

April 1943

## الغدة الدرقية Thyroid gland 8-

major function

metabolic rate & growth  
النمو + الأيض

\* active Thyroid gland تعبير

↑ activity increase ← hyperactive  
↓ activity decrease ← hypoactive

لو ممكننا عنا قورينم يجاتي هـ hyperthyroidism

إنتاج عالي من thyroid hormone

thyroid hormone  $T_3$ ,  $T_4$

لهم علاقة بـ الأيض

زيادة في الهرمونات ← hyperthyroidism ← يمين  
عسبة الأيض العالية

loss weight ↑ GI contraction ↑  
cardiac output ↑ breathing ↑

bone → هـ

bone resorption

تسريع

عنا واهم بيان من هذونات الغدة الدرقية  
قليلة عنده hypothyroidism

كيف يتم تكيف thyroid gland على استجابة  
more and more

في الهذونات ← في حلال hypothalamus

يتم ارسال/تعزيز استجابة في sensory neurons ← تنوع

في inter neuron الموجود في hypothalamus

يتم على افراز هرمون اسمه thyrotropin-releasing hormone

TRH & thyrotropin-releasing hormone.

TRH له يروع يحفز anterior pituitary gland يحفز على افراز TSH

TSH & Thyroid stimulating hormone.

TSH - يحفز الغدة الدرقية على إنتاج T3, T4

ت الغدة بينه TSH و TRH

و هو سكر thyroid gland

\* butterfly-shaped

↳ follicles structure هي عبارة

↳ تتكون من خلايا  
اسمها Follicular cells

داخ هذه الخلايا ← thyrotropin

لماذا يتم في البداية تعفيز thyrotropin قبل تعفيز الغدة الدرقية؟

لأنه thyrotropin انا سيقول ان  $T_3, T_4$

① اورد اسمي سيم تعفيز hypothalamus هي

تقدر على إفراز TRH هي على إنتاج ليفيد  
↳ ~~thyroid~~ thyroid hormone → thyrotropin

② بعد ذلك تعيد pituitary تقدر على إفراز TSH <sup>تعفيز</sup>

الذي يعمل على تحويل thyrotropin الى  $T_3, T_4$

$T_4$  :- Thyroxine → responsible for your metabolism, mood and body temperature.

$T_3$  :- Tri-iodo thyronine,

T<sub>4</sub> - have four iodine group

T<sub>3</sub> - have 3 three iodine group

iodine containing hormone 3, thyrotropin \* سيم تكوين

↳ thyroid hormone يعني

لو واحد عنده hyperthyroidism يعني T<sub>3</sub>, T<sub>4</sub> عالي.

Cause :- increase iodine.

← ~~disease~~ disease primary cause  
thyroid gland نفسها مشكلة الأساسية في  
disease secondary cause.

← منطقة أخرى لكنها ترتب عن thyroid gland

hyperthyroidism & (primary) → tumor → thyroid gland

واحد يأخذ أدوية / أدوية زادت عن نسبة  
iodine secondary cause.

hypothalamus, pituitary gland 3 tumor واحد عنده  
↑ TSH  
secondary cause.

T<sub>3</sub>, T<sub>4</sub> ← thyroid تنتقل  
blood بواسطة proteins داخل

- thyroxine-binding globuline ←
- pre albumin ←
- albumin ←

T<sub>3</sub>, T<sub>4</sub> strong bind with TBG

T<sub>3</sub>, T<sub>4</sub> weak bind with albumin and pre albumin  
قليلة جداً

\* لا بدنا نفحصه إذا شئنا وفه أمراضه في الفزة الدرقية  
blood test  
فنحلال وفرفة  
نسبة protein في الدم  
[ Diagnosis of Hypothyroidism  
or Diagnosis of Hyperthyroidism

- TBG more sensitive to bind with T<sub>4</sub> and T<sub>3</sub> than albumin and pre albumin

فقرته T3 and T4 → as total

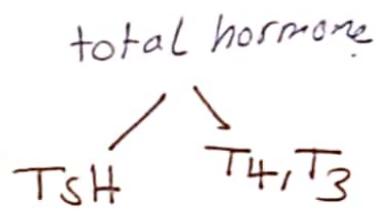
لأنه T4 لا يدخل على الخلية why ??

لا يدخل في شكل T4 يتحول إلى [T3] وينفسد

T4 ← مجموعة iodine وسيتول T3 group

↑ increase T3 ↑ increase metabolic rate ↑

عسانه صيرت فقرته thyroid hormone سته فقرته

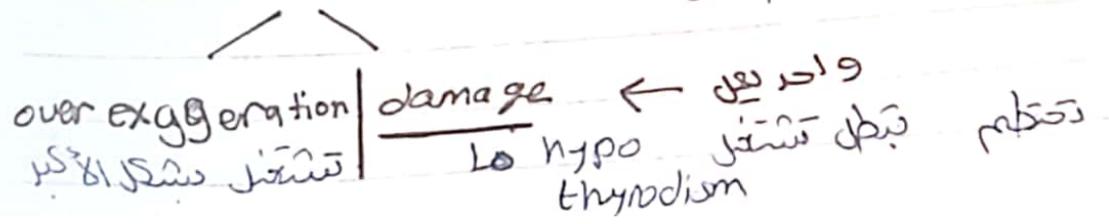


فقرته الأخرى

→ ~~Auto~~ thyroid auto antibody

auto immune disease يعني عنده  
التهاب المناعي يحتاج thyroid gland

\* Most common primary cause of hyper and hypothyroidism  
auto immune disease - thyroid gland التهاب المناعي من يحتاج



استهلاك

radiation :- في مجرىة ذرة الناس تأخذ كعلاج ←



increase T3 and T4 ← iodine يعمل على زيادة. تقول iodine

تأثير الادوية ← في عنزي ادوية تقوي iodine

amiodarone

وقاان :-  
عليها

ليست في  
مقال عليه

treat ventricular arrhythmia and atrial fibrillation

iodide chemical composition تقوي على iodide

Iodine او

لكن ليس عنا

هذا الدواء

hypothyroidism ←

hyperthyroidism ←

hypo : قصوى بعد  
hyper : وصوى

Cause : حسب ال Cause

tumor واحد مريض عنده مشددة في pituitary gland كنه في لها tumor

انتاج TSH عالي تمام ↑ في الكلة iodine

زيادة الونج سوي ←

most common

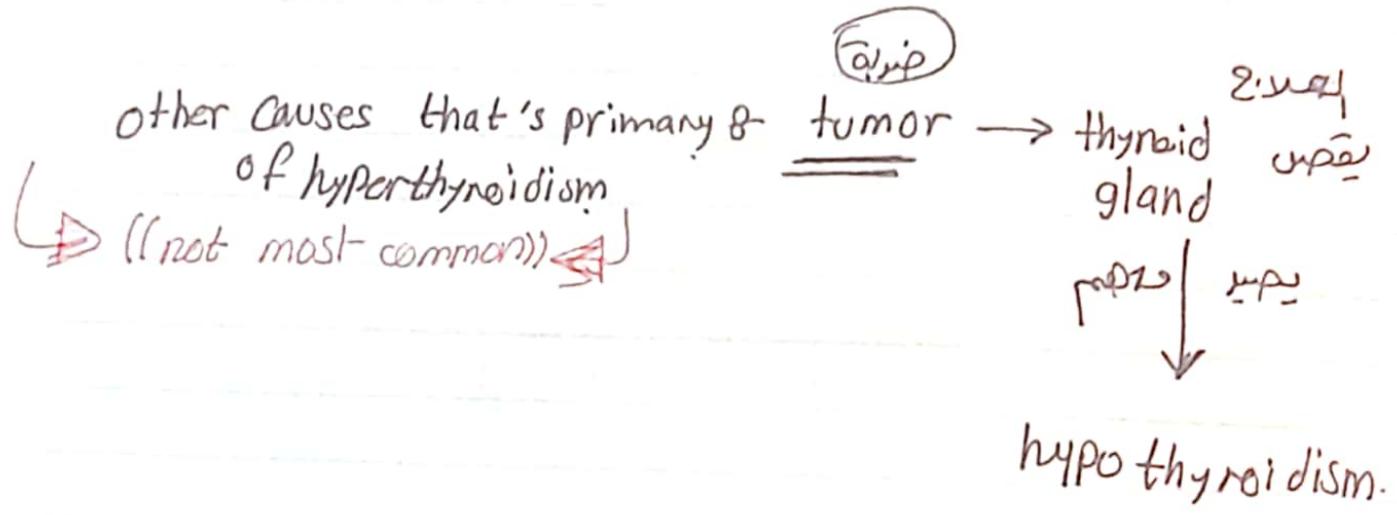
hyper thyroidism :- the name of primary cause :- autoimmune جاء بي

→ Grave's disease

most common

hypo thyroidism :- the name of primary cause autoimmune?

Hashimoto's disease



one of primary cause of hypothyroidism

↳ decrease of thyroid hormone **why!!!**

umon' عقده ~~عقده~~ عقده ~~عقده~~ عقده

size decrease ↓ thyroid hormone ↓ decrease

atrophy [Pituitary gland] في نقص الغدة النخامية

meaning of atrophy - decrease size of the body part, cell, tissue, etc

TSH ↓ decrease iodine not effect

new born baby's في نقص هرمون الغدة النخامية

related Growth, brain development

hypertthyroidism & ارتفاع في نسبة هرمون thyroid

increase of sympathetic nervous system.

as medication, beta blocker

side effect - hypothyroidism.

- ① dry skin
- ② fatigue
- ③ intolerance
- ④ slow speech

~~...~~  
↓  
~~...~~

hyperthyroidism :-

الغدة الكلى  
والغدة الكلى

- ↳ always be nervous.
- muscle weakness
- weight gain .
- tremor

الرجوع إلى السلايدان

contraction of skeletal muscle. ↑

tremor ← يصير حركات خفة .

thyroid gland الغدة الكلى

↑ TRH of hypothalamus → ↑ TSH of pituitary gland ↑

→ ↑ T<sub>3</sub>, T<sub>4</sub> of thyroid gland

hyperthyroidism :- ↑ T<sub>3</sub>, T<sub>4</sub> sympathetic ↑

علاج → beta blockers.

hypothyroidism :- T<sub>3</sub>, T<sub>4</sub> ↓ metabolic rate ↓

علاج → beta adrenergic agonist

Adrenal gland, Adrenal medulla

secreted epinephrine  
norepinephrine.

②

Adrenal cortex

structure of Adrenal cortex has 3 layer?

(outer layer) → (zona glomerulosa) → Aldosterone

(middle layer) → (zona fasciculata) → cortisol

(inner most layer) → (zona reticularis) → Androgenic

کی (layer) طبقہ تفریق ہے اپنا، فرقوں

① Aldosterone :-

results

water and salt in our body

How??

by increase the retention of sodium and water

*Nour al-Din R.*

\* لو واهم كان عنده مشكلة في اي صبغة فزهاي

الطبقات ~~في~~ راع يصير عنده في تنظيم الستيرويدات في  
الصبغة

1

zona glomerulosa atrophy??

Aldosterone decreases ~~hypoten~~ blood volume  $\downarrow$   
(hypovolemia)

$\downarrow$  concentration of  $Na^+$   $\downarrow$  (hyponatremia)

$\uparrow$  concentration of  $K^+$   $\uparrow$  (hyperkalemia)

يصير عندهم  $\rightarrow$  acidosis

\* الناس يلي يصير عندهم  
زيادة في  $K^+$  راع يصير عندهم  
زيادة  $H^+$

2

decrease of cortisol  $\downarrow$

decrease blood glucose  $\downarrow$  lipolysis, proteinolysis

3

Androgen :- more important For female than male

Female's adrenal  
cortex



cortisol :- Hyperpigmentation 'skin

مرض البهاق التصبغ

cortisol → pituitary gland يحفز

increase ↑ → Melanocyte stimulating hormone.

90% genetic يكون

cortisol فحص نسبة  
Saliva / urine

بهم ليرجوا ال Cause - إذا

Primary, secondary كيف !!

• يوظف اشئ يزيد من إنتاج cortisol

↳ dexamethasone (مثان)

↳ pituitary gland يزيد من هرمون

↳ ACTH increase, thyroid gland ↑

\* في حالة ال dexamethasone نسبة cortisol ↑

المشكلة في thyroid gland

ACTH

طبيياً

cortisol

إذا كان

Primary

21 يكون

↓

ACTH

بيئاً

cortisol

إذا كان

Secondary

21 يكون

↓

Pituitary gland

①

Thyroid gland

②

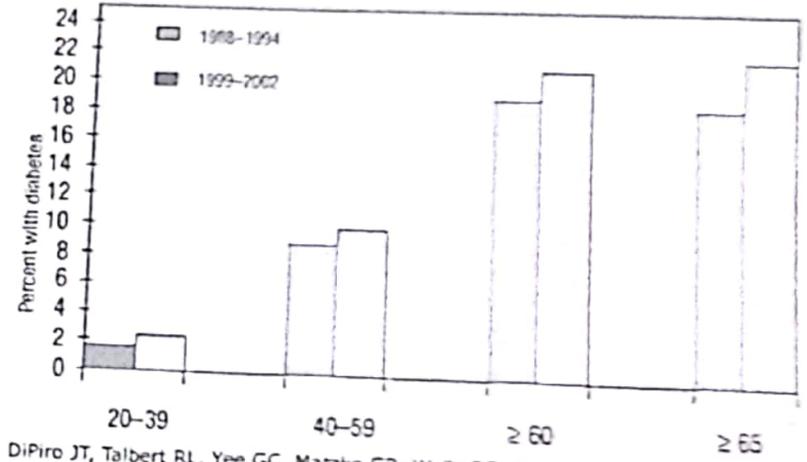
المشكلة 3

# Diabetes Mellitus

(hyperglycemia)

two types  
 الانتفاخ في مستوى  
 السكر

Diabetes mellitus is a group of metabolic disorders sharing the common underlying feature of hyperglycemia. Chronic hyperglycemia and attendant metabolic dysregulation may be associated with secondary damage in multiple organ systems, especially the kidneys, eyes, nerves, and blood vessels.



DiPiro JT, Talbert RL, Yee GC, Matzke GR, Wells BG, Posey LM: Pharmacotherapy: A pathophysiologic Approach, 7th Edition: <http://www.accesspharmacy.com>  
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Notes في السكري يمكن ياتر على organ الموجودة في الجسم اي الكلى , eye يصير عند هبوط

عن الالتهق / الالتهق في القرع type one  
 ارتباك في الحبر type two  
 Insulin dependte ← type one  
 Insulin independent ← type two

17  
 لكن اخطر ثلاث سنوات  
 صبار ! انه في type one  
 يصير عند الكلى  
 type two  
 يصير عند الكلى

➤ B-cells express an ATP-sensitive  $K^+$  channel on the membrane, which comprises two subunits: an inward rectifying  $K^+$  channel (kir6.2) and the sulfonylurea receptor (SUR1), the latter being the binding site for oral hypoglycemic agents (sulfonylureas) used in the treatment of diabetes.

➤ Metabolism of glucose by glycolysis generates ATP, increasing  $\beta$ -cell cytoplasmic ATP/ADP ratios. This inhibits the activity of the ATP-sensitive  $K^+$  channel, leading to membrane depolarization and the influx of extracellular  $Ca$  through voltage-dependent  $Ca$  channels. The resultant increase in intracellular  $Ca$  stimulates the secretion of insulin, presumably from stored hormone within the  $\beta$ -cell granules. This is the phase of the immediate release of insulin.

➤ If the secretory stimulus persists, a delayed and protracted response follows that involves the active synthesis of insulin. Other factors, including intestinal hormones and certain amino acids (leucine and arginine), also stimulate insulin release, but not its synthesis.

Handwritten notes in Arabic:

ما يدخل Glucose على الخلايا  $\beta$  يثبط  $K^+$  pump  
 بوتاسيوم، يقع الكالسيوم يدخل تفتق  
 افراز الانسولين.

على سطح  $\beta$  cell  
 Pump  $K^+$   
 Pump sulfonylureas  
 Pump  $Ca^{++}$



# Classification

Majority of diabetics are classified into 2 categories:

Type 1: absolute deficiency of insulin.

Type 2: the presence of insulin resistance with reduced insulin secretion.

Gestational diabetes:

Triggered by the stress of pregnancy.

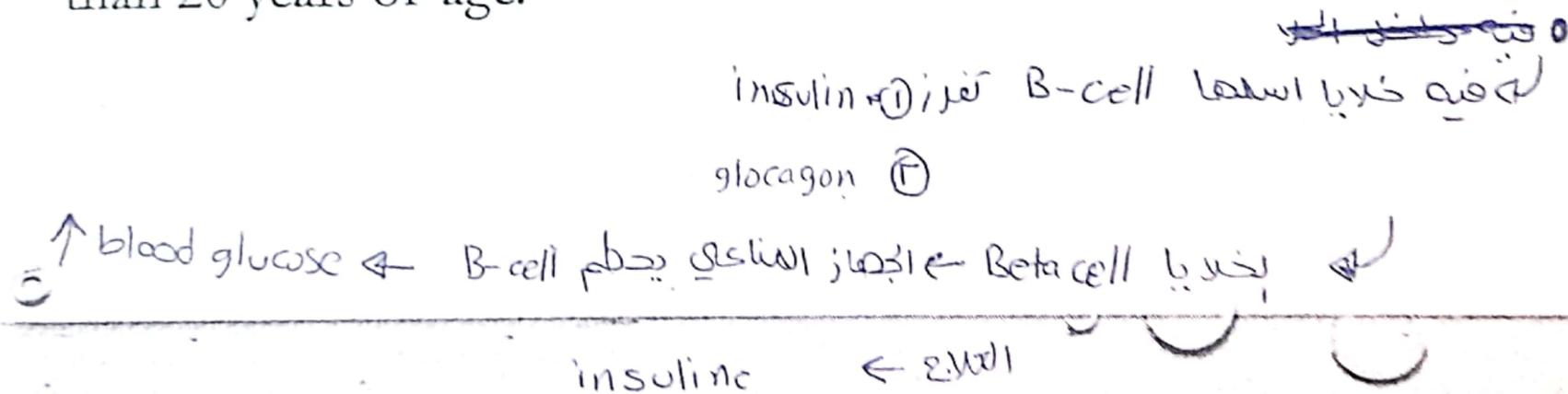
Other specific types:

Infections, drugs, endocrinopathies, pancreatic destruction, genetic defects.

# Classification

## Type 1 diabetes

An autoimmune disease characterized by pancreatic  $\beta$ -cell destruction and an absolute deficiency of insulin. It accounts for approximately 5% to 10% of all cases and is the most common subtype diagnosed in patients younger than 20 years of age.



# Symptoms

- The onset is marked by polyuria, polydipsia, polyphagia, and, when severe, ketoacidosis, all resulting from metabolic derangements. *Since insulin is a major anabolic hormone in the body, deficiency of insulin results in a catabolic state that affects not only glucose metabolism but also fat and protein metabolism.*
- Unopposed secretion of counter-regulatory hormones (glucagon, growth hormone, epinephrine) also plays a role in these metabolic derangements. The assimilation of glucose into muscle and adipose tissue is sharply diminished or abolished. Not only does the storage of glycogen in the liver and muscle cease, but also reserves are depleted by glycogenolysis. *The resultant hyperglycemia exceeds the renal threshold for reabsorption, and glycosuria ensues. The glycosuria induces an osmotic diuresis and thus polyuria, causing a profound loss of water and electrolytes.*

glucose ↑ → glucose blood ↑ → glucose urin ↑

polyuria بول كثير ، بول كثير

glycosuria

# Symptoms

- The obligatory renal water loss combined with the hyperosmolarity resulting from the increased levels of glucose in the blood tends to deplete intracellular water, *triggering the osmoreceptors of the thirst centers of the brain. In this manner, intense thirst (polydipsia) appears.*
- With a deficiency of insulin, the scales swing from insulin-promoted anabolism to catabolism of proteins and fats. Proteolysis follows, and the gluconeogenic amino acids are removed by the liver and used as building blocks for glucose. *The catabolism of proteins and fats tends to induce a negative energy balance, which in turn leads to increasing appetite (polyphagia), thus completing the classic triad of diabetes: polyuria, polydipsia, and polyphagia.* Despite the increased appetite, catabolic effects prevail, resulting in weight loss and muscle weakness. *The combination of polyphagia and weight loss is paradoxical and should always raise the suspicion of diabetes.*



# Diabetic ketoacidosis

- *The second major effect of an alteration in the insulin-to-glucagon ratio is activation of the ketogenic machinery.*
  - Insulin deficiency stimulates lipoprotein lipase, with the resultant breakdown of adipose stores, and an increase in levels of free fatty acids. When these free fatty acids reach the liver, they are esterified to fatty acyl CoA.
  - Oxidation of fatty acyl CoA molecules within the hepatic mitochondria produces ketone bodies (acetoacetic acid and  $\beta$ -hydroxybutyric acid).
  - The rate at which ketone bodies are formed may exceed the rate at which they can be utilized by peripheral tissues, leading to *ketonemia* and *ketonuria*.
  - If the urinary excretion of ketones is compromised by dehydration, systemic *metabolic ketoacidosis* results. Release of ketogenic amino acids by protein catabolism aggravates the ketotic state
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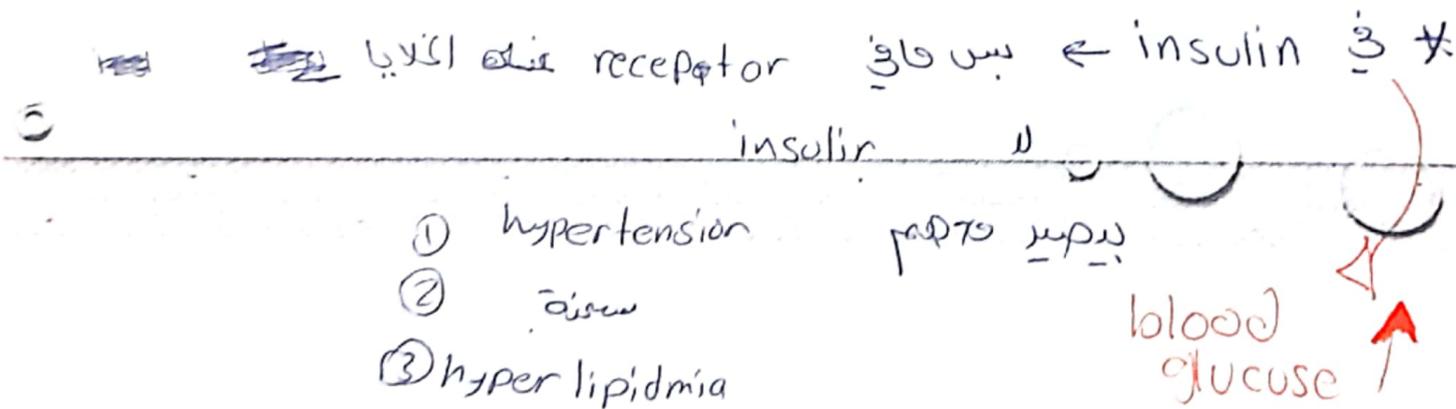
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# Classification

## Type 2 diabetes

- *Type 2 diabetes* is caused by a combination of peripheral resistance to insulin action and an inadequate secretory response by the pancreatic  $\beta$  cells ("relative insulin deficiency").
- Approximately 90% to 95% of diabetic patients have type 2 diabetes, and the vast majority of such individuals are **overweight**.
- Although classically considered "adult-onset," the prevalence of type 2 diabetes in children and adolescents is increasing at an alarming pace



# Prediabetes <sup>↑ high risk</sup> ← احتمال حدوث سكري

Levels of blood glucose proceed along a continuum. Individuals with fasting glucose concentrations less than 100 mg/dl, or less than 140 mg/dl following an OGTT, are considered to be euglycemic. However, those with fasting glucose concentrations greater than 100 mg/dl but less than 126 mg/dl, or OGTT values greater than 140 mg/dl but less than 200 mg/dl, are considered to have impaired glucose tolerance, also known as "pre-diabetes."

Pre-diabetic individuals have a significant risk of progressing to overt diabetes over time, with as many as 5% to 10% advancing to diabetes mellitus per year. In addition, pre-diabetics are at risk for cardiovascular disease, as a result of the abnormal carbohydrate metabolism as well as the coexistence of other risk factors such as low levels of high-density lipoprotein, hypertriglyceridemia, and increased plasminogen activator inhibitor-1 (PAI-1).

Fastening glucose ← يتم حسابه أثناء الصيام → أصصاوا

Random glucose ← يتم حسابه بشكل عشوائي في اليوم .

Oral glucose Tolerance ← يتم اعطاه وحصاوا يتم حسابه عتدوا سلعين

Hb A1c في فحص لى شخاص منهم سكري يعولوا كل ثلثة أشهر . هو why ?

## Complications

لنصصاوا يرتبط مع  
الهيوعلوبين و الازتبط

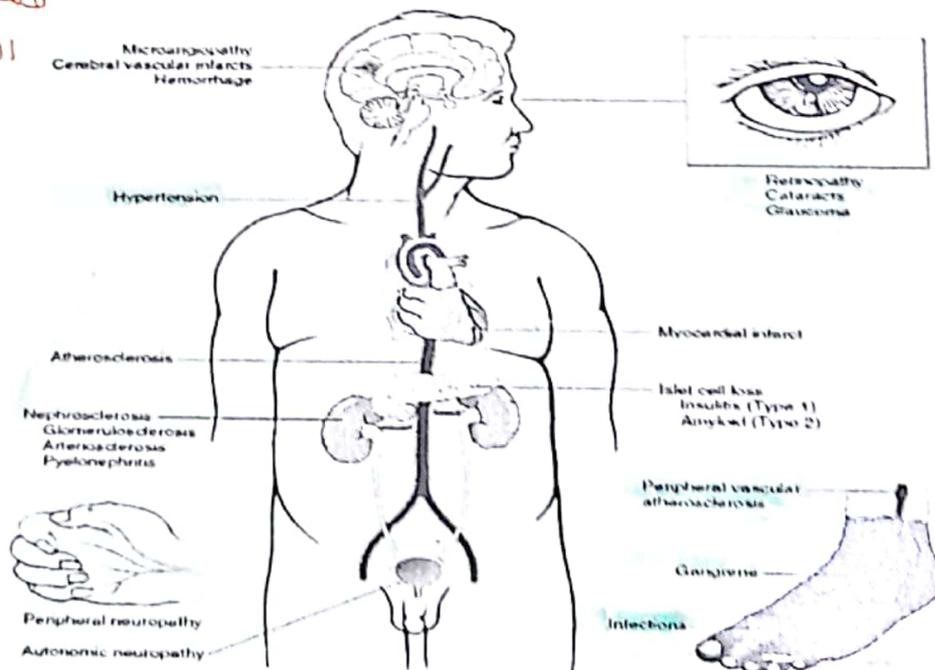


FIGURE 24-32 Long-term complications of diabetes

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