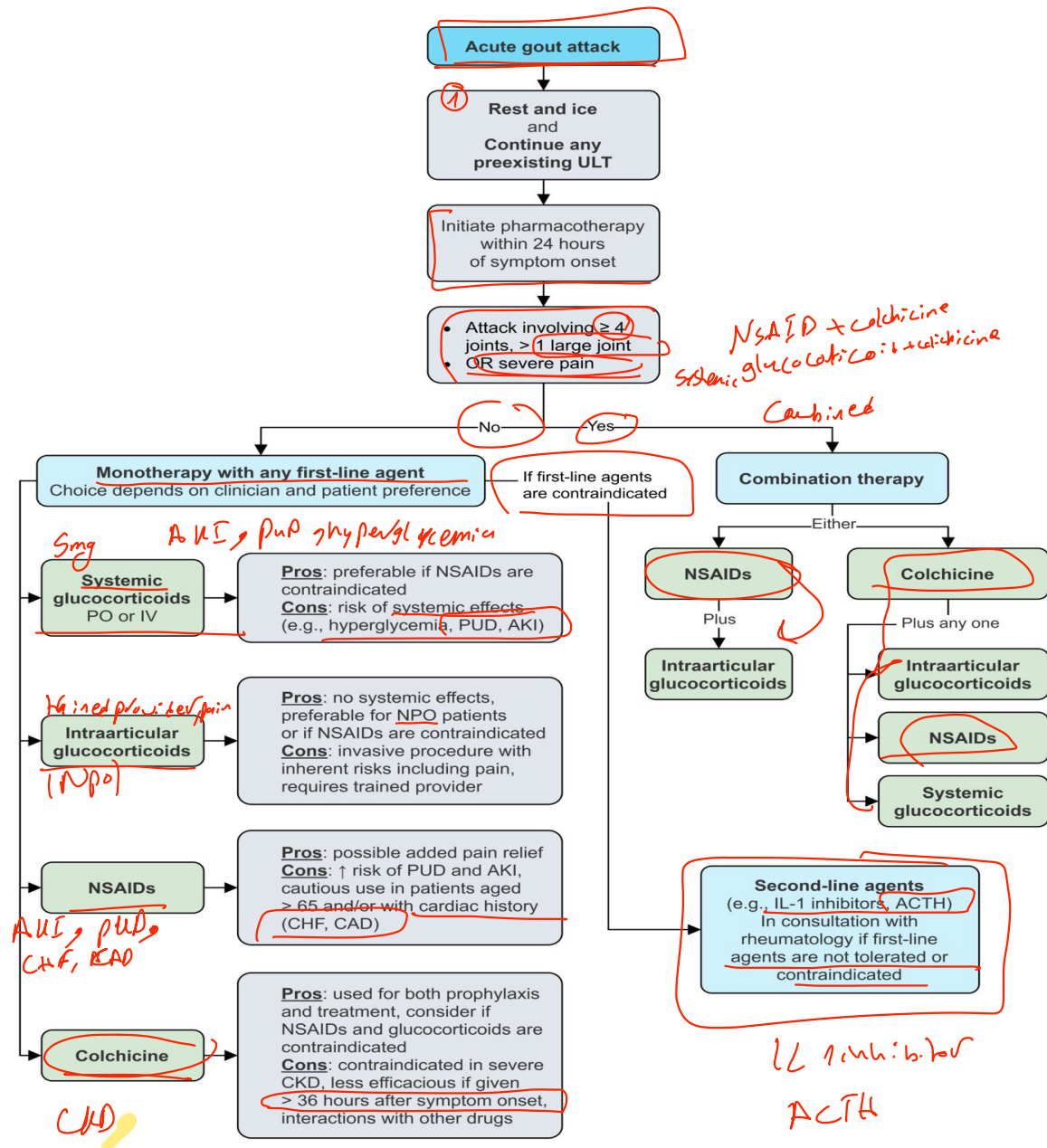
③ 1 attack $\rightarrow$ CKD 3 stages and male
 $\rightarrow$ serum uric acid ≥ 7
 $\rightarrow$ stones in UT

Acute gout attack



Purine metabolism





Antenatal care: Folate 5 mg

1st trimester

history, physical examination $\rightarrow$ obstetric history: miscarriages, preterm delivery, pre-eclampsia
 $\rightarrow$ BMI, Bp, auscultation of heart of fetus

Stop or change medications contraindicated in pregnancy ex: pregnant women with hypothyroidism should increase levothyroxine by 2 extra doses per week

determine estimated date of delivery at 1st trimester by US

assess vaccination status and administer $\rightarrow$ inactivated influenza vaccine

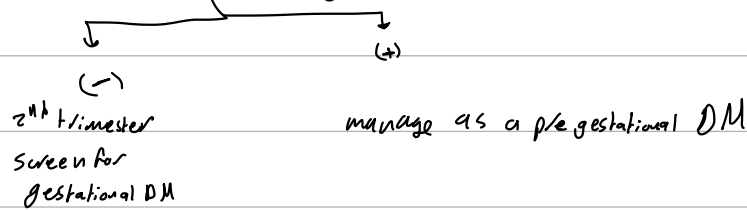
live attenuated influenza, varicella, MMR vaccines are

Covid19

Contraindicated in pregnancy delay until after delivery

- Screening

Subella, Urucella, H₂O, hyperglycemia testing



2nd trimester

Recommended laboratory screening studies at 24-28 week's gestation

CBC, OGTT

2nd trimester US:

- ① fetal anomalies
- ② estimation of gestational age

3rd trimester: preparing for delivery

- ① monitor fetal growth with symphysis-fundal height
- ② influenza vaccine and/or COVID-19 booster if due
- ③ 27-36 weeks gestation: Tetanus diphtheria Vaccine
- ④ 32-36 week: give respiratory syncytial virus vaccine
- ⑤ 36-37 + 6 weeks gestation: perform group B streptococcus screening

In 3rd trimester, prenatal visits increase to every 2 weeks between 28-36 weeks then weekly

- ⑥ screening for sexually transmitted infection

HTN:-

Normal	$< \frac{120}{80}$
elevated	$\frac{120-129}{< 80}$
Stage 1	$\frac{130-139}{80-89}$
Stage 2	$\geq \frac{140}{90}$

Epidemiology:-

♂ > ♀

primary $\rightarrow 90\%$

secondary $\rightarrow 10\%$

increase with age

Cause s

Arterial hypertension

Epidemiology

- Most common cardiovascular risk factor
- Prevalence (US): 1/3 – 1/2 of adults

Etiology

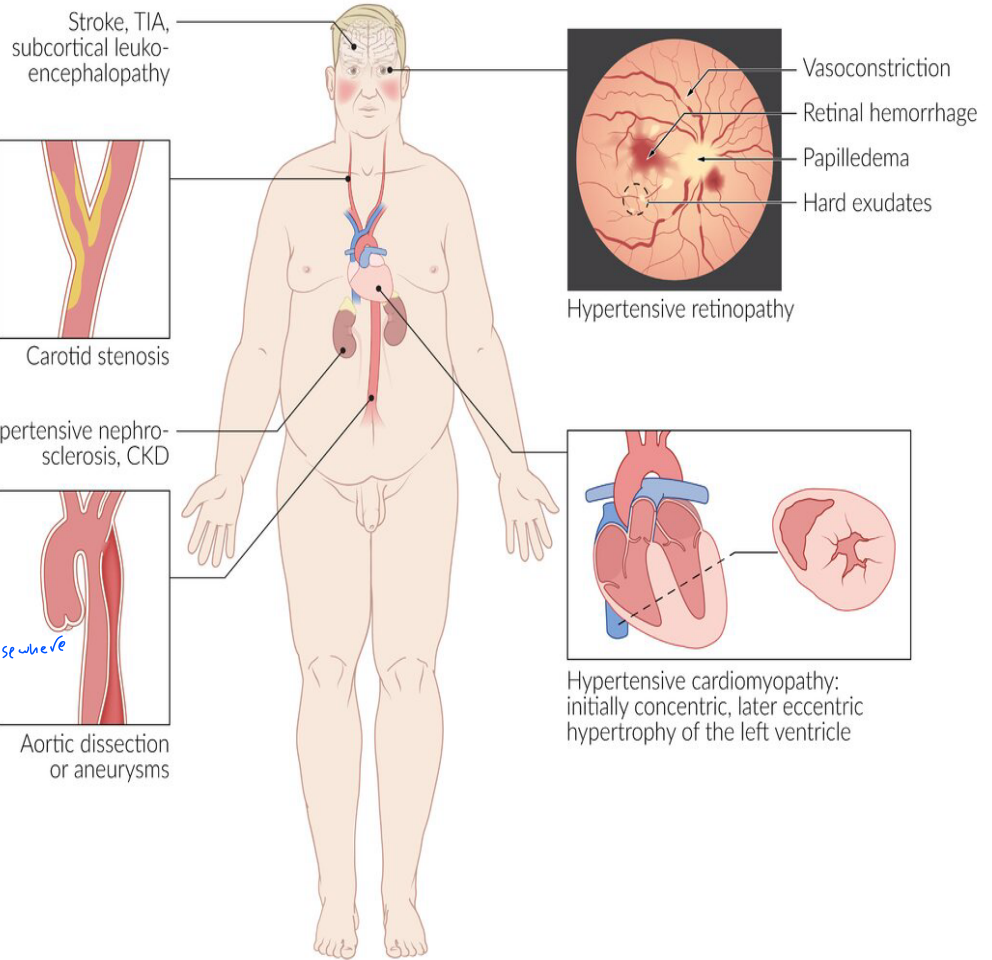
1. Primary hypertension (~ 90%)
2. Secondary hypertension (~ 10%):
 - Endocrine causes (e.g., hyperaldosteronism, hyperthyroidism, pheochromocytoma)
 - Renal causes (e.g., renal artery stenosis, renal parenchymal disease)
 - Coarctation of the aorta
 - Obstructive sleep apnea
 - Medications (e.g., corticosteroids, sympathomimetic drugs)

Diagnostic criteria

BP $\geq$ 130/80 mm Hg

Subtypes

- White coat hypertension
- Masked hypertension *normal in clinic but elevated elsewhere*
- Isolated systolic hypertension (i.e., systolic BP $\geq$ 140 mm Hg and diastolic BP $\leq$ 90 mm Hg)



History:

① duration

② previous therapies

③ Fx of HTN and CVD

④ diet, physical activity

⑤ DM, dyslipidemia, smoking

⑥ sweating, palpitations, hemoptysis, snoring for OSA

phx:

– weight + height

– BP on both arms

– Thyroid examination + carotid

– examination of blood vessels

– CHF and neurological examination

- Fundoscopy

Indication for secondary HTN

- ① < 30 / 70 العمر
- ② Malignant HTN (uncontrolled even with 3 drugs or need 4 to be controlled)
- ③ severe SBP > 180 / DBP > 120

labs

- electrolyte (ca, creatinine)

- Fasting glucose

- lipid profile

- CBC

- Echo

- TSH

- urinalysis

Treatments-

1. life style modification :

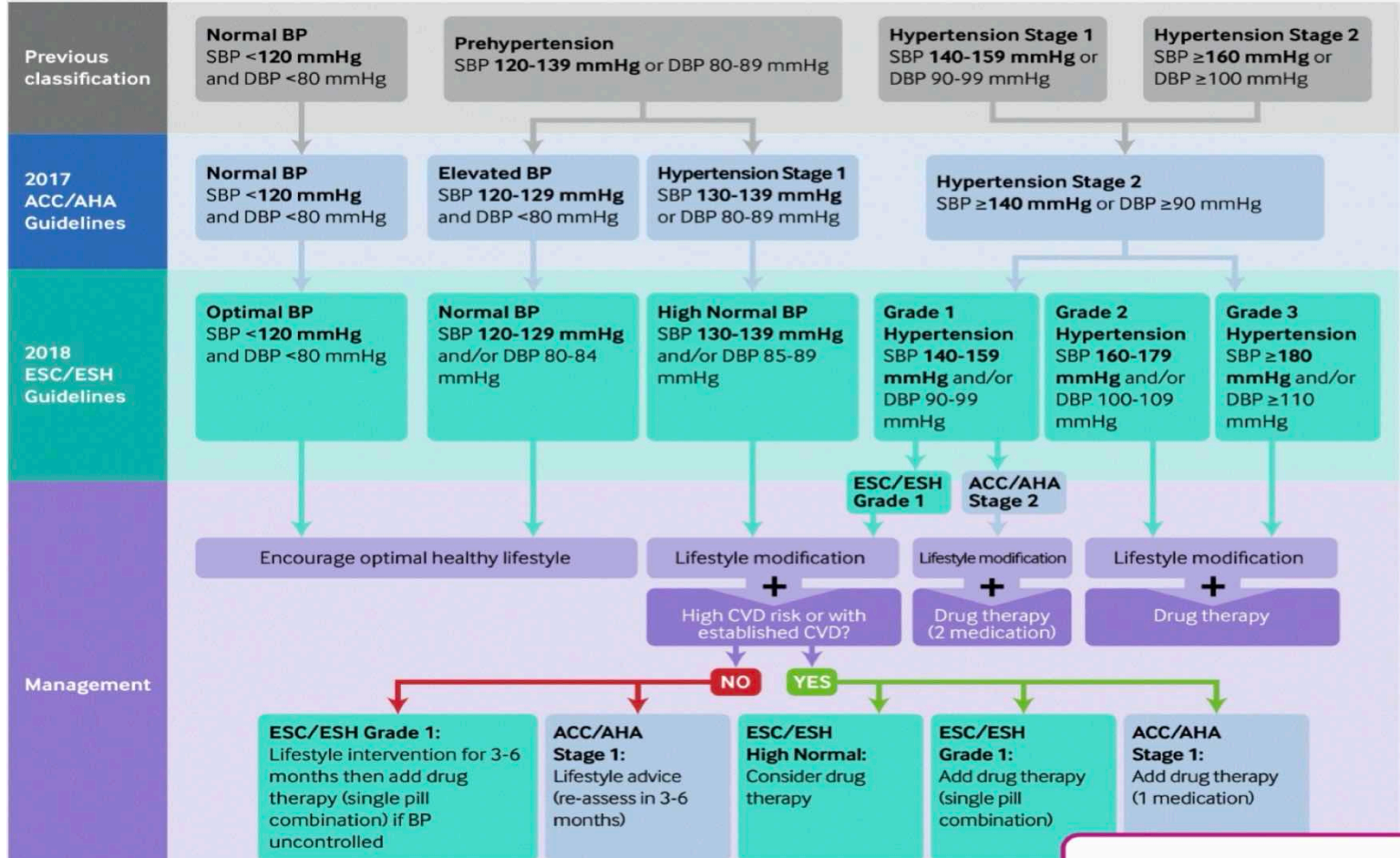
- ① weight loss 1mmHg for 1kg reduction in body weight
- ② Dash diet (rich in fruit, vegetables and grains & low fat)
- ③ Reduce salt intake (< 1500 mg/day), K^+ intake (3600-5000)
- ④ Physical activity 150 minutes/week
- ⑤ reduce alcohol + smoking

Goals

General : $< \frac{140}{90}$

DM, CVD, renal insufficiency $< \frac{130}{80}$

age > 60 , $< \frac{150}{90}$



Case 1:
A 28 year old male patient, previously healthy, came to our clinic for follow up, his BP at the clinic 135/85.
There is a family history of Hypertension and cardiovascular diseases
What initial steps should be taken to confirm the diagnosis and what lifestyle modifications can be recommended?

Case 2:
A 62 year old patient, known with controlled HTN, newly diagnosed with hyperthyroidism, today come to the clinic complaining of sweating and palpitation. BP 150/95.
What additional evaluations should be conducted for this patient?

Case 3:
A 75 year old patient, known HTN, heart failure, he is on enalapril, amlocard, and hydrochlorothiazide. His BP at the clinic 180/110. He reports no symptoms.
What are the potential causes of hypertension in this patient, and what management strategies should be considered?

Case 4:
A 55 year old patient, known DM type 2 on metformin. His BP is 145/85. There is no other significant medical history.
What are the target blood pressure goals for this patient, and which classes of medications are appropriate for initial therapy?

(ACEI) Ca channel blocker (diuretic)

Case 1: Confirm by home or ambulatory BP monitoring

Lifestyle modification: salt intake, stop smoking, stressy dash diet / weight loss

Case 2: TSH / T4, ECG, Echocardiogram
24h BP monitoring, renal function, glucose, lipid profile

Case 3:

causes: non compliance, drug interactions, resistant HTN, progression of heart failure
secondary causes: renal artery stenosis, primary aldosteronism, pheochromocytoma

Management: Renal Function (Cr, BUN), add BB or spironolactone (K⁺ sparing diuretic) / TSH, urinalysis

Case 4: target < 130/80 / ACEI, ARBs

TOPICS	POPULATION	TOOLS	FREQUENCY	GRADE
Pre-DM & T2DM	35-75 Years (overweight & obese)	HBA1C	ANNUALLY	B
DYSLIPIDEMIA	Male: above 35 years Female : above 45 years (younger if high risk for coronary heart disease)	Lipid profile	Every 4-6 years if low risk , more frequent if at high risk or abnormal results	B
HYPERTENSION	Above 18 years	Office blood pressure measurements	Every 2 years	B
OSTEOPOROSIS	*Women 65 years & older. *postmenopausal women younger than 65 years at increased risk of osteoporosis	DEXA SCAN		B
ABDOMINAL AORTIC ANEURYSM	For males only (65-75) who ever smoked	Abdominal US	once	B
BREAST CANCER	50-74 years female	mammogram	Every 2 years	B
COLON CANCER	45-75 Years	*FIT,FOB annually *DNA FIT every 3-5years *Colon CT or flexible sigmoidoscopy every 5 years * colonoscopy every 10 years * flexible sigmoidoscopy every 10 years with FIT annually		A
CERVICAL CANCER	* 21-29 years in sexually active female	*Pap smear Q 3 years		

	*30-65 years female	*PAP smear Q 3 years , HPV Q 5 years , Contesting Q 5 years		B
LUNG CANCER	50-80 years if : -Has 20 pack year smoking history -Current smoke -Quit smoking within 15years	Low density CT	annually	B

Anemia: a decrease in the number of circulating RBC

Men Hb < 13

Women Hb < 12

Pregnant Hb < 11

Classification of anemia

Microcytic: MCV < 80

Iron deficiency anemia, lead poisoning, thalassemia (globin chain defect), anemia of chronic diseases, sideroblastic anemia

Normocytic: MCV $80 - 100$

hemolytic anemia

intrinsic defects: hemoglobinopathies: sickle cell disease, hemoglobin disease, G6PD, pyruvate kinase deficiency

extrinsic: autoimmune hemolytic anemia (EBV infection, SLE, non Hodgkin lymphoma, chronic lymphocytic leukemia)

- Microangiopathic hemolytic anemia (TTP, hemolytic uremic syndrome, HELLP syndrome)
↳ schistocyte on blood smear

- macroangiopathic hemolytic anemia

non hemolytic

blood loss, aplastic anemia, anemia of chronic kidney disease

Macrocytic > 100 : defective DNA synthesis or repair

Megaloblastic anemia: folate, B12 deficiency (hypersegmented neutrophils)

Non megaloblastic: liver disease, alcohol hypothyroidism, Multiple myeloma,

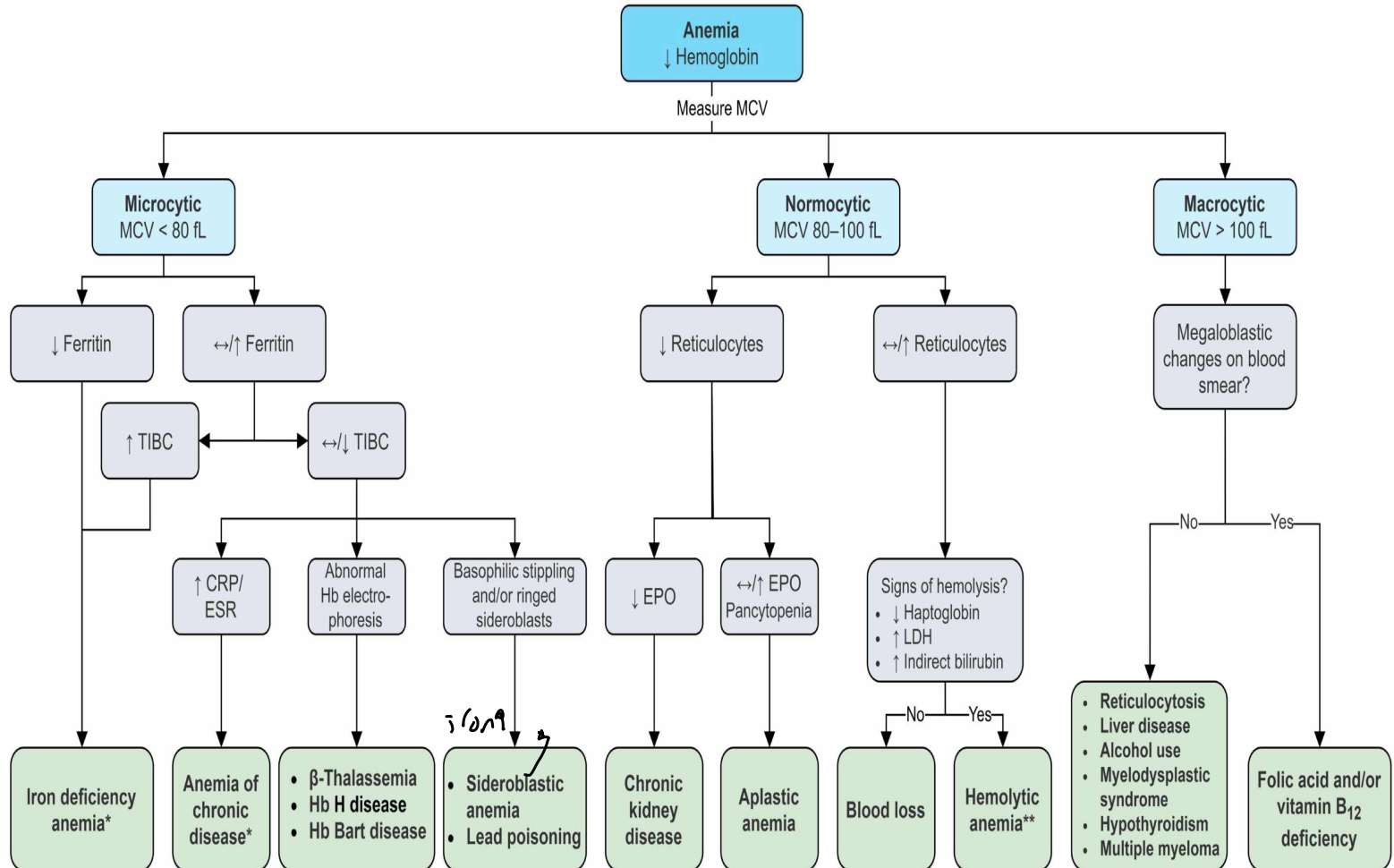
Clinical Features

Features of IDAs: pICA, brittle nails, angular cheilitis, atrophic glossitis

Features of megaloblastic anemias: oral sores, mucosal bleeding, paresthesia, ataxia

Features of thalassemias (features of extramedullary hematopoiesis)

- hepatosplenomegaly
- para vertebral mass
- widening of diaphic spaces of the skull



*Anemia of chronic disease and iron deficiency anemia may also manifest as normocytic anemia.

**Hemolytic anemias may also manifest as microcytic anemia with normal iron studies.

Management

- Iron $\rightarrow$ IDA

- Stop bleeding $\rightarrow$ acute hemolytic anemia

- Epo $\rightarrow$ Chronic kidney disease

- $\downarrow$ B₁₂, folate $\rightarrow$ megaloblastic anemia

- $\downarrow$ B₆ (pyridoxine) $\rightarrow$ sideroblastic anemia

- Blood transfusion in $Hb < 7$ or 8 , moderate anemia in high risk or symptomatic

- BM transplantation $\rightarrow$ aplastic anemia