وزارة الصحة
Ministry of Health

التجمع الصحي بالأحساء
Al-Ahsa Health Cluster

DM Complications

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رؤية VISION 2030
المملكة العربية السعودية
KINGDOM OF SAUDI ARABIA

صدقة جارية

تجمع الأحساء الصحي
AlAhsa Health Cluster

Dr. Majdi Allasim

بِسْمِ ٱللَّهِ ٱلرَّحْمَٰنِ ٱلرَّحِيمِ

وَٱخْفِضْ لَهُمَا جَنَاحَ ٱلذُّلِّ مِنَ ٱلرَّحْمَةِ

وَقُل رَّبِّ ٱرْحَمْهُمَا

كَمَا رَبَّيَانِى صَغِيرًا

OBJECTIVES

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1. **To understand the symptoms that raise suspicion of acute DM complications:**
   - Hypoglycemia
   - Diabetic ketoacidosis (DKA)
   - Hyperglycemic hyperosmolar non-ketotic state (HHNS)
2. **To define important urgent lab workups in case of acute DM complications.**
3. **To understand the role of PHC physicians in case of acute DM complications.**
4. **To gain the knowledge how to screen for chronic DM complications:**
   - **WHAT?** (Micro and Macro)
   - **WHEN?** (How often you should do the screening)
   - **HOW?** (Physical exam and lab tests)
5. **To define the criteria of referral of chronic DM complications to sub-specialties:**
   - **RETINOPATHY** → fundoscopy by ophthalmologist
   - **NEUROPATHY** → DM foot examination
   - **NEPHROPATHY** → how to calculate GFR, when to refer to nephrologist
   - **CORONARY ARTERY DISEASE (CAD)** → Stress ECG
   - **PERIPHRAL ARTERY DISEASE (PAD)** → ankle-brachial pressure index, when to refer to vascular surgery.

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OVERVIEW

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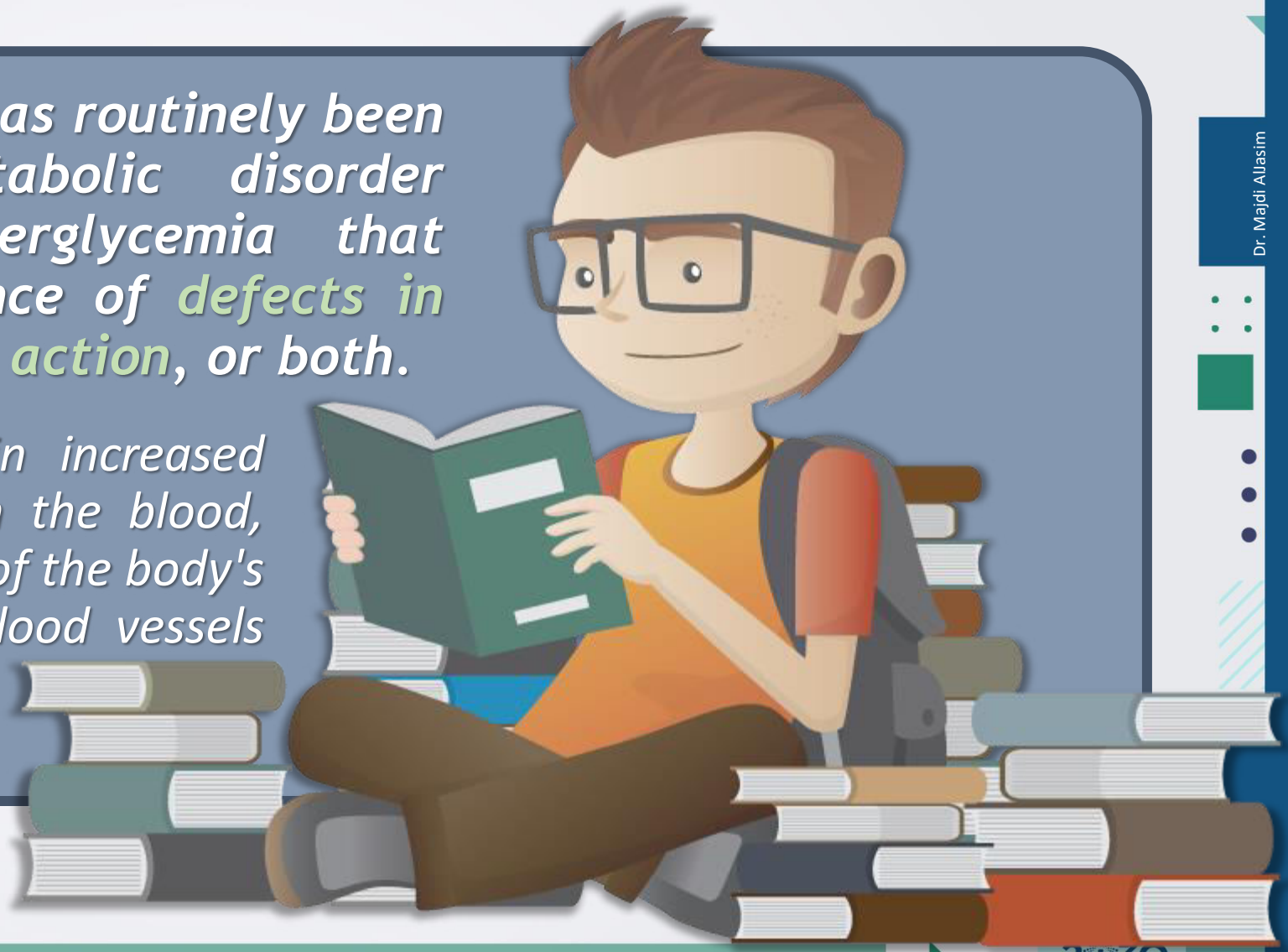
Diabetes mellitus (DM) has routinely been described as a metabolic disorder characterized by hyperglycemia that develops as consequence of defects in insulin secretion, insulin action, or both.

Such a deficiency results in increased concentrations of glucose in the blood, which in turn damage many of the body's systems, in particular the blood vessels and nerves.



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COMPLICATIONS OF DM

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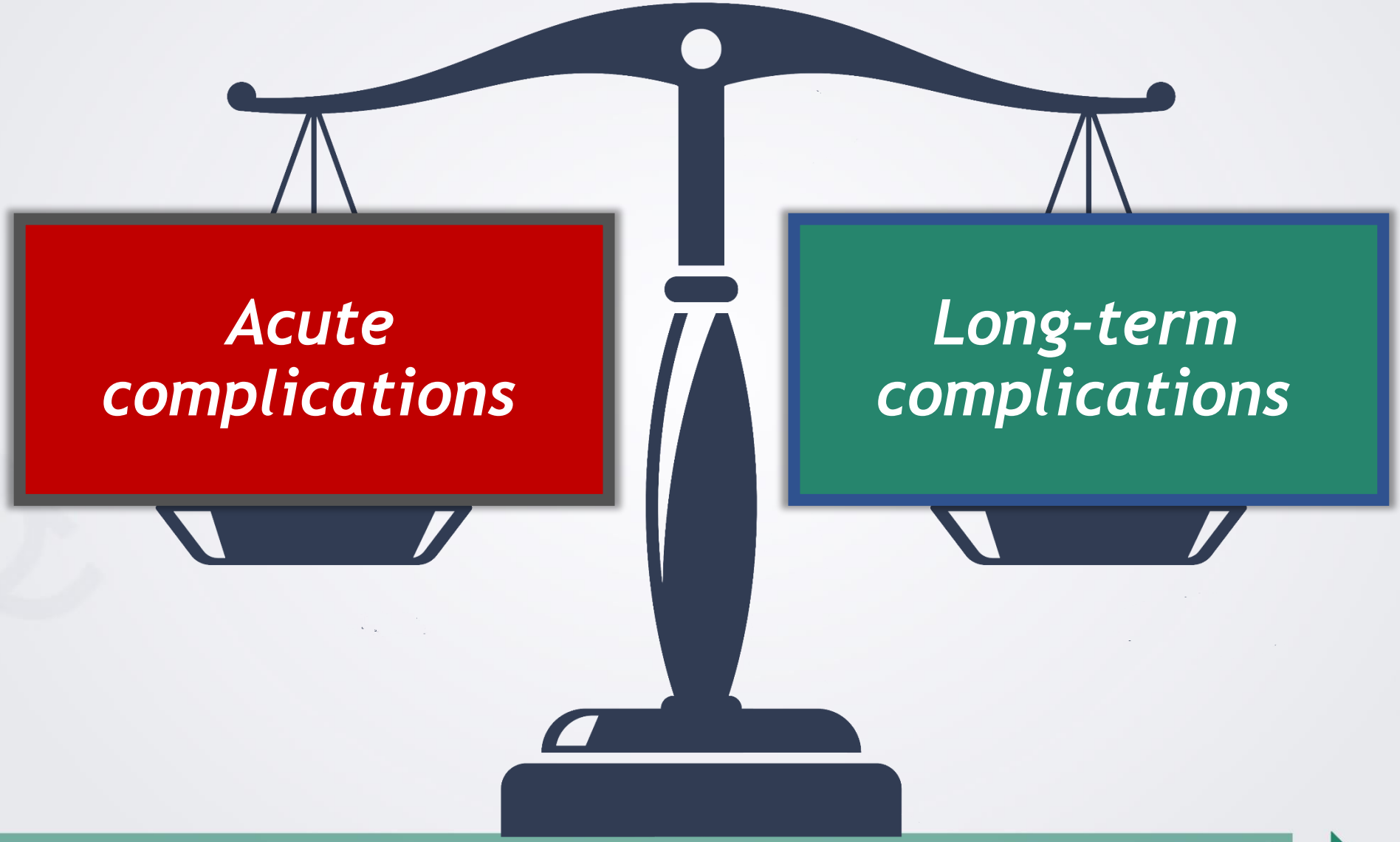

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ACUTE COMPLICATIONS OF DM

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There are three major acute complications of diabetes related to short-term imbalances in blood glucose levels:

- ***Hypoglycemia*** (abnormal low blood glucose level)
- ***Diabetic Ketoacidosis*** (DKA)
- ***Hyperglycemic hyperosmolar non-ketotic state*** (HHNS)

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LONG-TERM COMPLICATIONS OF DM

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Long-term complications are becoming more common as more people live longer with diabetes. The long-term complications of diabetes can affect almost every organ system of the body.

Long-term complications are seen in both type 1 and type 2 diabetes but usually do not occur within the first 5 to 10 years of the diagnosis, and it is divide into:

1. ***Microvascular complications:*** *Affecting small blood vessels.*
2. ***Macrovascular complications:*** *Affecting large blood vessels.*

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LONG-TERM COMPLICATIONS OF DM

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Long-term complications of diabetes

Microvascular

Retinopathy

Nephropathy

Neuropathy

Macrovascular

Ischemic heart disease

Cerebral vascular disease

Peripheral artery disease

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LONG-TERM COMPLICATIONS OF DM

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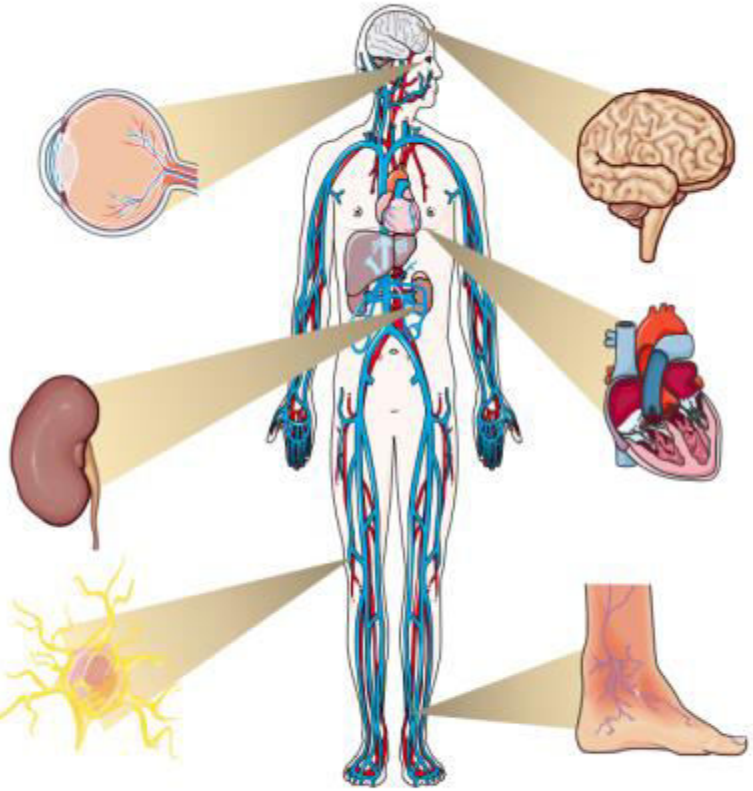
Microvascular

Eye
High blood glucose and high blood pressure can damage eye blood vessels, causing retinopathy, cataracts and glaucoma

Kidney
High blood pressure damages small blood vessels and excess blood glucose overworks the kidneys, resulting in nephropathy.

Neuropathy
Hyperglycemia damages nerves in the peripheral nervous system. This may result in pain and/or numbness. Feet wounds may go undetected, get infected and lead to gangrene.



Macrovascular

Brain
Increased risk of stroke and cerebrovascular disease, including transient ischemic attack, cognitive impairment, etc.

Heart
High blood pressure and insulin resistance increase risk of coronary heart disease

Extremities
Peripheral vascular disease results from narrowing of blood vessels increasing the risk for reduced or lack of blood flow in legs. Feet wounds are likely to heal slowly contributing to gangrene and other complications.

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Acute Complications

HYPOGLYCEMIA

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Hypoglycemia: overview

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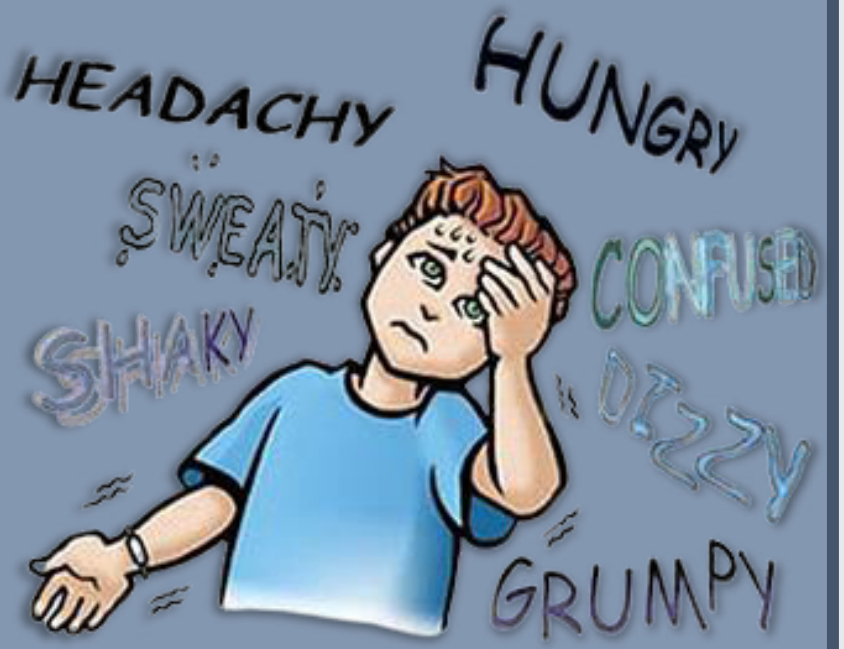
Hypoglycemia *(abnormally low blood glucose level) occurs when the blood glucose falls to less than 70 mg/dL (3.9 mmol/L).*

Level	Definition	Glucose Value
1	Hypoglycemia alert value (Take action)	< 70mg/dL
2	Clinically significant hypoglycemia	< 54mg/dL
3	Severe hypoglycemia	No specific glucose threshold hypoglycemia. Associated with severe cognitive impairment requiring external assistance for recovery



It can be caused by too much insulin or oral hypoglycemic agents, too little food, or excessive physical activity.

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Hypoglycemia: symptoms

The clinical manifestations of hypoglycemia may be grouped into two categories:

- *Adrenergic symptoms*
- *Central nervous system symptoms*

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Hypoglycemia: symptoms

Adrenergic symptoms

In mild hypoglycemia, as blood glucose level falls, the sympathetic nervous system is stimulated, resulting in a surge of epinephrine and norepinephrine

- SWEAT
- TREMOR
- PALPITATION
- HUNGER
- NERVOUSNESS

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Hypoglycemia: symptoms

CNS symptoms

In moderate hypoglycemia, the fall in blood glucose deprives brain cells of glucose needed for functioning:

- CONFUSION
- HEADACHE
- DROWSINESS
- IMBALANCE
- LIGHT HEADEDNESS

Hypoglycemia: symptoms

CNS symptoms

In severe hypoglycemia, CNS function is so impaired that the patient needs the assistance of another person for treatment of hypoglycemia

COMA

DIFFICULT SLEEP AROUSING

SEIZURE

DISORIENTATION

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Hypoglycemia: management

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IMMEDIATE TREATMENT MUST BE GIVEN WHEN HYPOGLYCEMIA OCCURS

In conscious patient, give 15g of fast-acting concentrated carbohydrate orally; like:

- *3-4 commercially prepared glucose tablets.*
- *150-200mL of fruit juice or regular soda.*
- *2-3 teaspoons of regular sugar or honey.*
- *6-10 pieces of hard candies.*

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Hypoglycemia: management

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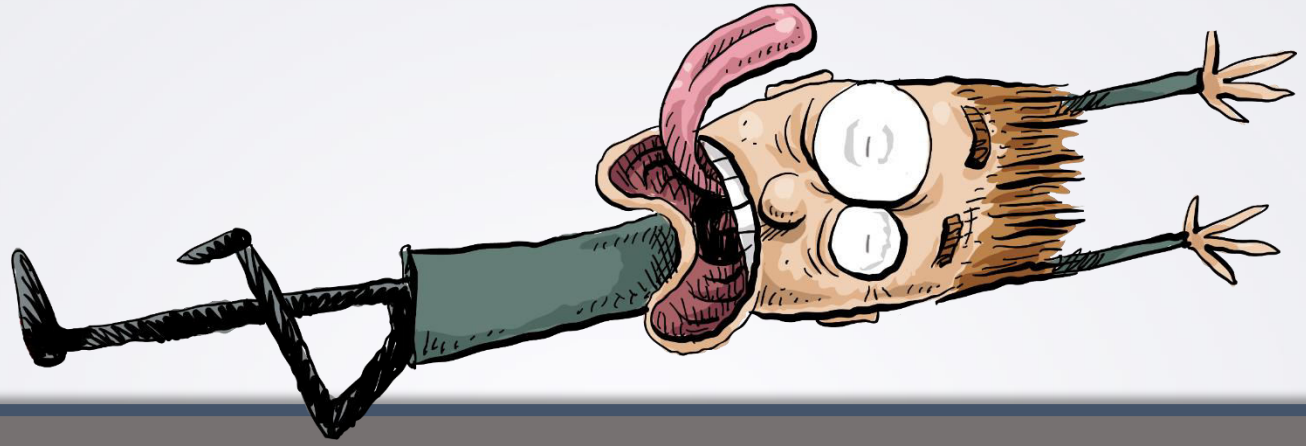

- ***Glucometer is available:*** *Retest blood glucose level every 15 minutes and retreated if it is less than 70 mg/dL (3.8 mmol/L).*
- ***Glucometer is non-available:*** *If the symptoms persist more than 10 to 15 minutes after initial treatment, the treatment is repeated even if blood glucose testing is not possible.*
- *Once the symptoms resolve, a snack containing protein and starch (e.g., milk or cheese and crackers) is recommended.*

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Hypoglycemia: management

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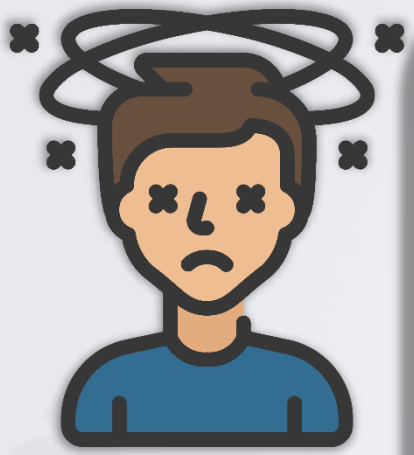
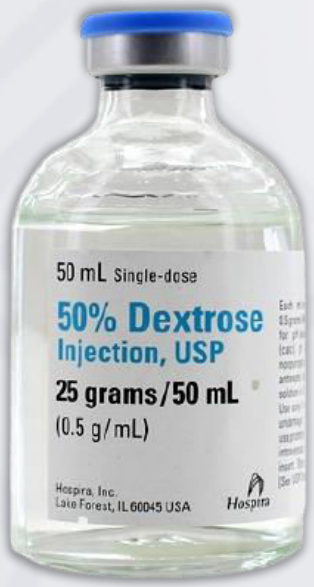
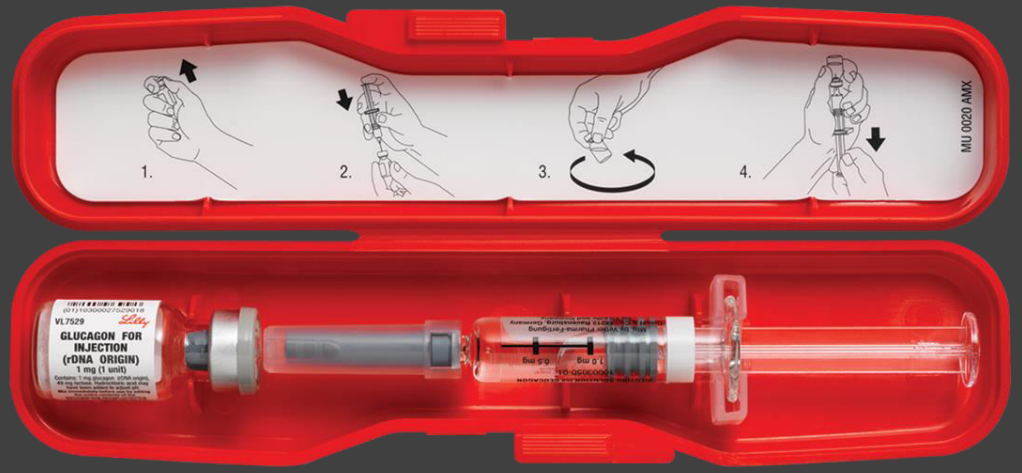




If the patient is unconscious or can not swallow:

- *In non-health care institute: 1mg vial of Glucagon IM or SC is given. The effect is seen within 20 min.*



- *In health care institute: 25-50mL of 50% dextrose IV. The effect is seen in short time.*

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Acute Complications

Diabetic Ketoacidosis

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DKA: overview

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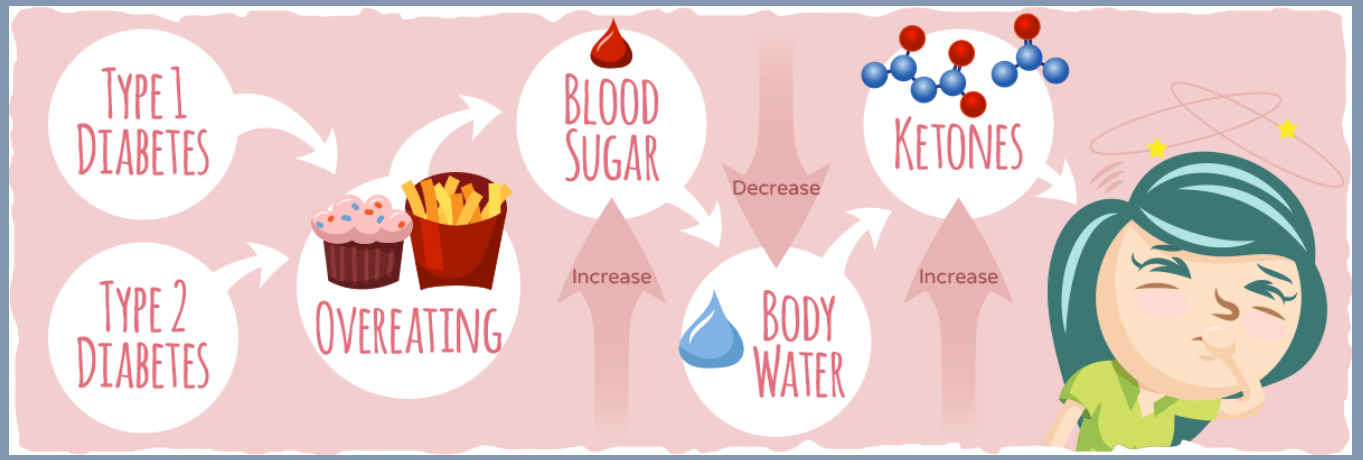
DKA is caused by absence or markedly inadequate amount of insulin. This deficit in insulin results in disorders in metabolism of carbohydrate, protein and fat. It is more common in type1 DM.

The three main clinical feature of DKA are:

1. *Hyperglycemia.*
2. *Dehydration and electrolytes imbalance.*
3. *Acidosis.*



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DKA: pathophysiology

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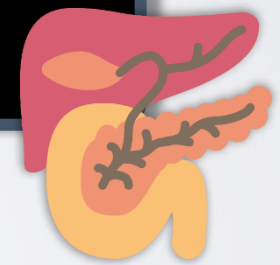
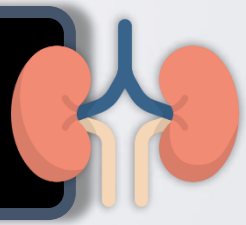
Insufficient insulin → ↓ glucose entering cells and ↑ liver gluconeogenesis.



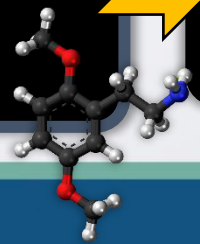
MARKED HYPERGLYCEMIA

Kidneys try to get-rid of excessive glucose → polyuria (glucose excretion with water and electrolytes).



DEHYDRATION
ELECTROLYTE IMBALANCE

Build up of acidic ketone bodies from liver



METABOLIC ACIDOSIS

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DKA: precipitating factors

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The three main causes of DKA are:

1. Decreased or missed dose of insulin.

2. Infection → ↑ stress hormones (cortisone, adrenalin, glucagon, growth hormone → hyperglycemia → unadjusted insulin dose → DKA)

3. Undiagnosed type 1 DM cases (usually DKA is the first manifestation in type 1 DM)

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DKA: symptoms

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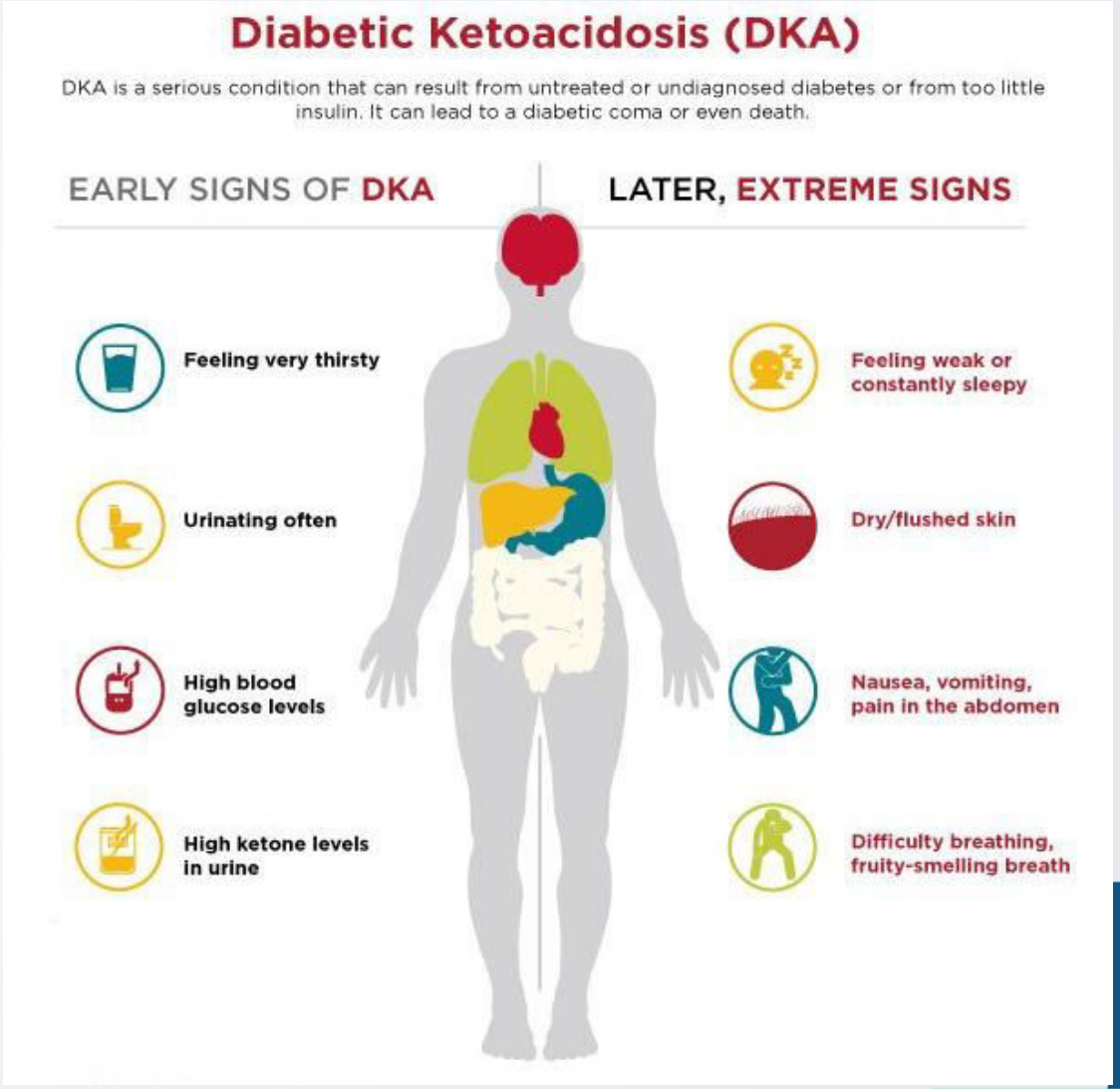
- *Hyperglycemia leads to polyuria, polydipsia, blurred vision, weakness and headache.*
- *Dehydration leads to orthostatic hypotension.*
- *Ketone bodies and acidosis lead to GI symptoms like nausea, vomiting, abdominal pain.*
- *Ketone bodies build-up lead to acetone breath (fruity odor).*



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DKA: management

In addition to treat hyperglycemia, management of DKA is aimed to correcting dehydration, electrolytes loss and acidosis.

Your role in PHC:

When you suspect patient with DKA, do **urgent RBS** and **urine dipstick for ketones**. Once you have ↑ RBS and positive urine ketone, your patient is most likely in DKA and you need ABG to confirm acidosis.

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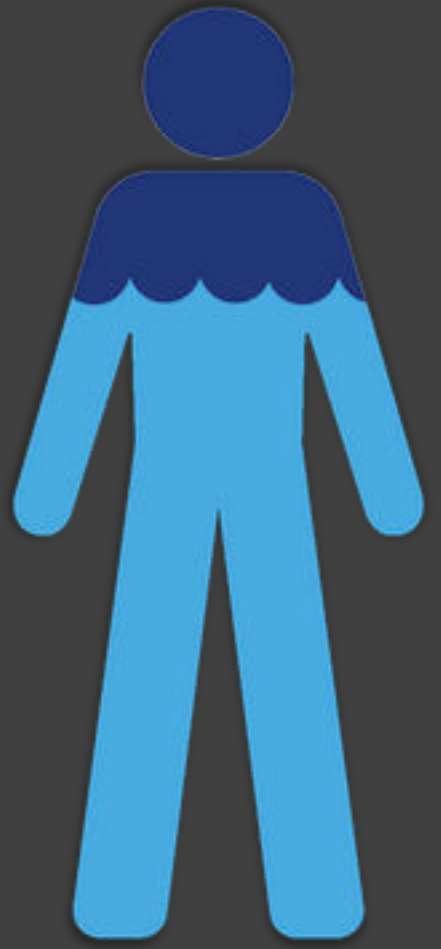
DKA: management

Treatment in PHC:

The patient needs **urgent referral to ER**.

Establish IV cannula for IV fluid replacement.

- Always start with 0.9% normal saline.
- Avoid large fluid boluses unless patient is orthostatic or in shock.
- If blood sugar gets bellow 250mg/dL (or falls more than 90mg/dL in one hour) → add D5 or D10 to IVF.
- Rate of IVF = deficit + Maintenance / hr



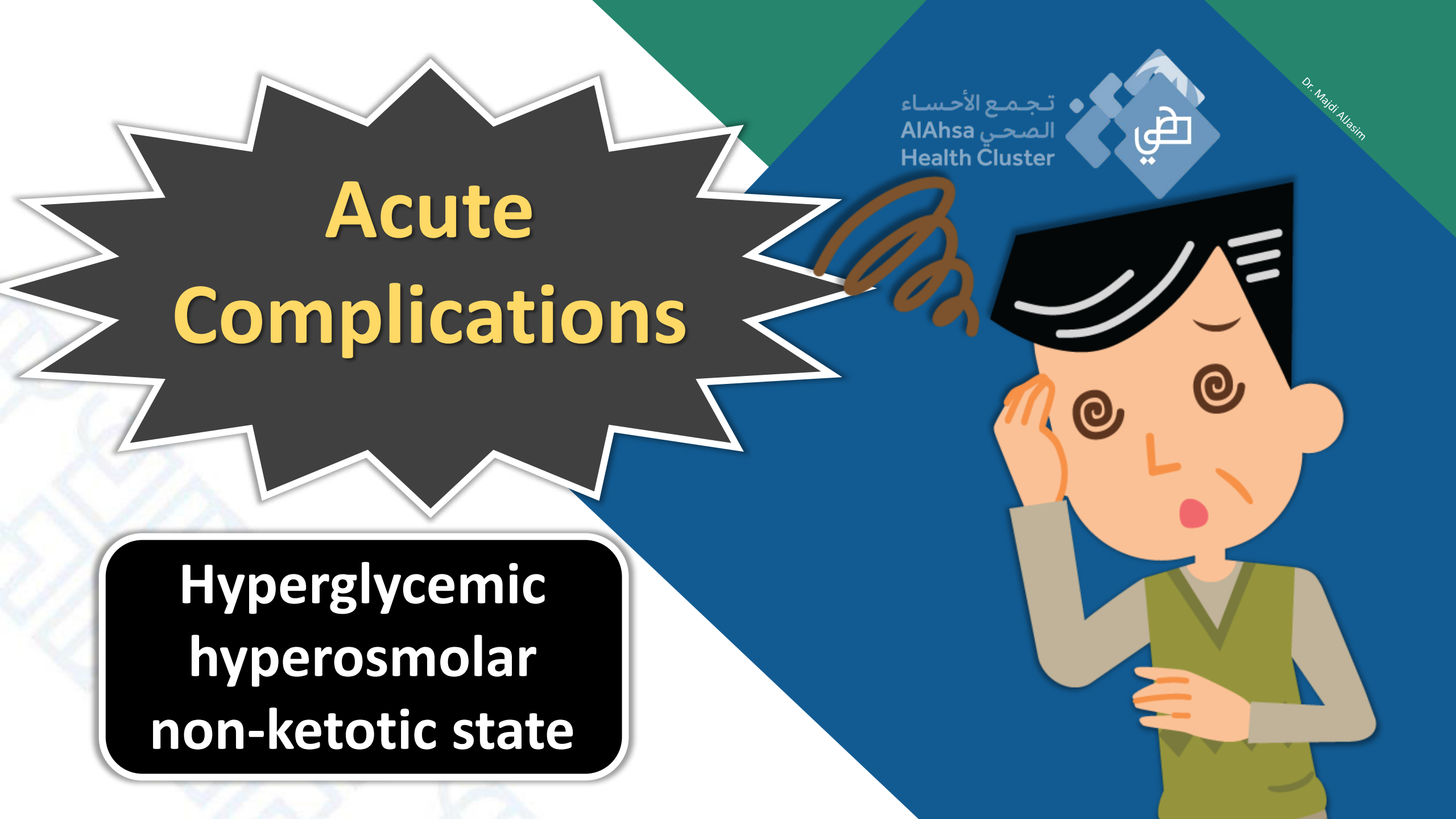
DKA: management

DO NOT:

- Don’t give insulin unless you correct hemodynamic instability and hypokalemia.
- Don’t give insulin as SC bolus, always give insulin as infusion (0.1u/kg/hr) after 1-2 hrs of starting IVF.

Acute Complications

Hyperglycemic hyperosmolar non-ketotic state



HHNS: overview

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HHNS *is characterized by* ***hyperglycemia*** *(blood glucose >600 mg/dL),* ***hyperosmolarity*** *(plasma osmolarity >310 mOsm/L) and* ***dehydration****, in* ***absence of ketoacidosis****.*

It mostly happens in elderly patients.

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HHNS: manifestations

MARKED HYPERGLYCEMIA

Polyuria

Dehydration

Hypotension, tachycardia

↑ serum osmolarity → pulling water out of body cells including brain cells.

↑ Risk of thromboembolic events

Neurological manifestations like seizure, hemiparesis, aphasia, nystagmus, visual hallucination, visual field defect… etc

HHNS: management

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Your role in PHC:

When you suspect patient with DKA, do **urgent RBS**, **urine dipstick for ketones**, **serum osmolarity** (to calculate it, you will need BUN, Na and glucose).

Once you have ↑ RBS, negative urine ketone, and ↑ serum osmolarity, your patient is most likely in HHNS and you need to refer the case to ER.

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HHNS: management

Treatment in PHC:

Same as that for DKA

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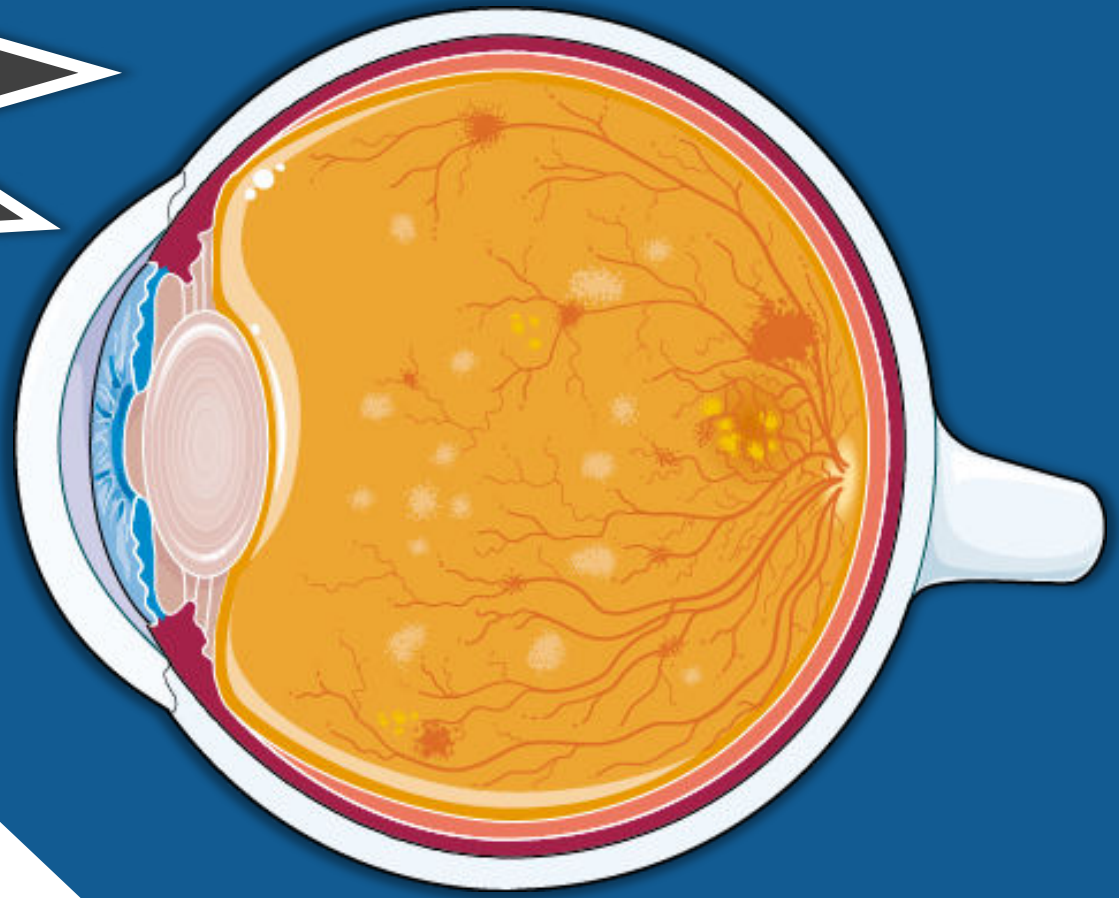
Long-term Complications



Microvascular Complications: RETINOPATHY

Retinopathy: overview

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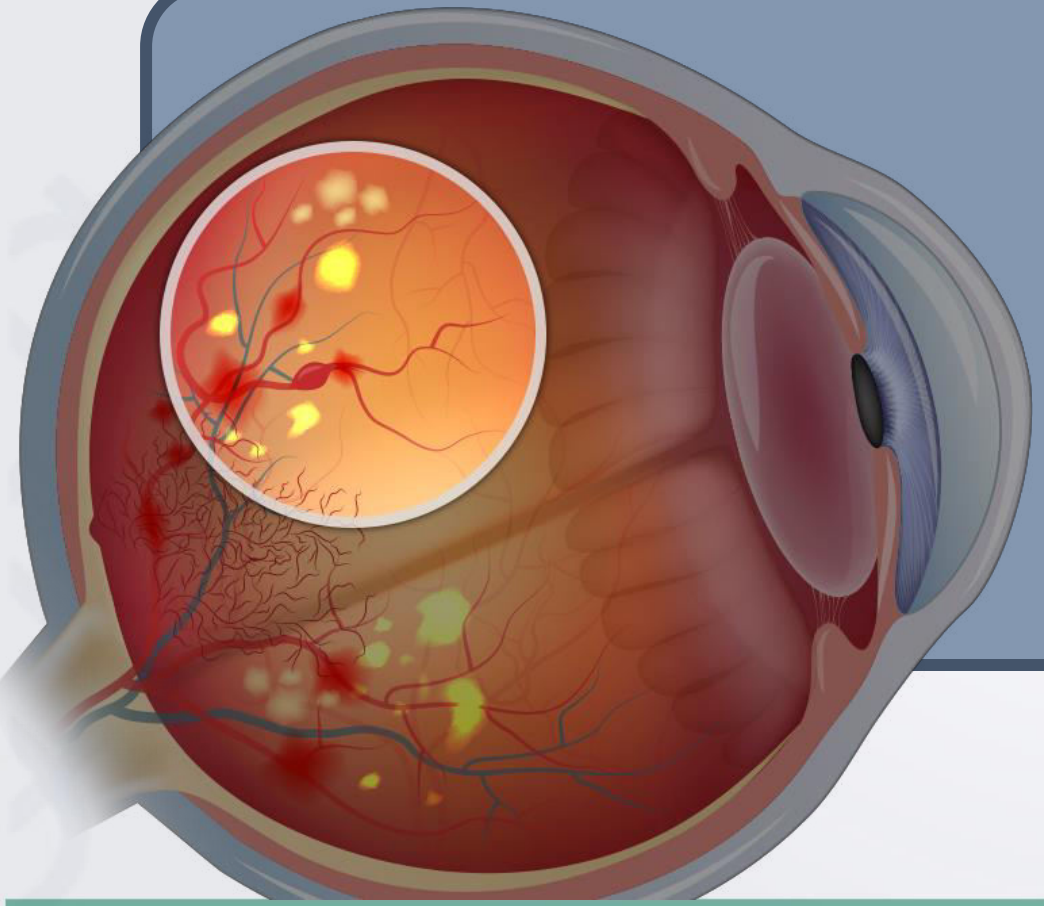

Diabetic retinopathy *is a leading cause of blindness and visual disability.*

It is caused by small blood vessel damage to the back layer of the eye, the retina, leading to progressive loss of vision, even blindness.

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Retinopathy: Types

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There are two major types of diabetes retinopathy:

Non-proliferative

(micro-aneurysm → leakage → hard and soft exudate and dot hemorrhage)

Proliferative

(formation of new blood vessels → rupture → bleeding)

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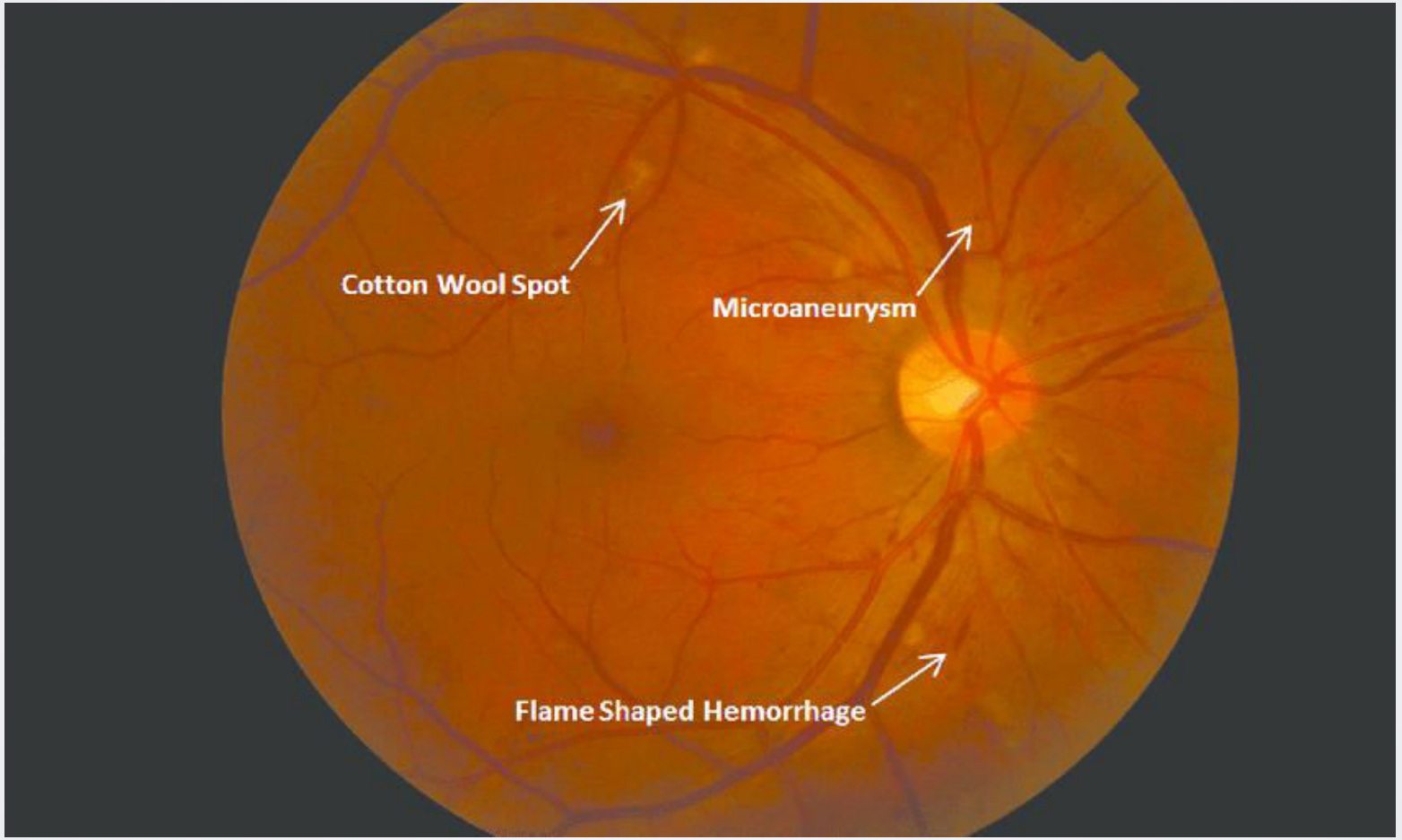
Retinopathy: Types



Non-proliferative diabetic retinopathy

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Retinopathy: Types

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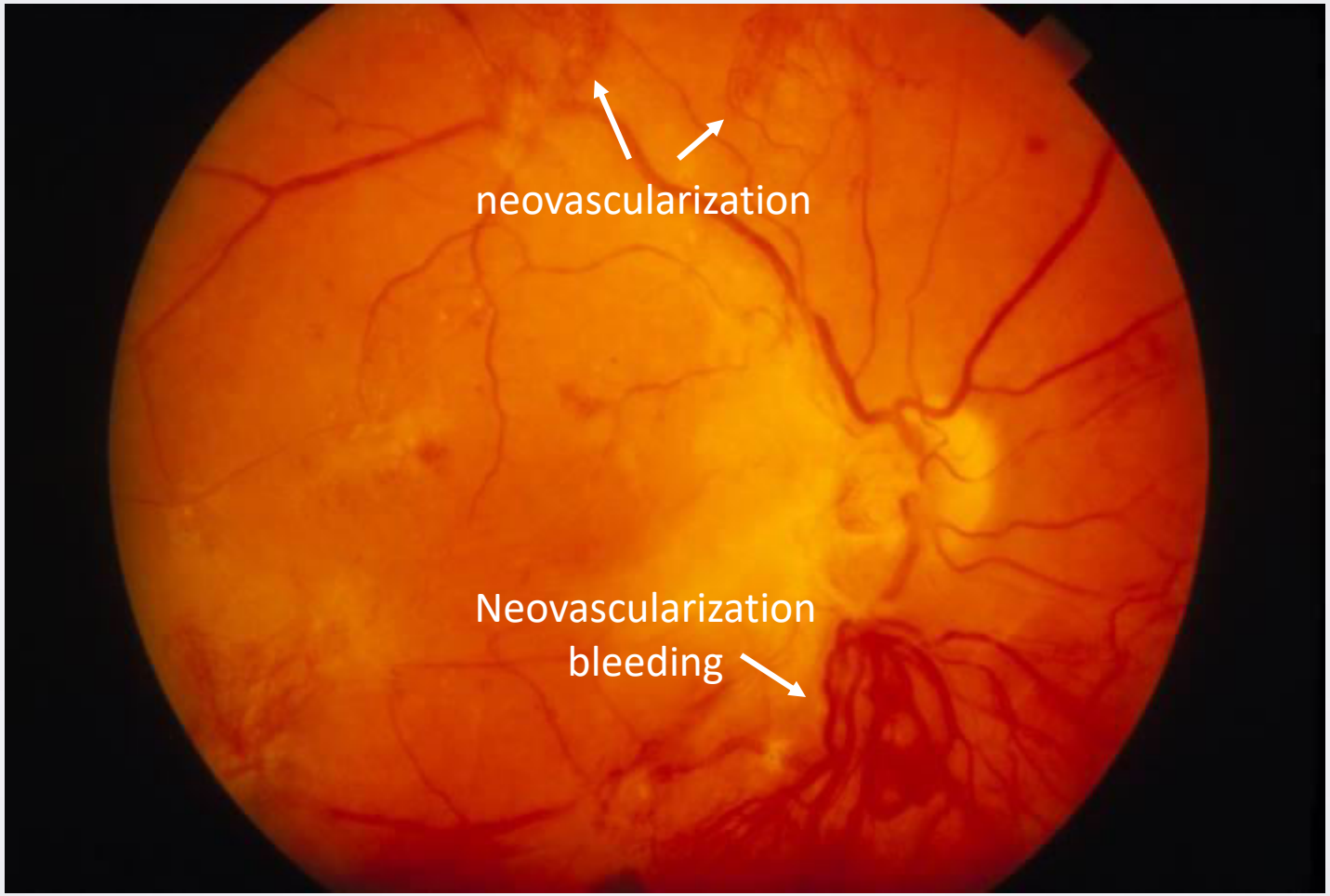

Proliferative diabetic retinopathy

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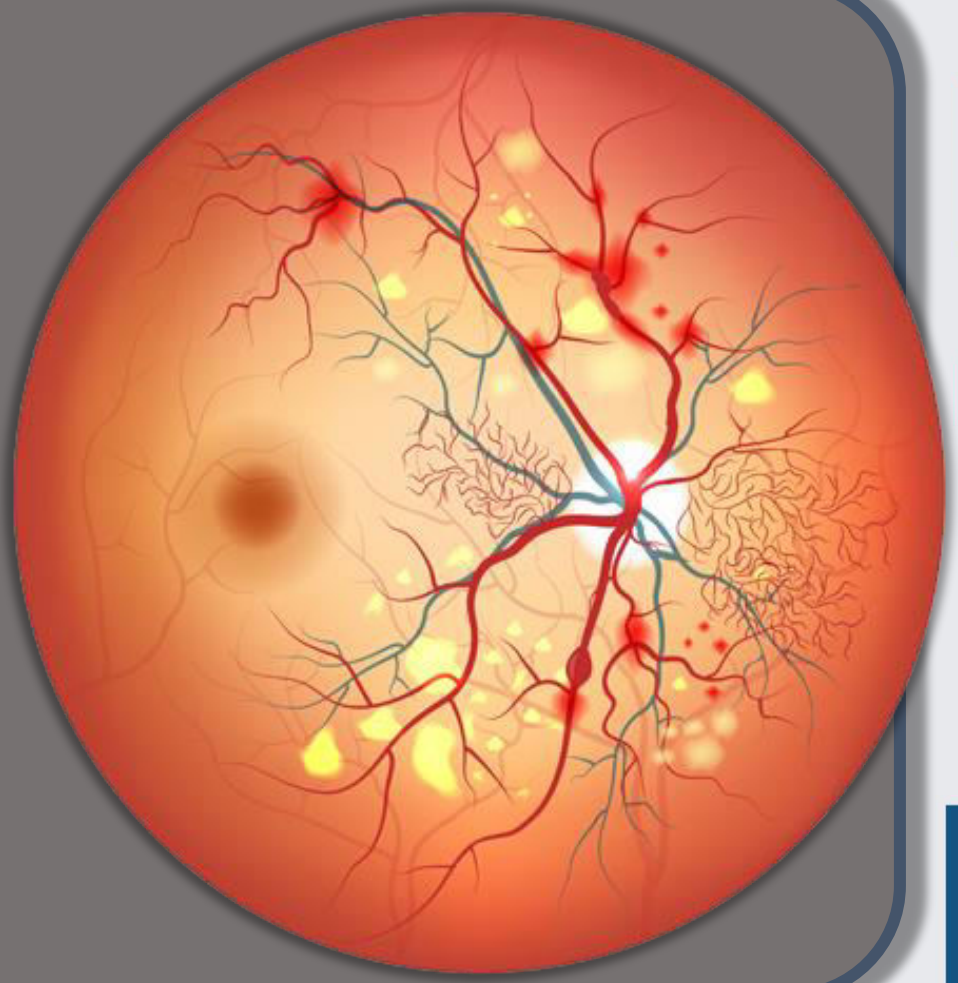
Retinopathy: Symptoms

- ***In non-proliferative retinopathy:***
  - ✓ *Usually it is asymptomatic.*
  - ✓ *blurred vision secondary to macular edema.*
- ***In proliferative retinopathy:***
  - ✓ *Fragile new vessels → bleeding into the vitreous → clouds or even blocks the vision.*
  - ✓ *Poor blood supply to retina → scar tissue → retinal detachment.*
  - ✓ *Neovascularization of iris → glaucoma.*



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Retinopathy: Symptoms

Vitreous hemorrhage vision

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Retinopathy: Symptoms

Retinal detachment vision

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Retinopathy: Symptoms

Tunnel vision in glaucoma

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Retinopathy: screening

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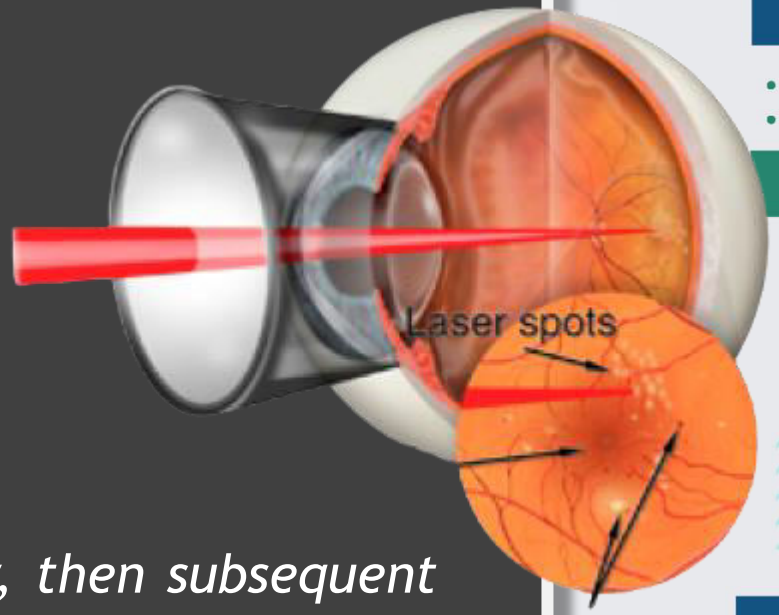
Your role in PHC:

- **For type 1 DM:** Initial referral to ophthalmologist should be done within 5 years after the onsets of type 1 DM.

- **For type 2 DM:** Initial referral to ophthalmologist should be done at the time of diagnosis.



If initial screening showed no evidence of retinopathy, then subsequent referral screening will be done annually.

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Retinopathy: prevention

Prevention:

- Control of blood glucose.
- Control of hypertension.
- Control of lipid.
- Smoking cessation.
- Early detection and treatment of vision-threatening retinopathy.

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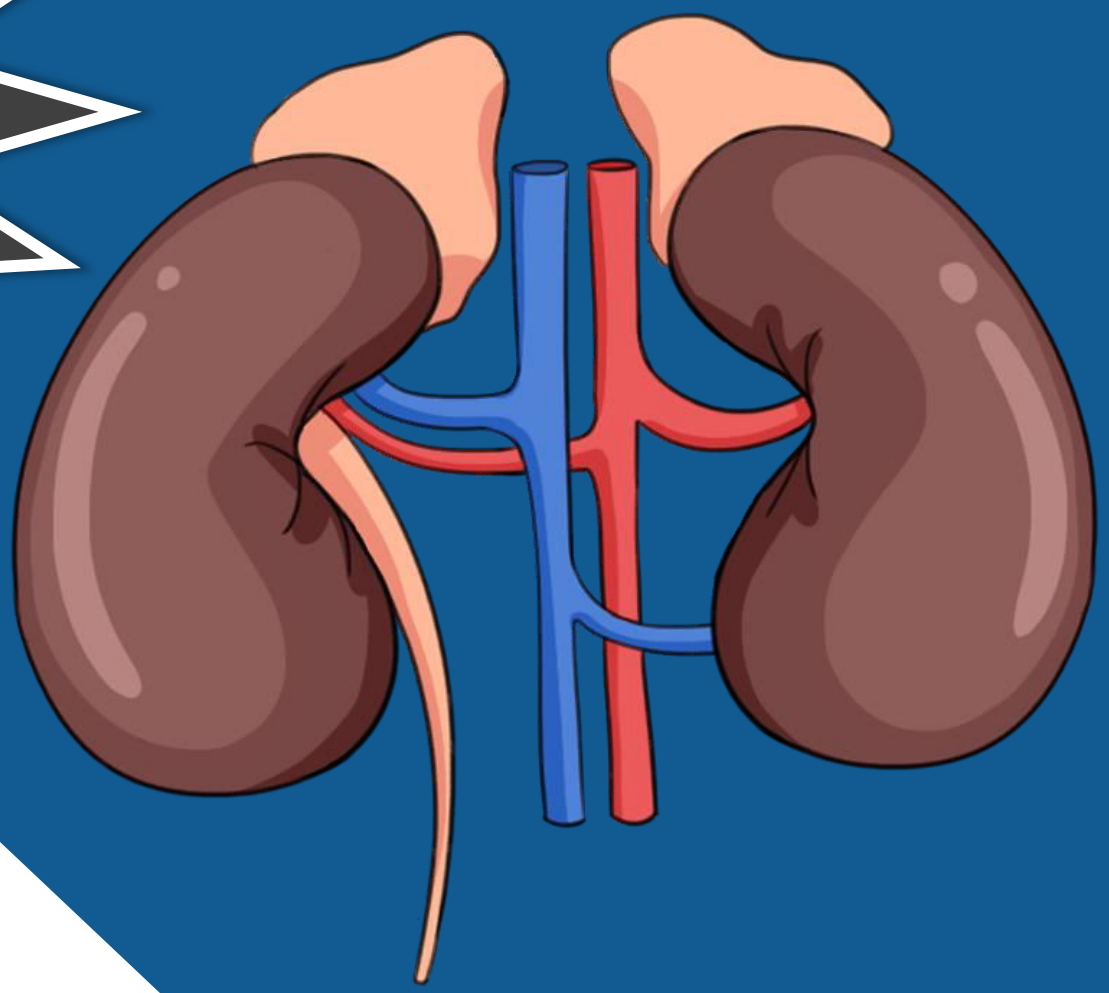
Long-term Complications

Microvascular Complications: NEPHROPATHY



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Nephropathy: overview

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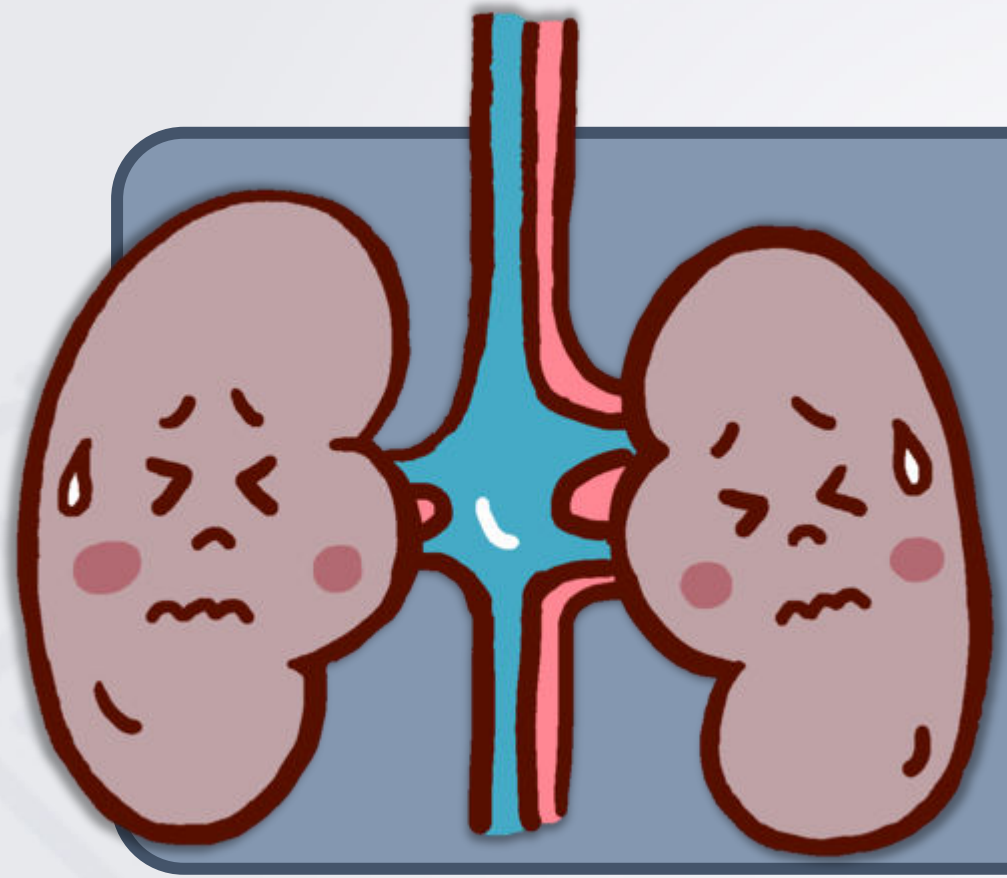

Diabetic nephropathy *is a leading cause of dialysis and kidney transplant.*

The patient is considered to have diabetic nephropathy if values of chronic kidney disease (CKD) persist at least for 3 months "eGFR < 60 and/or UACR ≥ 300mg/g".

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Nephropathy: Symptoms

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The early stages of diabetic nephropathy show no symptoms. As the disease progresses, the following symptoms may appear:

1. Swollen ankle, feet and hands
2. Nausea and loss of appetite
3. Fatigue and insomnia
4. Dry, itchy skin
5. Difficulty in concentration

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Nephropathy: screening

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Screening:

How: *Spot urinary albumin-to-creatinine ratio (UACR) and estimated glomerular filtration rate (eGFR) "calculated by CKD-EPI Creatinine equation".*

WHEN: *Any patient with DM (type 1 and 2) with duration of ≥5 years regardless of treatment.*

FREQUENCY: *Annually if normal tests. Those with UACR ≥300mg/g and/or eGFR ≤60 ml/min/1.73m² should be monitored more frequent to guide therapy.*

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Nephropathy: screening

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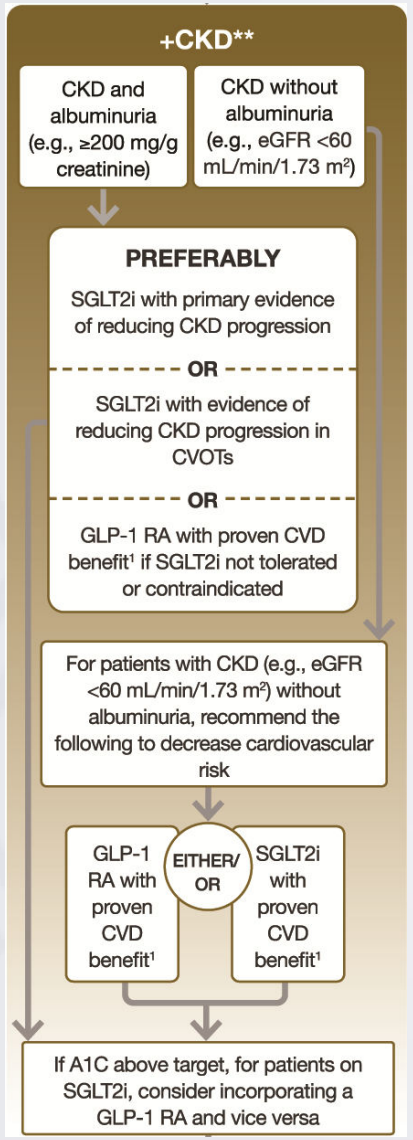
CKD is classified based on: • Cause (C) • GFR (G) • Albuminuria (A)				Albuminuria categories Description and range		
				A1	A2	A3
				Normal to mildly increased	Moderately increased	Severely increased
				<30 mg/g <3 mg/mmol	30-299 mg/g 3-29 mg/mmol	≥300 mg/g ≥30 mg/mmol
GFR categories (mL/min/1.73 m^2) Description and range	G1	Normal to high	≥90	1 if CKD	Treat 1	Refer* 2
	G2	Mildly decreased	60-89	1 if CKD	Treat 1	Refer* 2
	G3a	Mildly to moderately decreased	45-59	Treat 1	Treat 2	Refer 3
	G3b	Moderately to severely decreased	30-44	Treat 2	Treat 3	Refer 3
	G4	Severely decreased	15-29	Refer* 3	Refer* 3	Refer 4+
	G5	Kidney failure	<15	Refer 4+	Refer 4+	Refer 4+

Source:
Standards of Medical Care in Diabetes—2022, Diabetic Care, American Diabetes Association, December 2021 (45):S1-S264

Nephropathy: prevention



Source:
Standards of Medical Care in Diabetes—2022, Diabetic Care, American Diabetes Association, December 2021 (45):S1-S264

DM with CKD and no hypertension:

Use of a SGLT2i in patients with an eGFR ≥25 mL/min/1.73 m2 and UACR ≥300 mg/g is recommended to reduce chronic kidney disease progression and cardiovascular events.

If can not use SGLT2i, GLP1a or nonsteroidal mineralocorticoid receptor antagonist (finerenone) is recommended.

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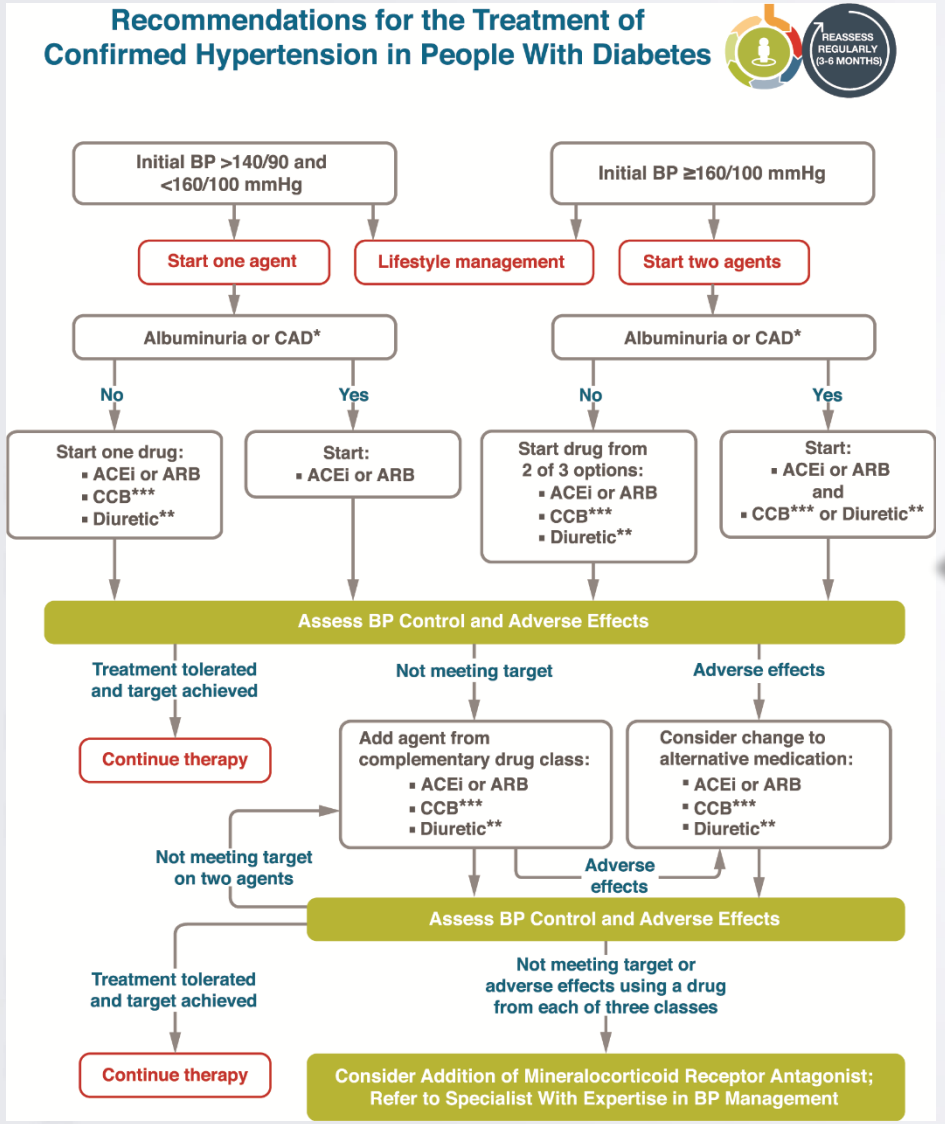
Nephropathy: prevention



Source:
Standards of Medical Care in Diabetes—2022, Diabetic Care, American Diabetes Association, December 2021 (45):S1-S264

DM with CKD and hypertension:
Use of a ACEI or ARB is recommended to reduce chronic kidney disease progression and cardiovascular events beside SGLT2i.

No role of ACEI or ARB in prophylaxis in normotensive DM patient with or without nephropathy.

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Nephropathy: prevention

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SGLT2i use in preventing or decreasing the disease progression in diabetic CKD

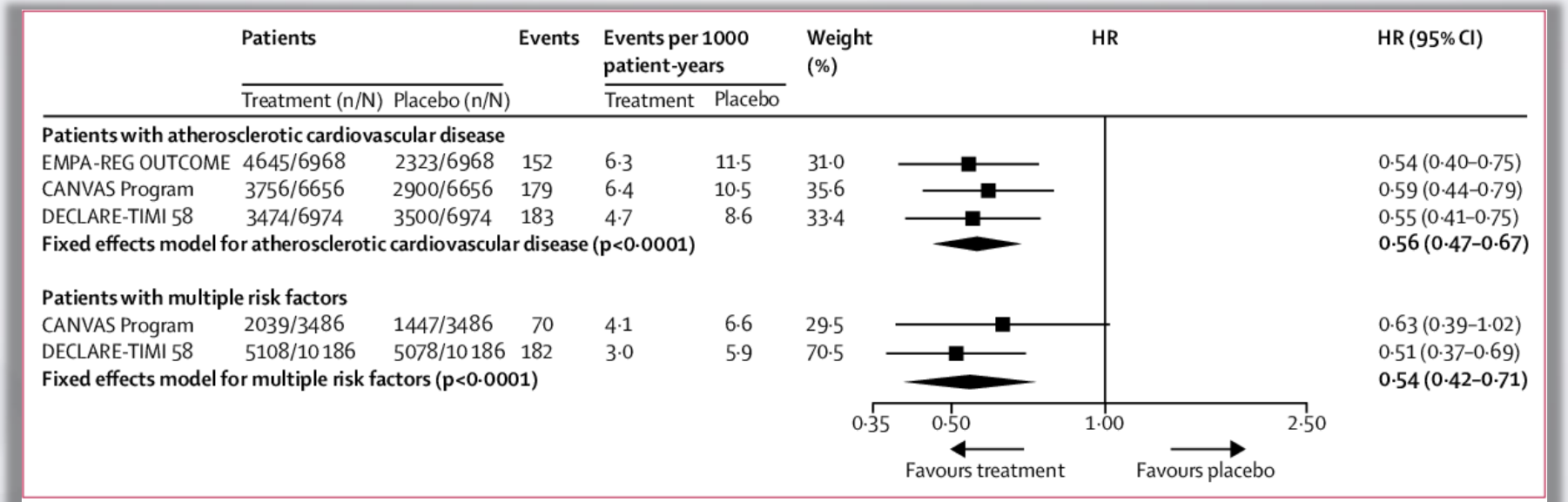

Figure 4: **Meta-analysis of SGLT2i trials on the composite of renal worsening, end-stage renal disease, or renal death stratified by the presence of established atherosclerotic cardiovascular disease**

Atherosclerotic cardiovascular disease: Q statistic=0·19, p=0·91, I^2=0%; multiple risk factors: Q statistic=0·52, p=0·47, I^2=0% The p value for subgroup differences was 0·71. Tests for subgroup differences were based on F tests in a random effect meta-regression estimated using restricted maximum likelihood and Hartung Knapp adjustment. HR=hazard ratio. SGLT2i=sodium-glucose cotransporter-2 inhibitors.

Zelniker TA et al. SGLT2 inhibitors for primary and secondary prevention of cardiovascular and renal outcomes in type 2 diabetes: a systematic review and meta-analysis of cardiovascular outcome trials. Lancet 2019; 393:31–39

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Nephropathy: prevention

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GLP-1a use in preventing or decreasing the disease progression in diabetic CKD

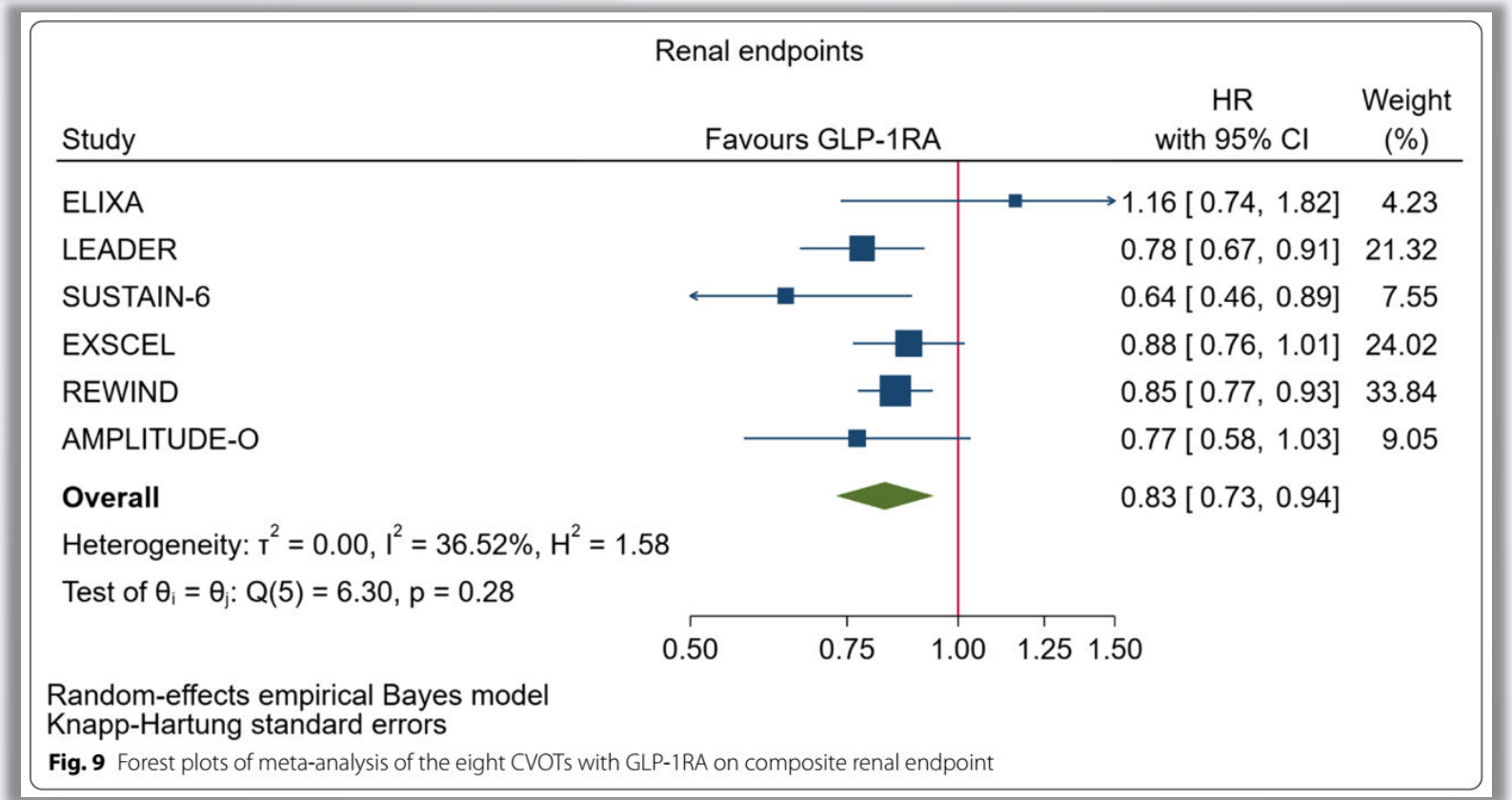

Fig. 9 Forest plots of meta-analysis of the eight CVOTs with GLP-1RA on composite renal endpoint

Giugliano etal. GLP-1 receptor agonists and cardiorenal outcomes in type 2 diabetes: an updated meta-analysis of eight CVOTs. Cardiovasc Diabetol. 2021; 20(189).

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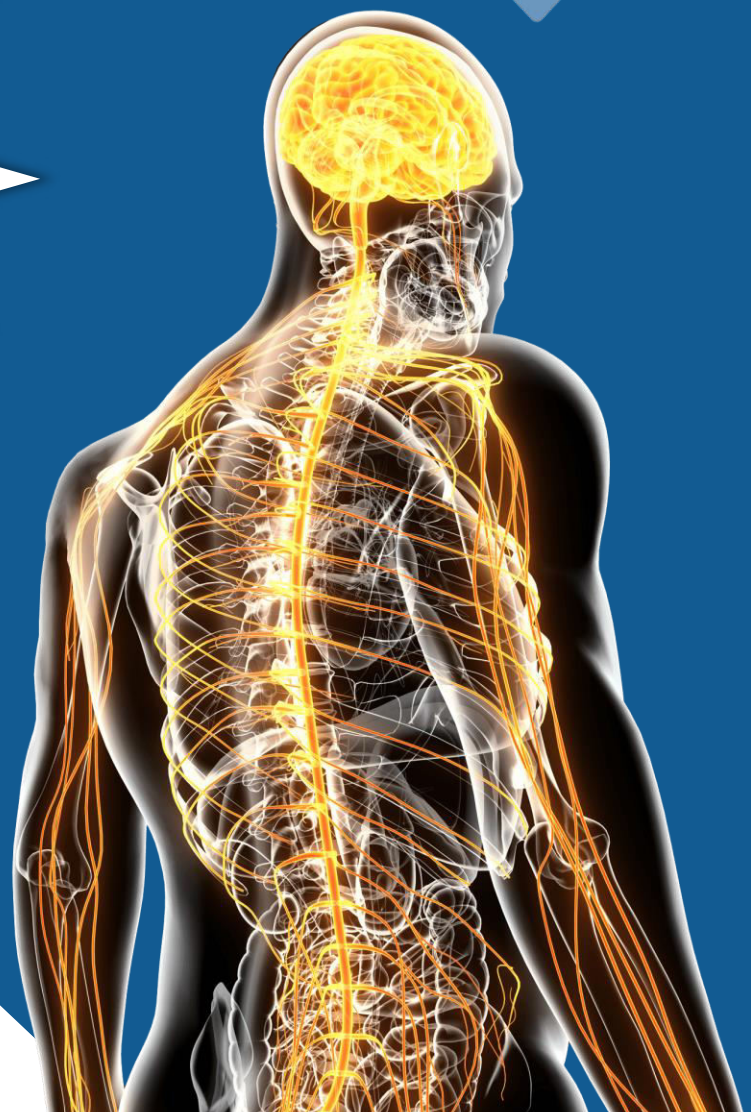
Long-term Complications

Microvascular Complications: NEUROPATHY



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Neuropathy: overview

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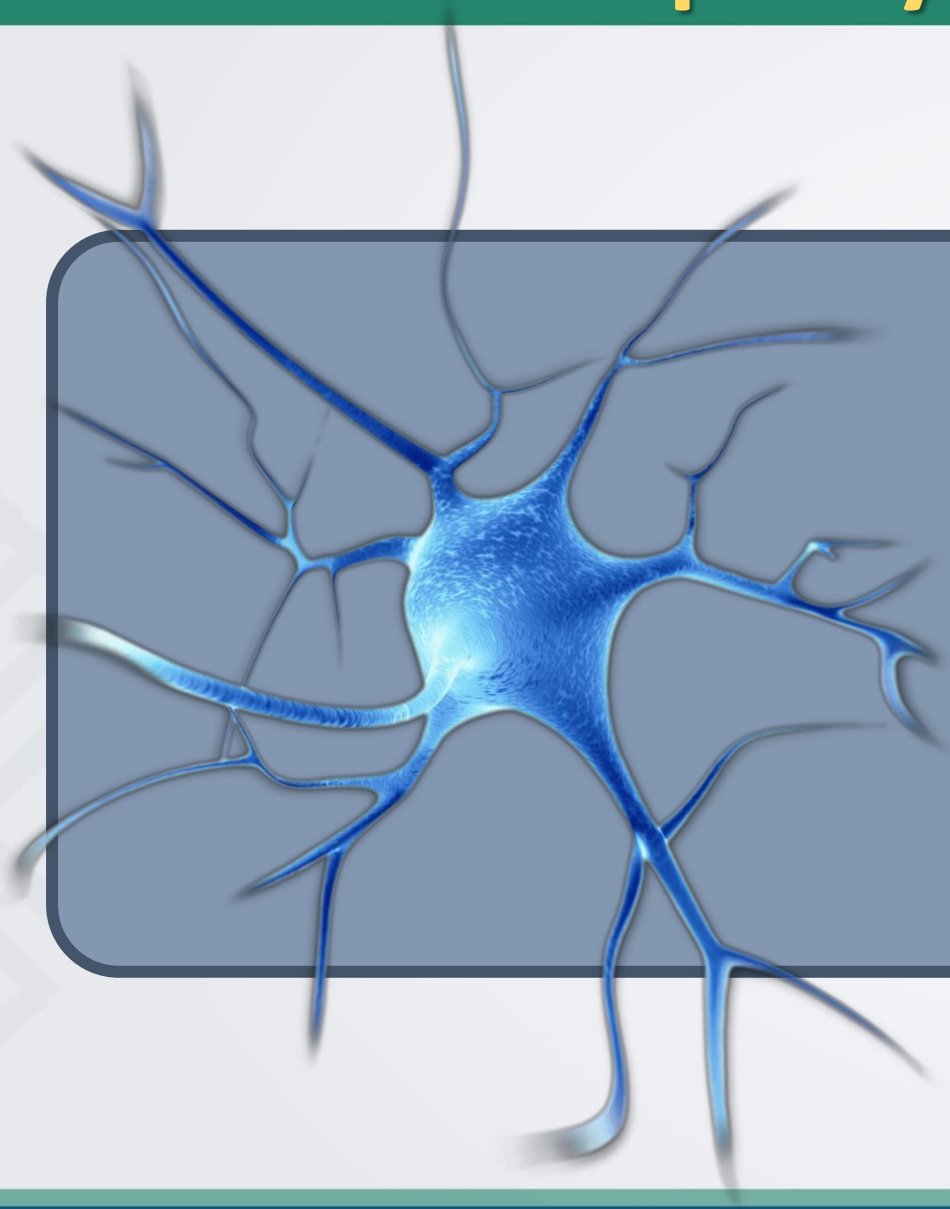

Diabetic neuropathy *is caused by nerve damage through different mechanisms, including direct damage by the hyperglycemia and decreased blood flow to nerves by damaging small blood vessels.*

This can lead to sensory motor, autonomic and spinal nerve damage.

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Neuropathy: Symptoms

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Neuropathy: DM foot

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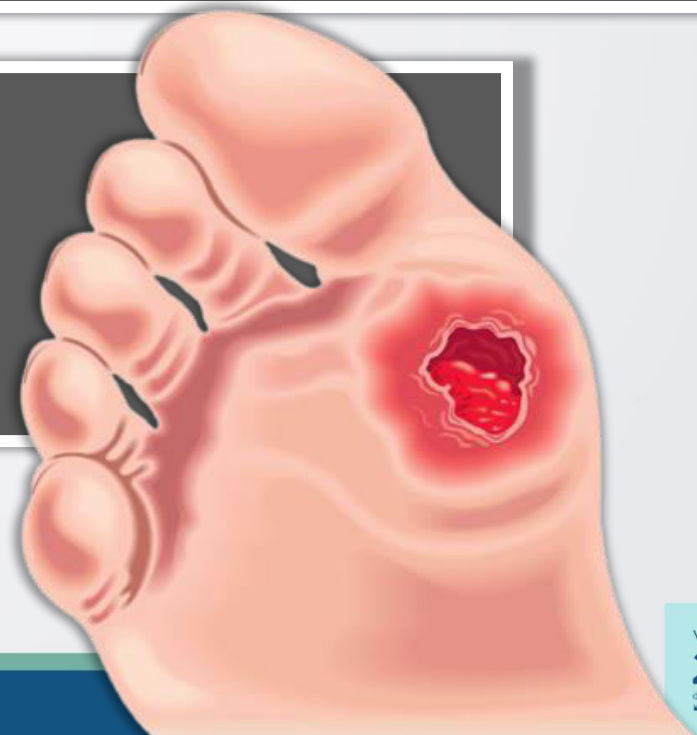
From 50% to 75% of lower extremity amputations *are performed on people with diabetes.*

More than 50% of these amputations are thought to be preventable, provided patients are taught foot care measures and practice them on a daily basis.

*It results from both **vascular and neurological disease processes** due to changes in blood vessels and nerves (the patient can not feel if they got cut on their feet), **often leads to ulceration and subsequent limb amputation**.*



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Neuropathy: DM foot screening

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Screening:

How: *check the following video.*

WHEN: *For type 2 DM → immediately. For type 1 DM → after 5 years of diagnosis.*

FREQUENCY: *Annually by physician or podologist; and daily by patient (see the patient instruction). Patient with evidence sensory loss or prior ulceration or amputation should have their feet inspected at every clinic visit.*

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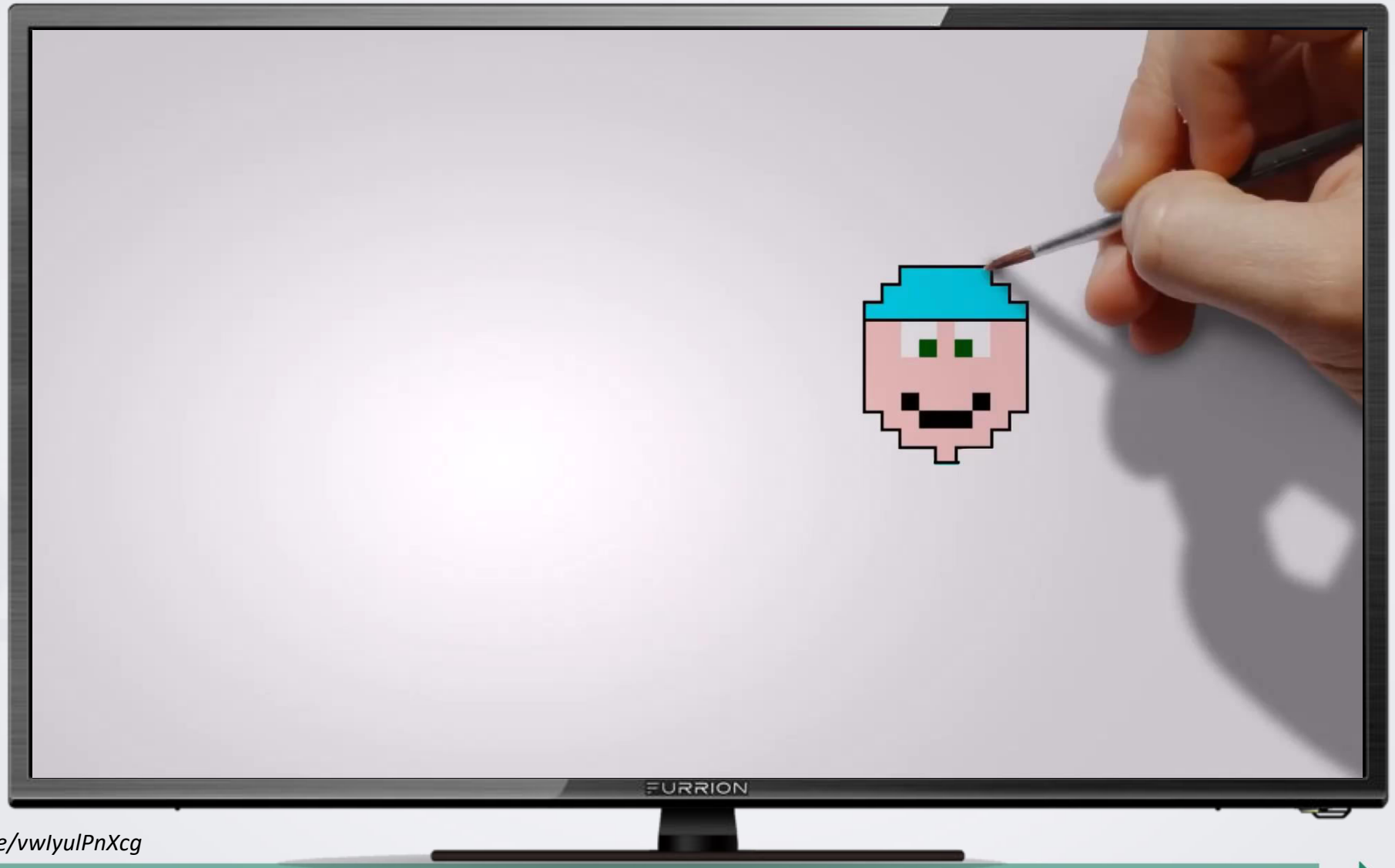
Neuropathy: DM foot screening - Physician



Source: https://youtu.be/vwIyulPnXcg

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Neuropathy: DM foot screening - Patient

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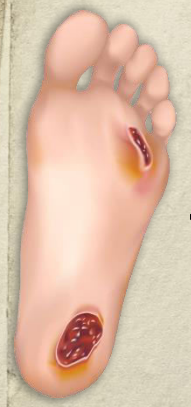

Instructions to patient

1. Inspect your feet every day from every side (use mirror to help you to check planter side), especially after going outside home.

2. Use soft shoes (no bare feet or slippers) when you go outside. Use slightly bigger shoes than you usually wear.

3. Always check bathtub for proper water temperature using your elbow before going inside the bathtub.

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Neuropathy: Treatment

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The following medications are recommended as initial pharmacologic treatments for neuropathic pain in diabetes:

1. Pregabalin
2. Duloxetine
3. Gabapentin

Referral criteria to foot care specialists : (any of the followings)

1. Smoker + age > 50 years.
2. History of prior lower-extremity complications.
3. Loss of protective sensation.
4. Structural abnormalities.
5. Peripheral arterial disease.

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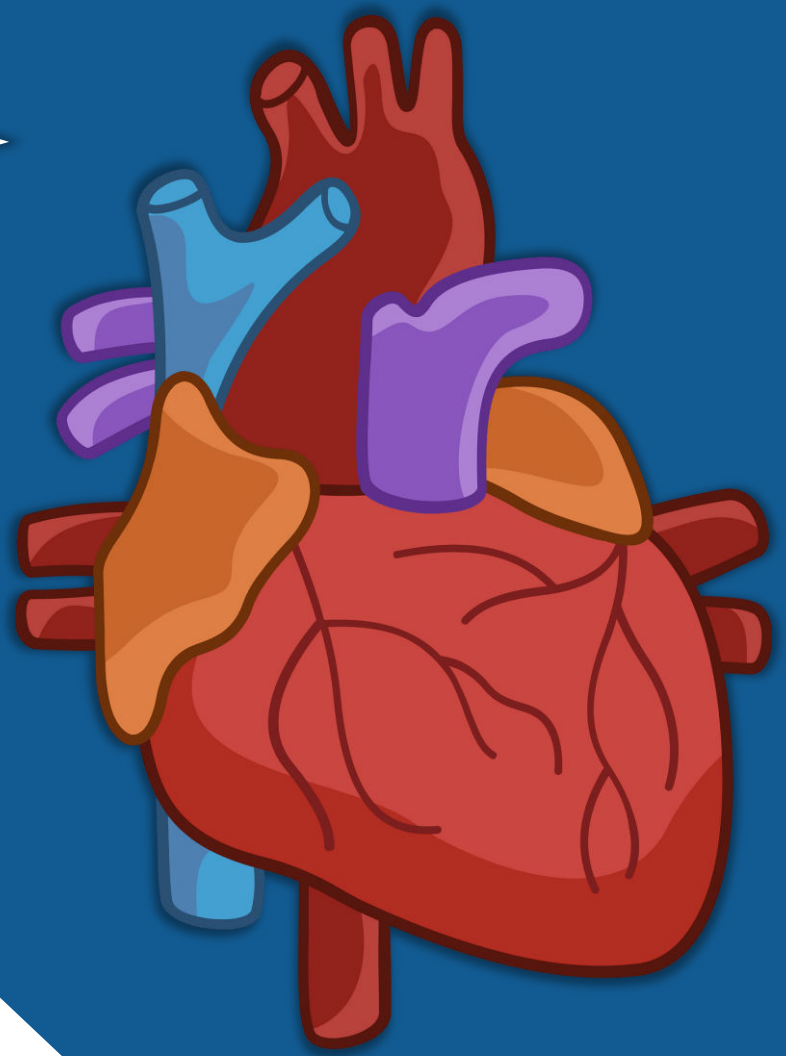
Long-term Complications

Macrovascular Complications:

Coronary Artery Disease (CAD)



CAD in DM: overview

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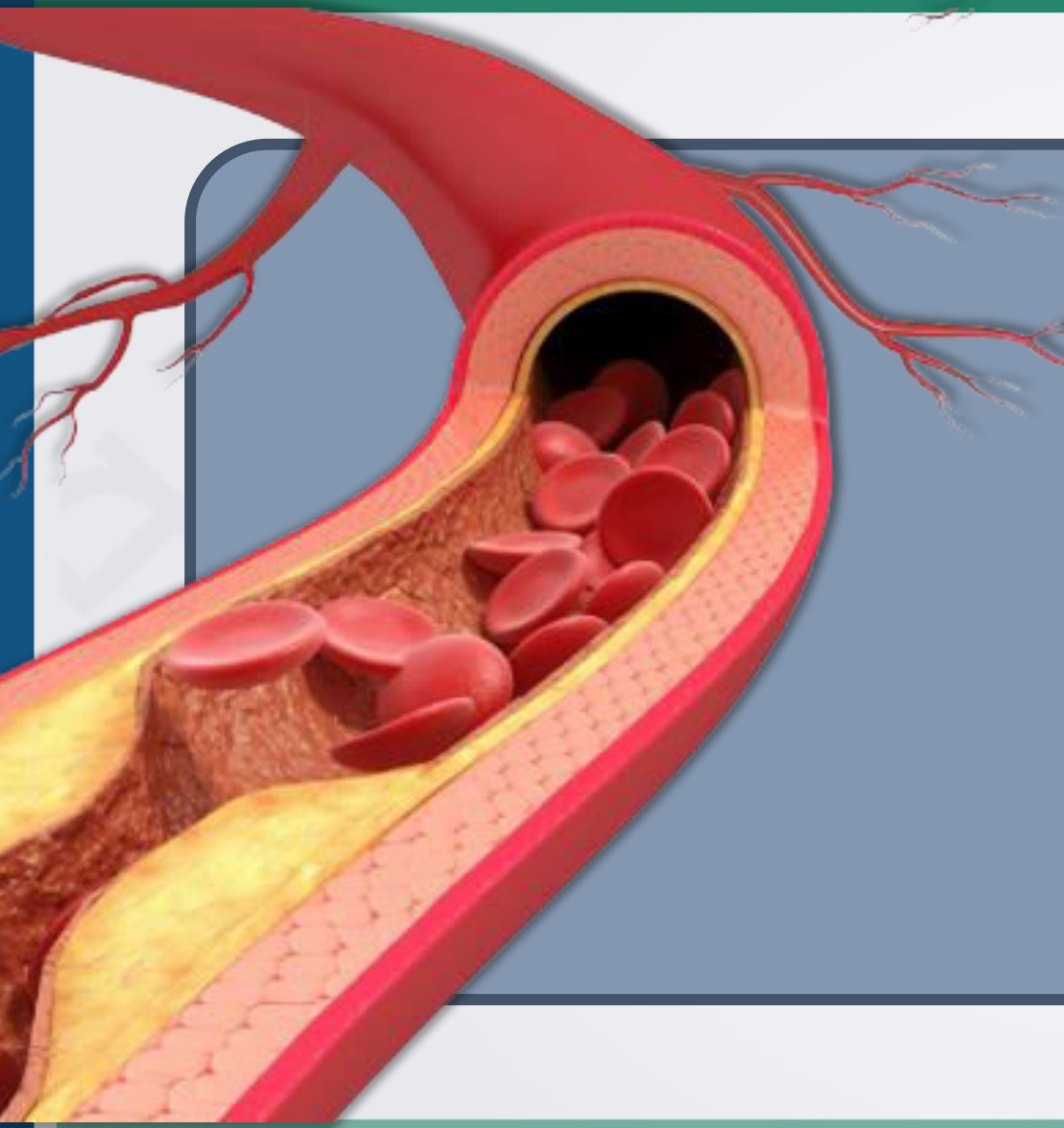

Diabetic macrovascular complications *result from changes in the medium to large blood vessels. Blood vessel walls thicken, sclerose, and become occluded by plaque that adheres to the vessel walls. Eventually, blood flow is blocked.*

These atherosclerotic changes tend to occur more often and at an earlier age in diabetes.

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CAD in DM: risk factors

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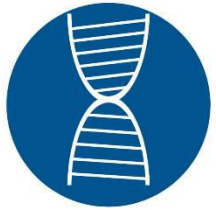
SMOKING


INFECTIONS


ALCOHOL


GENETIC PREDISPOSITION


UNHEALTHY FOOD


OBESITY

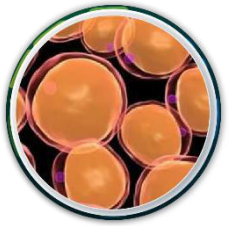

SEDENTARY LIFESTYLE

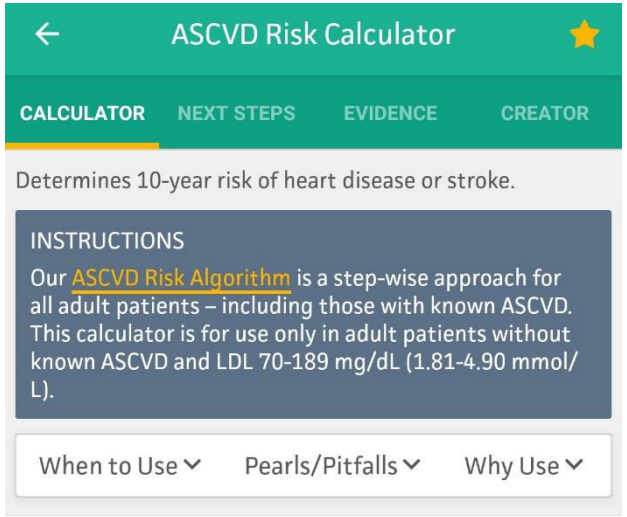

AGE


STRESS


DIABETES


HYPERTENSION


HYPERLIPIDEMIA



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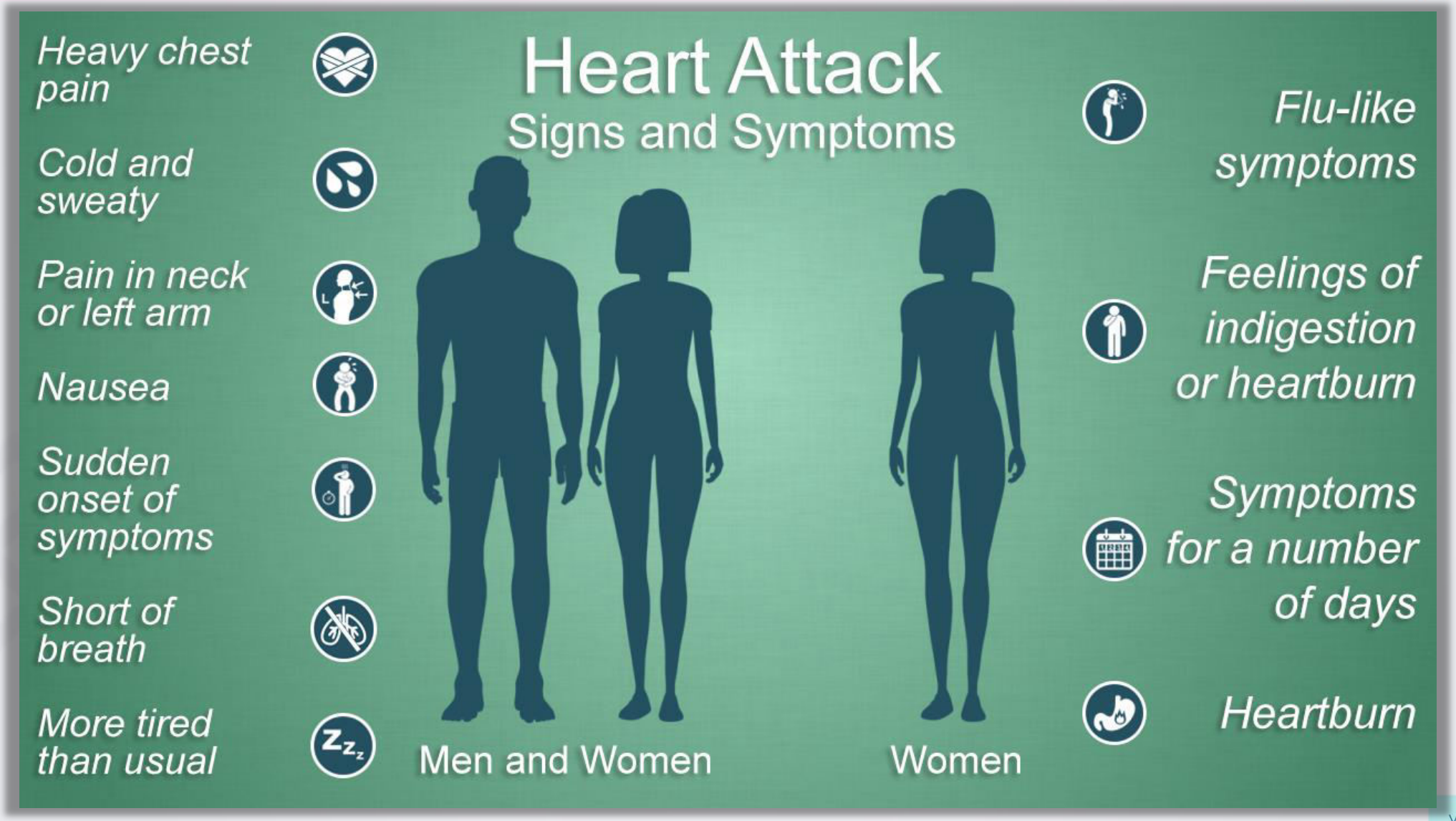
CAD in DM: symptoms



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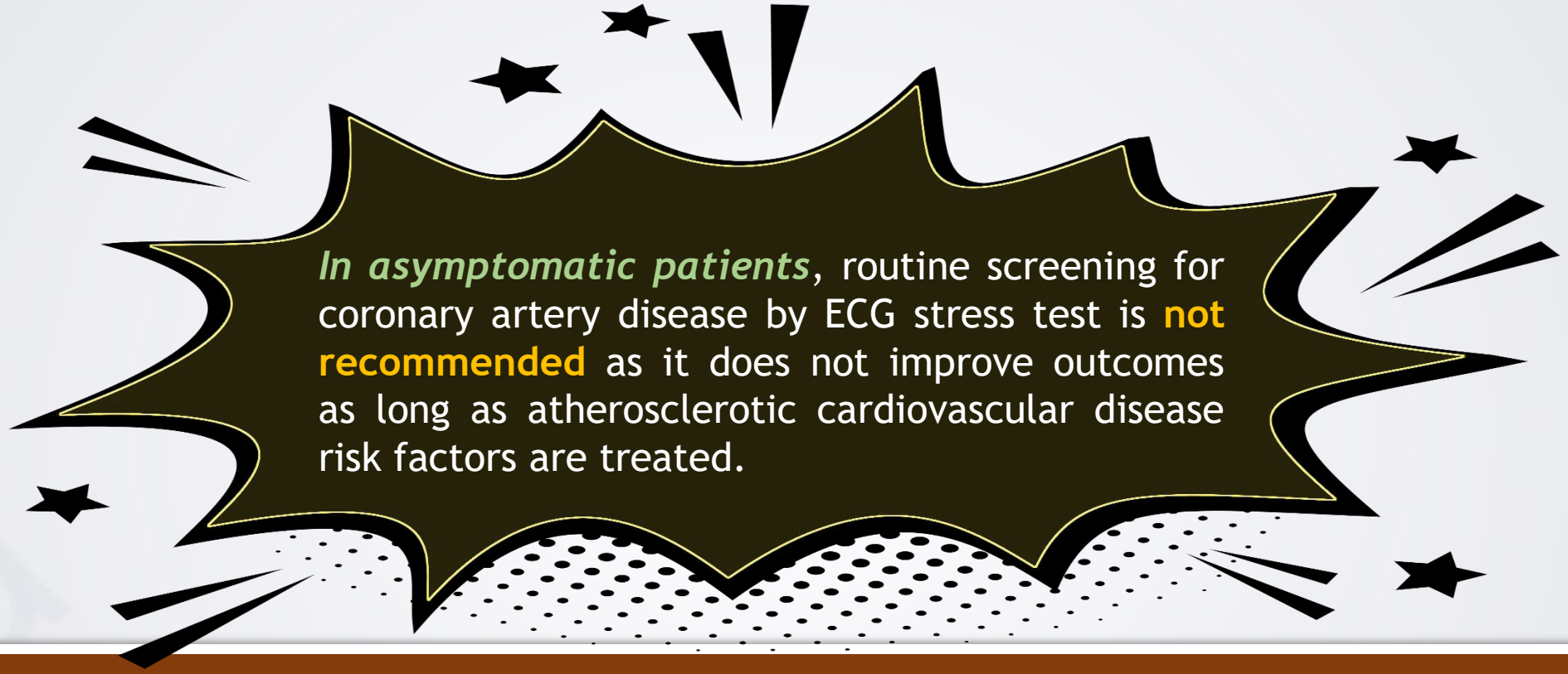
CAD in DM: ECG stress test screening



Consider ECG stress test in the presence of any of the following:

- Atypical cardiac symptoms (e.g., unexplained dyspnea, chest discomfort).
- Signs or symptoms of associated vascular disease (carotid bruits, transient ischemic attack, stroke, claudication, or peripheral arterial disease; or abnormal resting ECG).

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CAD in DM: reduction of risk

DM Rx

SGLT2i ± GLP1a

In any DM-2 patient with established atherosclerotic cardiac disease or with risk factors.

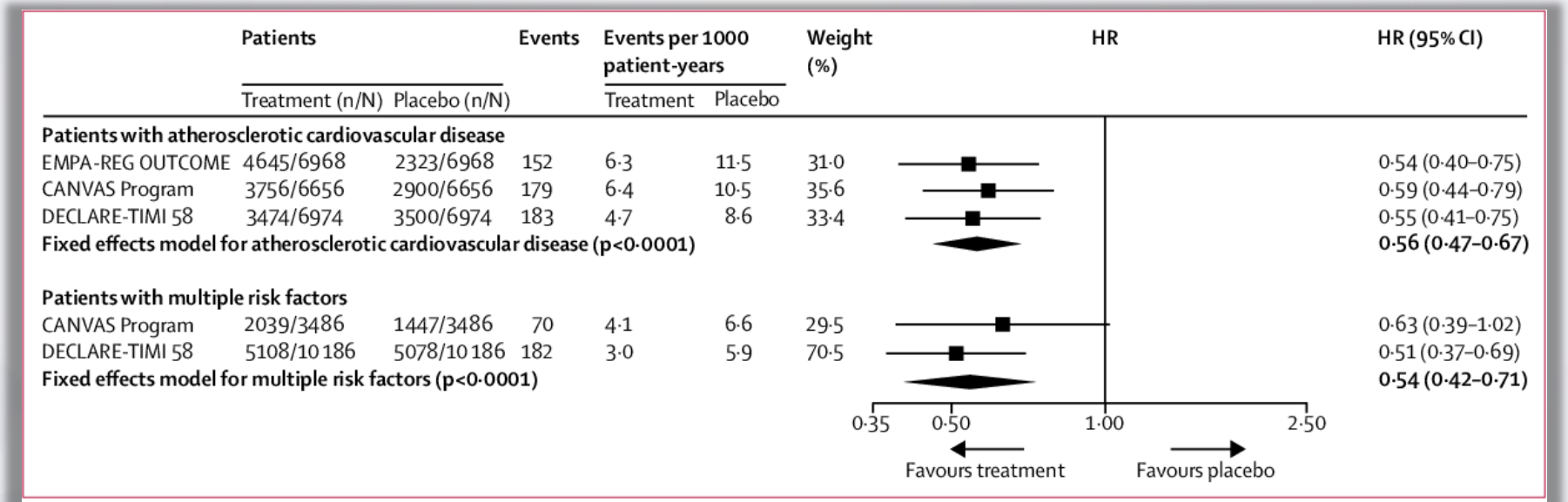
Non-DM Rx

ABAS (ACEI OR ARB, β-BLOCKER, ASPIRIN, STATIN)

In any DM-2 patient with established atherosclerotic cardiac disease.

CAD in DM: reduction of risk

SGLT2i use in preventing or decreasing major cardiovascular events



	Patients		Events	Events per 1000 patient-years		Weight (%)	HR	HR (95% CI)
	Treatment (n/N)	Placebo (n/N)		Treatment	Placebo			
Patients with atherosclerotic cardiovascular disease								
EMPA-REG OUTCOME	4645/6968	2323/6968	152	6·3	11·5	31·0		0·54 (0·40–0·75)
CANVAS Program	3756/6656	2900/6656	179	6·4	10·5	35·6		0·59 (0·44–0·79)
DECLARE-TIMI 58	3474/6974	3500/6974	183	4·7	8·6	33·4		0·55 (0·41–0·75)
Fixed effects model for atherosclerotic cardiovascular disease (p<0·0001)								**0·56 (0·47–0·67)**
Patients with multiple risk factors								
CANVAS Program	2039/3486	1447/3486	70	4·1	6·6	29·5		0·63 (0·39–1·02)
DECLARE-TIMI 58	5108/10 186	5078/10 186	182	3·0	5·9	70·5		0·51 (0·37–0·69)
Fixed effects model for multiple risk factors (p<0·0001)								**0·54 (0·42–0·71)**

Figure 4: **Meta-analysis of SGLT2i trials on the composite of renal worsening, end-stage renal disease, or renal death stratified by the presence of established atherosclerotic cardiovascular disease**

Atherosclerotic cardiovascular disease: Q statistic=0·19, p=0·91, I^2=0%; multiple risk factors: Q statistic=0·52, p=0·47, I^2=0% The p value for subgroup differences was 0·71. Tests for subgroup differences were based on F tests in a random effect meta-regression estimated using restricted maximum likelihood and Hartung Knapp adjustment. HR=hazard ratio. SGLT2i=sodium-glucose cotransporter-2 inhibitors.

Zelniker TA et al. SGLT2 inhibitors for primary and secondary prevention of cardiovascular and renal outcomes in type 2 diabetes: a systematic review and meta-analysis of cardiovascular outcome trials. Lancet 2019; 393:31–39

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CAD in DM: reduction of risk

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GLP-1a use in preventing or decreasing major cardiovascular events (MACE)

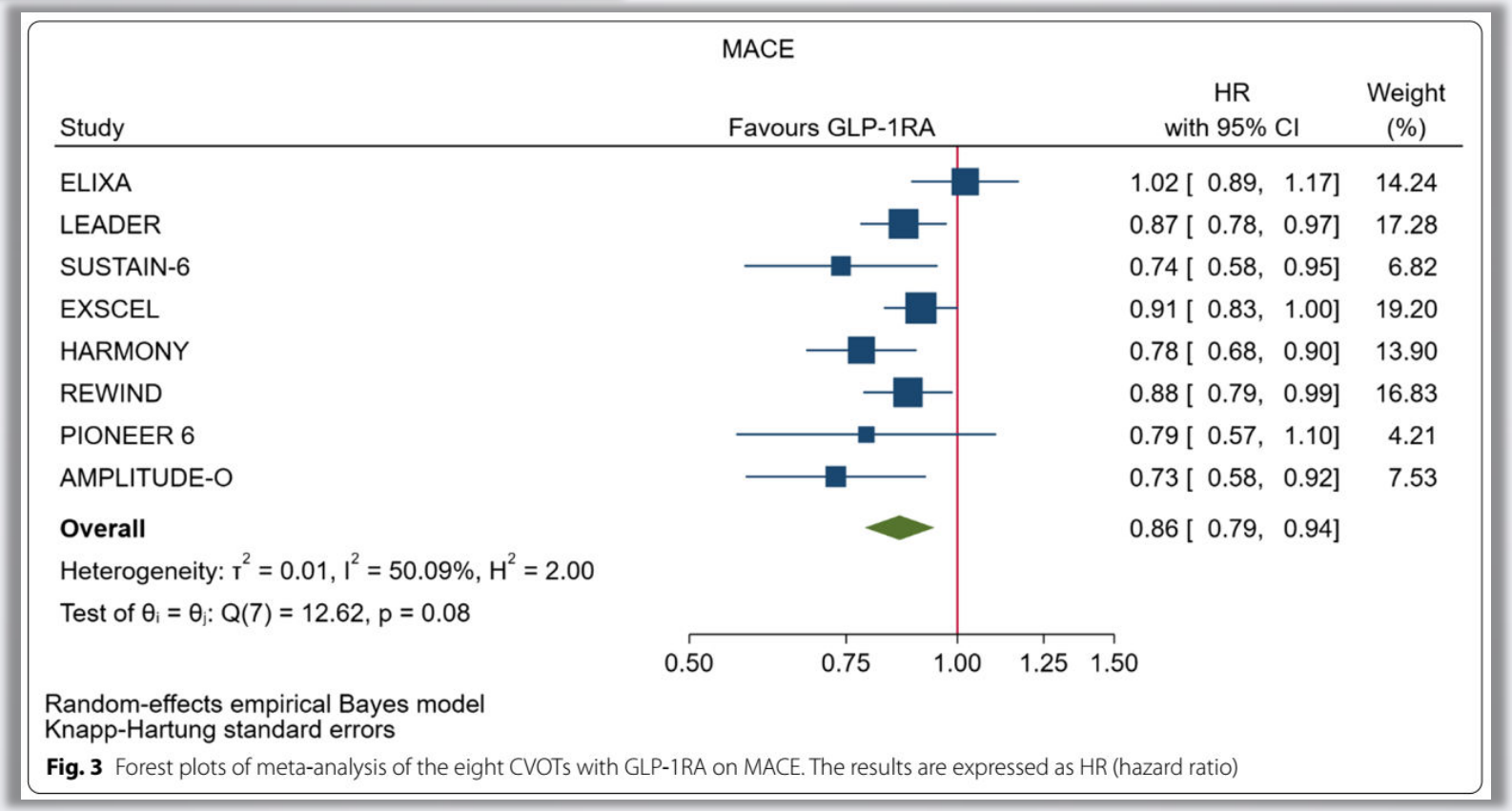

Fig. 3 Forest plots of meta-analysis of the eight CVOTs with GLP-1RA on MACE. The results are expressed as HR (hazard ratio)

Giugliano etal. GLP-1 receptor agonists and cardiorenal outcomes in type 2 diabetes: an updated meta-analysis of eight CVOTs. Cardiovasc Diabetol. 2021; 20(189).

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Long-term Complications

Macrovascular Complications:

Peripheral Artery Disease (PAD)

PAD: overview

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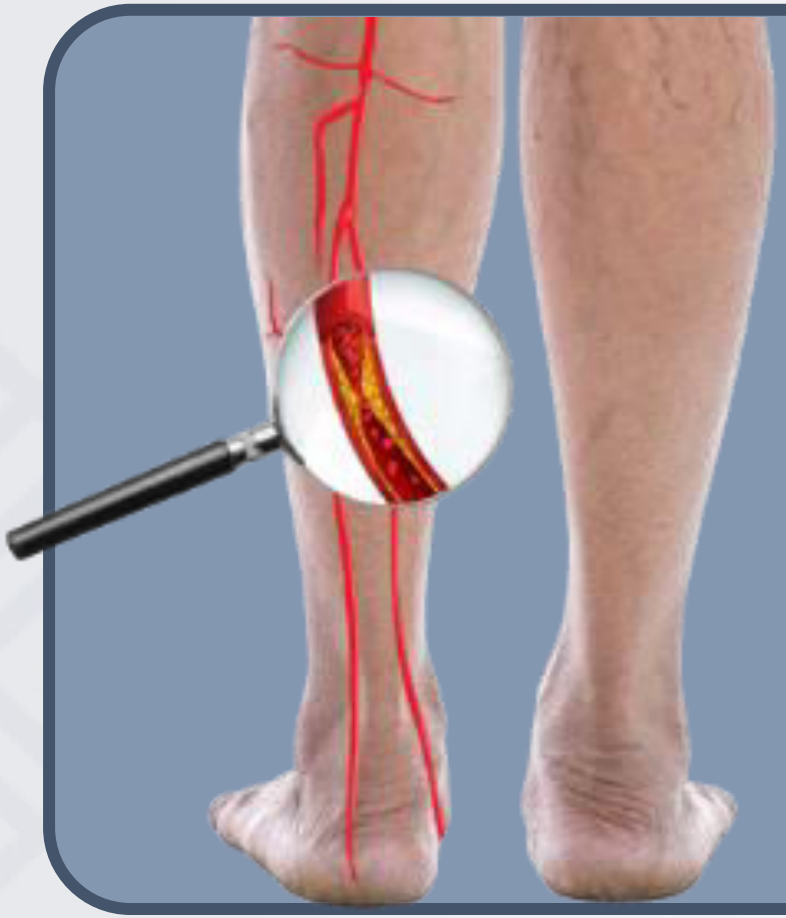

PVD *result from narrowing and hardening of the arteries that supply legs and feet leading to nerve and tissue damage to the extremities.*

Risk factors:

Smoking, DM, CAD, Hyperlipedemia, CKD, CVA, A-fibrilation

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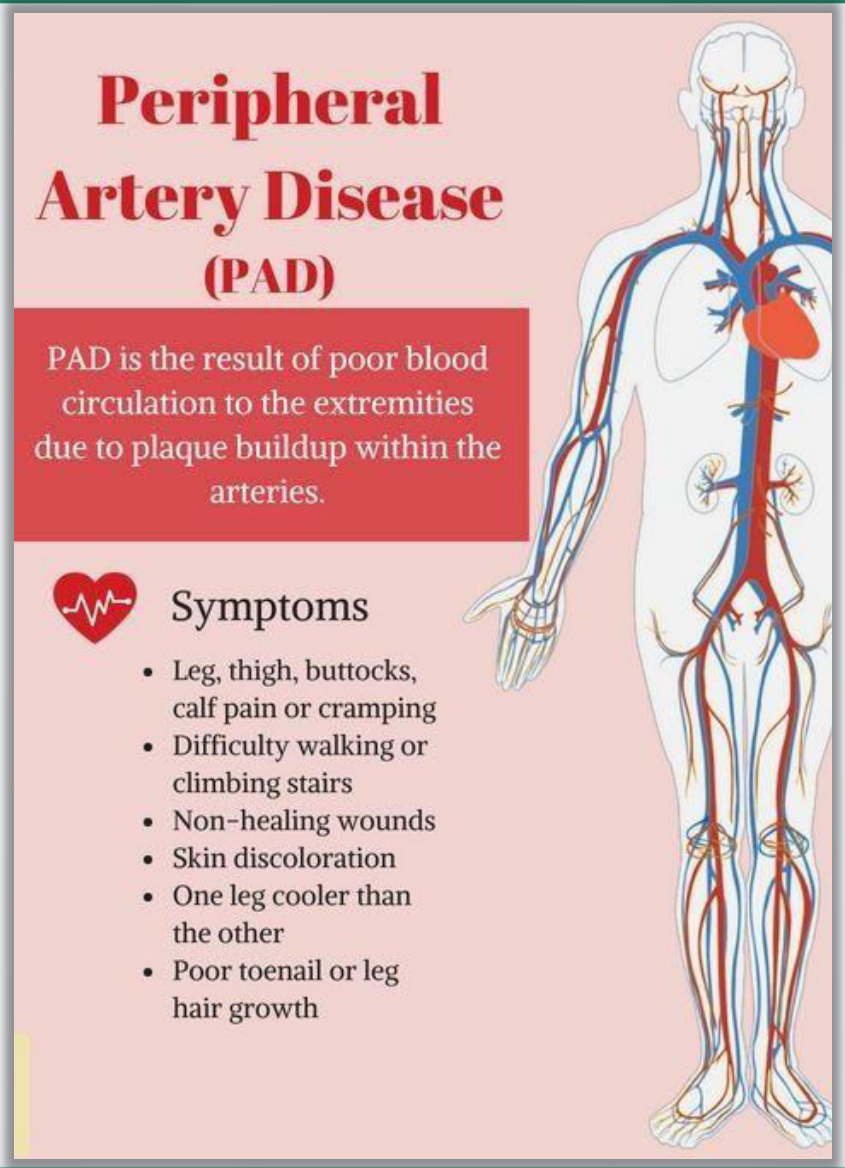
PAD: symptoms



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PAD: screening (Ankle-Brachial Pressure Index)

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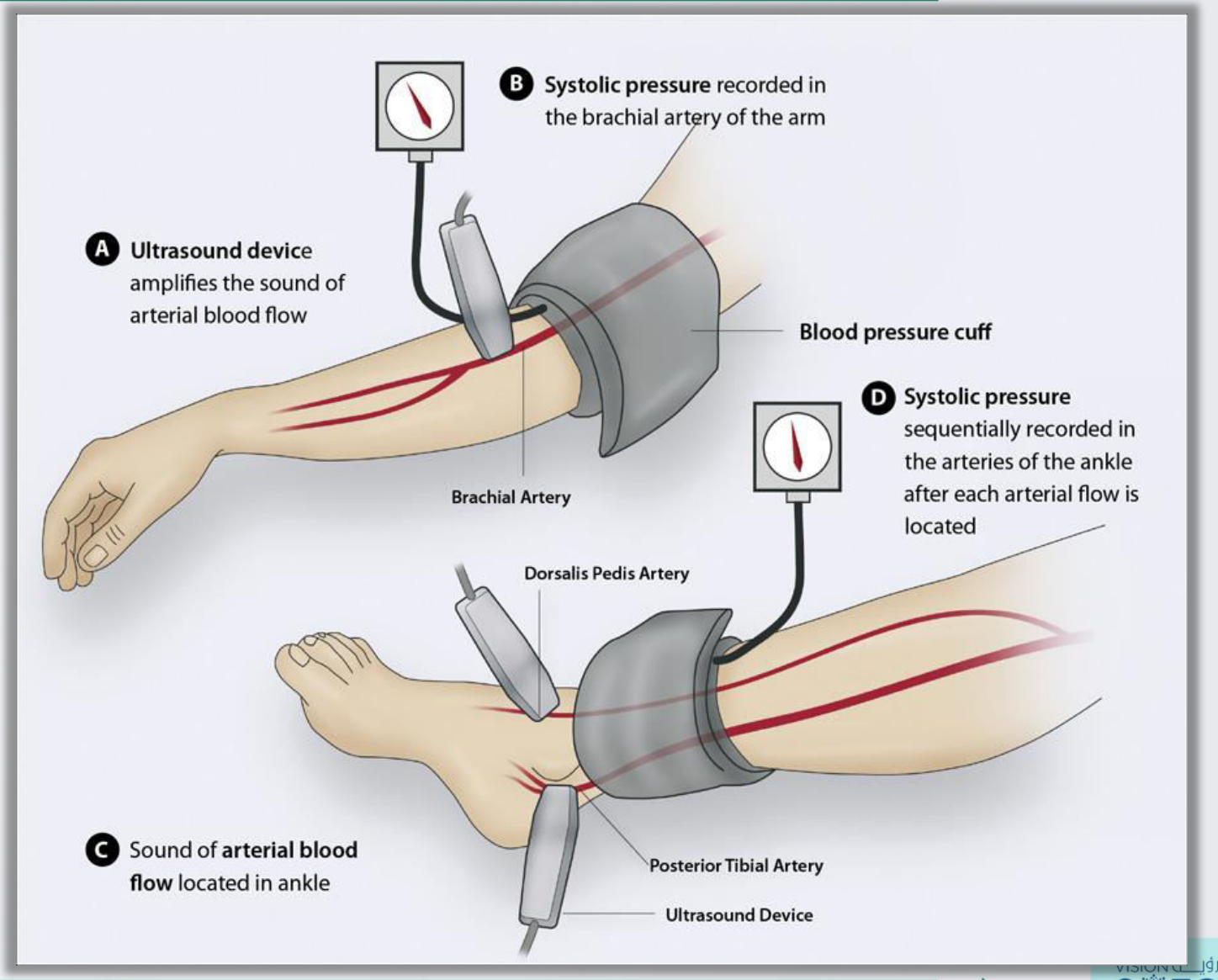
In asymptomatic patients, routine screening for PAD by Ankle-Brachial Index (ABI) is **not recommended**.



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PAD: screening (Ankle-Brachial Pressure Index)

Source: https://youtu.be/KnJDrmfIXGw

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PAD: screening (Ankle-Brachial Pressure Index)

Ankle-Brachial Index (ABI)

ABI value	Interpretation	Recommendation
> 1.4	Calcification	Referral to vascular specialist
1.0 - 1.4	Normal	None
0.9 - 1.0	Acceptable	None
0.8 - 0.9	Some arterial disease	Treat risk factors
< 0.8	Arterial disease	Referral to vascular specialist

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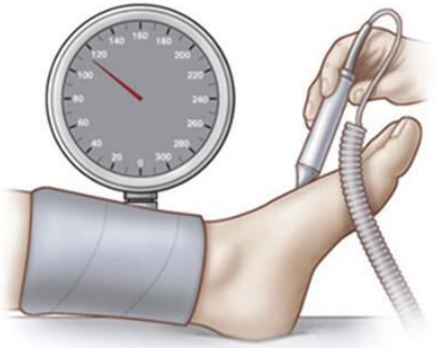
PAD: screening (Ankle-Brachial Pressure Index)

Critical Analysis and Limitations of Ankle-Brachial Index (ABI) In Diagnosis of Peripheral Arterial Disease (PAD)

Retrospective review

2226 ABIs and 1383 duplex ultrasound (DUS) examinations

Resting ABI to detect ≥ 50% stenosis on DUS



Patients with PAD	Sensitivity	Overall Accuracy
All	57%	74%
Diabetics	51%	66%
Non-Diabetics	66%	81%
With CKD*	43%	67%
No CKD*	60%	76%

*CKD = Chronic Kidney Disease

43% of symptomatic patients with PAD with ≥50% stenosis on DUS had normal/ inconclusive resting ABIs

(49% in diabetic, 57% in CKD patients)

JVS Journal of Vascular Surgery
Official Publication of the Society for Vascular Surgery

AbuRahma et al. *J Vasc Surg, March 2020*

@JVascSurg
@TheJVascSurg

Duplex Ultrasound **is preferred with sensitivity of 90% and specificity greater than 95% in detecting 50% or greater stenosis**

AbdulRahma etal. Critical analysis and limitations of resting ankle-brachial index in the diagnosis of symptomatic peripheral arterial disease patients and the role of diabetes mellitus and chronic kidney disease. Journal of Vascular Surgery. March 2020; 71(3):937-45

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وَقُل رَّبِّ زِدْنِي عِلْمًا

قَالُوا۟ سُبْحَٰنَكَ
لَا عِلْمَ لَنَآ إِلَّا مَا عَلَّمْتَنَآ ۖ
إِنَّكَ أَنتَ ٱلْعَلِيمُ ٱلْحَكِيمُ


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Any Questions
Thank You
Dr. Majdi Allasim