
label
البرنامج التدريبي لإعداد الصيادلة

DIABETES MELLITUS

لماذا نعالجه عند غير الحوامل؟

1. Primary goal: Prevent the onset of acute or chronic complications
2. Acute complications: Hypoglycemia, diabetic ketoacidosis (DKA), hyperglycemic hyperosmolar nonketotic syndrome
3. Chronic complications
    a. Microvascular: Retinopathy, nephropathy, and neuropathy
    b. Macrovascular: Cardiovascular, cerebrovascular, and peripheral vascular diseases
4. Glycemic therapy goals
    a. A1C less than 7.0% (Note: The American College of Endocrinology/AACE guidelines recommend 6.5% or less.)
        i. Obtain every 6 months in patients at goal A1C and every 3 months in those over goal.
        ii. Less-stringent A1C targets may be appropriate in those with a short life expectancy (e.g., terminal cancer), advanced diabetic complications, longstanding diabetes that is difficult to control (e.g., frail older adults with a history of hypoglycemia at risk of falls), or extensive other comorbidities (clinical atherosclerotic cardiovascular disease). (In such situations, a higher A1C [e.g., less than 8%] may be sufficient to limit the risk of acute complications of hyperglycemia such as dehydration and electrolyte deficiencies while preventing exacerbation of comorbidities.)
    b. FPG or premeal 80–130 mg/dL. Frequency of monitoring depends on regimen, type of DM, and current glycemic control.
    c. Peak postprandial glucose (1–2 hours after a meal) less than 180 mg/dL

5. Non-glycemic therapy goals

a. Blood pressure

i. The ADA recommends a blood pressure goal of less than 130/80 mm Hg in patients at high risk of cardiovascular disease (existing cardiovascular disease or greater than a 15% ten-year risk) and a goal of less than 140/90 mm Hg in those at lower risk.

ii. ACC/AHA hypertension guidelines recommend less than 130/80 mm Hg.

b. Lipids

i. ADA: No specific LDL goal is currently recommended.

ii. ACC/AHA 2018 guidelines suggest lowering LDL by 30%–49% in patients with diabetes age 40–75 and by at least 50% if at higher risk, e.g. age 50-75 or patient has other risk factors.

iii. No specific TG or HDL goals are currently recommended.

لماذا نعالجه عند الحوامل؟

1. Primary goal: Prevent complications to mother and child.
2. Glycemic therapy goals (more stringent)
    a. FPG of 95 mg/dL or less
    b. 1-hour postprandial glucose 140 mg/dL or less
    c. 2-hour postprandial glucose 120 mg/dL or less
3. Potential complications of hyperglycemia during pregnancy
    a. Mother: Hypertension, preeclampsia, T2D after pregnancy
    b. Fetus/child: Macrosomia, hypoglycemia, jaundice, respiratory distress syndrome

لماذا نعالجه؟

1. Glycemic control
    a. Reduces the risk of developing retinopathy, nephropathy, and neuropathy in T1D and T2D
    b. Prospective studies, specifically designed to assess optimizing glycemic control and effect on cardiovascular events, have shown little or no reduction in cardiovascular outcomes.
    c. However, the “legacy” effect in the Diabetes Control and Complications Trial of T1D and the UK Prospective Diabetes Study of T2D suggests early control has future cardiovascular benefit.
    d. No profound benefit of aggressive glycemic control in T2D (A1C less than 6.5%)
2. Blood pressure control: Reduction in both macrovascular and microvascular complications
3. Lipid control: Reduction in LDL with moderate-intensity statin therapy reduces cardiovascular complications.

كيف نصنفه؟

1. T1D

a. Attributable to cellular-mediated β-cell destruction leading to insulin deficiency (insulin needed for survival)

b. Accounts for 5%–10% of DM

c. Formerly known as insulin-dependent diabetes and juvenile-onset diabetes

d. Usually presents in childhood or early adulthood but can present in any stage of life

e. Usually symptomatic with a rapid onset in childhood, but a slower onset can occur in older adults

f. The ADA now recommends a staging of patients with T1D based on the degree of dysglycemia and symptoms.

i. Stage 1: Multiple autoantibodies present but glucose concentrations are normal

ii. Stage 2: Multiple autoantibodies present, glucose concentrations consistent with prediabetes, and patient is asymptomatic

iii. Stage 3: Symptomatic and glucose concentrations consistent with diabetes

2. T2D

a. Results primarily from insulin resistance in muscle and liver, with subsequent defect in pancreatic insulin secretion, though GI, brain, liver, and kidneys are all involved in the pathophysiology

b. Accounts for 90%–95% of diabetes mellitus

c. Formerly known as non–insulin-dependent diabetes or adult-onset diabetes

d. Often asymptomatic, with a slow onset over 5–10 years. Rationale for early, frequent screening of those at risk and initial assessment for complications at diagnosis

e. Disturbing increased trends in T2D in children and adolescents attributed to rise in obesity

3. Maturity-onset diabetes of the young
    a. Result of genetic disorder leading to impaired secretion of insulin with little or no impairment in insulin action
    b. Onset usually before age 25 and can mimic T1D or T2D
    c. Often misdiagnosed for type 1 diabetes. Patients may respond to sulfonylurea therapy.
4. Gestational diabetes
    a. Glucose intolerance occurring during pregnancy
    b. Prevalence: 1%–14% of pregnancies (complicates about 4% of pregnancies)
    c. Most common in third trimester
5. Prediabetes
    a. Impaired glucose tolerance
    b. Impaired fasting glucose
6. Other DM types
    a. Genetic defects in β-cell function or insulin action
    b. Diseases of the pancreas (e.g., pancreatitis, neoplasia, cystic fibrosis)
    c. Drug or chemical induced (e.g., glucocorticoids, nicotinic acid, protease inhibitors, atypical antipsychotics)

كيف نراقب حدوثه؟

1. T1D

    a. Symptomatic patients

    b. Asymptomatic patients at higher risk

        i. Relatives with T1D

        ii. Measure islet autoantibodies to assess risk of T1D.

        iii. If screen is positive for antibodies, counsel on symptoms of hyperglycemia and risk of DM. Consider enrollment in observational study.

2. T2D

a. Age 45 or older, repeat every 3 years if normal

b. Screen regardless of age if BMI is 25 kg/m2 or greater (23 kg/m2 or greater in Asian Americans) and at least one of the following risk factors:

i. History of cardiovascular disease

ii. A1C is 5.7% or greater, impaired glucose tolerance, or impaired fasting glucose in previous testing

iii. History of PCOS

iv. HDL less than 35 mg/dL or TG greater than 250 mg/dL

v. Hypertension

vi. Women with a diagnosis of gestational diabetes

vii. High-risk ethnicity: African American, Latino, Native American, Asian American, Pacific Islander

viii. First-degree relative with T2D

ix. Physical inactivity

x. Insulin resistance conditions (e.g., severe obesity, acanthosis nigricans)

3. Gestational DM

a. Screen at first prenatal visit for undiagnosed T2D in all patients with T2D risk factors present.

b. Screen at 24–28 weeks' gestation using OGTT.

c. If a diagnosis of gestational DM is made, screen for diabetes 4–12 weeks after delivery.

d. Continue to screen patients who have had gestational DM every 3 years for T2D for life.

كيف نشخصه؟

1. T1D and T2D diagnosis
    a. Glycemic values in nonpregnant patients
        i. FPG
            (a) Easy
            (b) 126 mg/dL or greater
        ii. Random plasma glucose
            (a) 200 mg/dL or greater with symptoms of hyperglycemia
            (b) Common hyperglycemia symptoms include polyuria, polydipsia, and unexplained weight loss.
        iii. OGTT
            (a) Plasma glucose concentration obtained 2 hours after a 75-g oral glucose ingestion
            (b) 200 mg/dL or greater
            (c) More sensitive and specific than FPG but more cumbersome to perform

iv. A1C (glycated hemoglobin)

(a) 6.5% or greater

(b) May be less sensitive than FPG in identifying mild diabetes, but does not require fasting and has less variability from day to day

(c) A1C values may be inaccurate in patients with anemia, chronic malaria, sickle cell anemia, pregnancy, or significant blood loss or recent blood transfusion.

v. The diagnosis of both T1D and T2D requires two abnormal tests showing hyperglycemia and can be obtained from the same sample (using two of the above criteria) or in two separate test samples (can be the same type of test).

b. Other useful diagnostic tests if type of DM is in question

i. C-peptide (measure of endogenous insulin secretion, usually negligible in T1D and normal or elevated early in T2D)

ii. Presence of islet cell autoantibodies, autoantibodies to insulin, glutamic acid decarboxylase,or tyrosine phosphatase (all suggest autoimmune activity)

2. Gestational diabetes diagnosis: Glycemic values in pregnancy
    a. Updated and simplified diagnostic criteria
    b. “One-step” approach: 75-g OGTT at 24–28 weeks’ gestation
        i. Fasting: 92 mg/dL or greater
        ii. 1 hour after OGTT: 180 mg/dL or greater
        iii. 2 hours after OGTT: 153 mg/dL or greater
    c. “Two-step” approach: 50-g OGTT (nonfasting) at 24–28 weeks’ gestation
        i. If 1 hour after 50-g OGTT is less than 140 mg/dL, no further workup
        ii. If 140 mg/dL or greater, do additional fasting OGTT using 100 g (see the ADA guidelines for diagnostic glucose criteria)
3. Prediabetes diagnosis (high-risk population)
    a. Impaired fasting glucose: FPG 100–125 mg/dL
    b. Impaired glucose tolerance: 2-hour plasma glucose after OGTT (75 g) of 140–199 mg/dL
    c. A1C 5.7%–6.4%