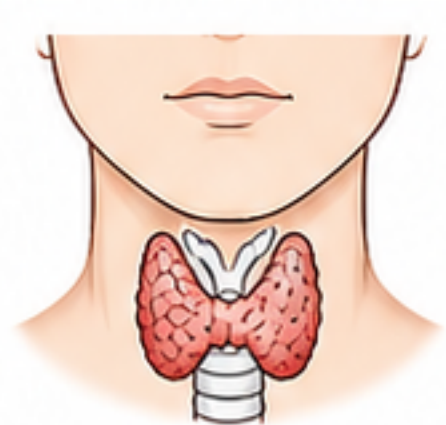




HYPOTHYROIDISM

High-Yield Exam Summary



1 DEFINITION

- Low thyroid hormone state
- Decreased T3/T4
- Hypometabolic state



3 KEY SYMPTOMS & SIGNS



- Fatigue



- Lethargy



- Cold intolerance



- Weight gain



- Constipation



- Dry skin



- Hair loss



- Slowed speech



- Slowed reflexes



- Bradycardia

2 COMMON CAUSES

- Hashimoto thyroiditis (most common)
- Thyroid surgery
- Radioactive iodine
- Iodine deficiency
- Thyroiditis
- Pituitary/hypothalamic causes for central hypothyroidism



4 LABS / DIAGNOSIS

- Primary hypothyroidism = high TSH, low free T4
- Secondary (central) hypothyroidism = low TSH, low free T4
- Anti-TPO often positive in Hashimoto



5 PATHOPHYSIOLOGY PEARLS

- Low thyroid hormone slows metabolism
- Decreased heat production explains cold intolerance
- Slower GI motility causes constipation



6 COMPLICATIONS / IMPORTANT POINTS

- Myxedema coma is severe form
- More common in women
- Most common primary cause is Hashimoto disease



7 MANAGEMENT BASICS

- Levothyroxine replacement
- Monitor TSH



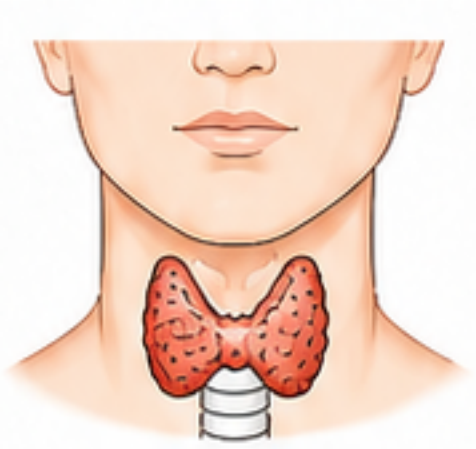
8 QUICK COMPARE

HYPOTHYROIDISM vs HYPERTHYROIDISM

HYPOTHYROIDISM	HYPERTHYROIDISM
Weight gain	
Cold intolerance	
Constipation	
Dry skin	

9 EXAM PEARLS

- ★ Hashimoto = most common cause
- ★ Primary: ↑ TSH, ↓ FT4
- ★ Anti-TPO positive
- ★ Cold intolerance + weight gain



HYPERTHYROIDISM

High-Yield Exam Summary



1 DEFINITION

- Excess thyroid hormone
- Increased T3/T4
- Hypermetabolic state



2 COMMON CAUSES

- Graves disease (most common)
- Toxic multinodular goiter
- Toxic adenoma
- Thyroiditis
- Iodine / amiodarone-induced
- Rare TSH-secreting pituitary tumor



3 KEY SYMPTOMS & SIGNS



- Weight loss



- Heat intolerance



- Sweating



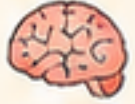
- Palpitations



- Tachycardia



- Tremor



- Anxiety



- Diarrhea



- Hyperreflexia

4 LABS / DIAGNOSIS

- Primary hyperthyroidism = low TSH, high free T4/T3
- TSH-secreting tumor = high TSH and high T4
- Thyroid-stimulating immunoglobulins (TSI) in Graves disease



5 GRAVES DISEASE PEARLS

- Autoimmune TSH receptor stimulation
- Diffuse goiter
- Ophthalmopathy
- Pretibial myxedema



6 COMPLICATIONS / IMPORTANT POINTS



- Atrial fibrillation



- Thyroid storm



- Osteoporosis risk



7 MANAGEMENT BASICS



- Beta-blocker for symptom control



- Antithyroid drugs (methimazole / propylthiouracil)



- Radioactive iodine



- Surgery in selected cases

8 QUICK COMPARE

HYPERTHYROIDISM TRAITS



Weight loss



Heat intolerance



Diarrhea



Tremor

9 EXAM PEARLS



Graves = most common cause



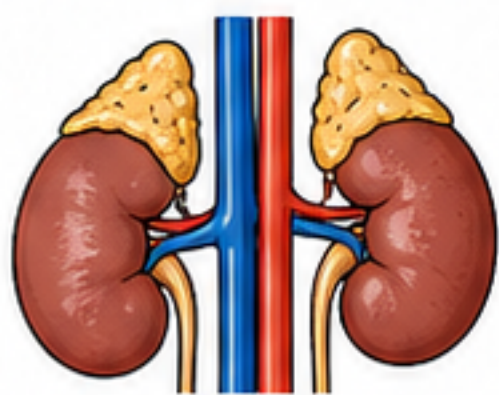
Primary:
↓ TSH, ↑ FT4



Eye signs suggest Graves



TSH tumor:
↑ TSH + ↑ T4



ADDISON DISEASE

Primary Adrenal Insufficiency – High-Yield Exam Summary



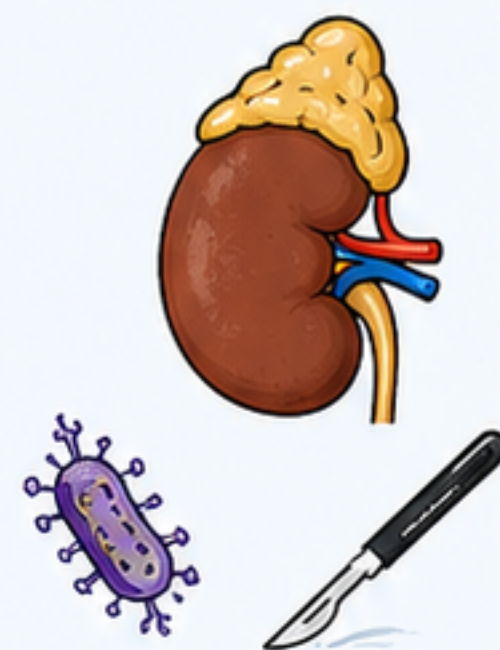
1 DEFINITION

- Primary adrenal cortical insufficiency
- Low cortisol
- Low aldosterone



2 CAUSES

- Autoimmune destruction (most common)
- TB / infection
- Adrenal hemorrhage
- Metastatic destruction
- Adrenalectomy



3 KEY SYMPTOMS & SIGNS



- Fatigue



- Weakness



- Weight loss



- Anorexia



- Hypotension



- Dizziness



- Hyperpigmentation



- Salt craving

- Dehydration

4 LABS / DIAGNOSIS

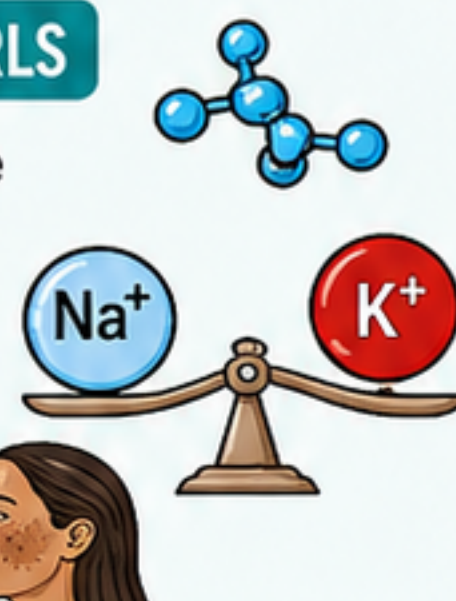
- Low cortisol
- High ACTH
- Hyponatremia ($\downarrow \text{Na}^+$)
- Hyperkalemia ($\uparrow \text{K}^+$)
- Poor response to ACTH stimulation test



CORTISOL	$\downarrow$
ACTH	$\uparrow$
Na^+	$\downarrow$
K^+	$\uparrow$

5 PATHOPHYSIOLOGY PEARLS

- Low cortisol $\rightarrow$ weakness, fatigue
- Low aldosterone $\rightarrow \text{Na}^+$ loss, K^+ retention $\rightarrow$ dehydration, hypotension
- High ACTH (and MSH) $\rightarrow$ hyperpigmentation



6 COMPLICATIONS / IMPORTANT POINTS

- Adrenal crisis can cause shock
- Primary disease = hyperpigmentation present; secondary disease usually does not

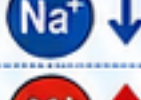
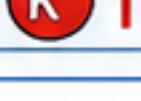


7 MANAGEMENT BASICS

- Glucocorticoid replacement (e.g., hydrocortisone)
- Mineralocorticoid replacement (e.g., fludrocortisone)
- Stress-dose steroids in illness or surgery



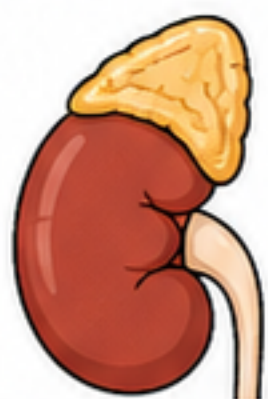
8 QUICK COMPARE 4 ADDISON CLUES

	Hypotension	Low aldosterone $\rightarrow$ volume depletion
	Hyperpigmentation	High ACTH / MSH
	Hyponatremia	Na^+ loss
	Hyperkalemia	K^+ retention

9 EXAM PEARLS

- ★ Autoimmune = most common cause
- ★ $\downarrow$ Cortisol, $\uparrow$ ACTH
- ★ $\text{Na}^+ \downarrow$, $\text{K}^+ \uparrow$
- ★ Think adrenal crisis in severe illness





CUSHING SYNDROME

— Cortisol Excess – High-Yield Exam Summary —



1 DEFINITION

- Syndrome of excess glucocorticoids (cortisol).



2 CAUSES

- Exogenous glucocorticoid therapy (most common)
- ACTH-secreting pituitary adenoma (Cushing disease)
- Adrenal tumor
- Ectopic ACTH production



3 KEY SYMPTOMS & SIGNS



- Central obesity



- Moon face



- Buffalo hump



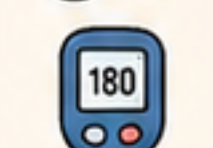
- Purple striae



- Muscle weakness



- Hypertension



- Hyperglycemia



- Easy bruising



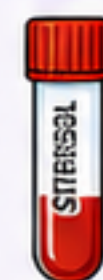
- Poor wound healing



- Hirsutism

4 LABS / DIAGNOSIS

- Elevated cortisol
- Screening options:
 - Low-dose dexamethasone suppression test
 - Late-night salivary cortisol
 - 24-hour urinary free cortisol
- ACTH level helps localize source



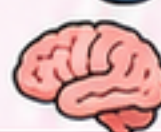
5 PATHOPHYSIOLOGY PEARLS

- Cortisol increases glucose ($\uparrow$ gluconeogenesis)
- Causes protein breakdown ($\uparrow$ catabolism)
- Mineralocorticoid-like effect: Na retention and K loss
- Suppresses immune function



6 COMPLICATIONS / IMPORTANT POINTS

- Increased risk of infections
- Osteoporosis
- Diabetes mellitus
- Mood changes (depression, anxiety)



7 MANAGEMENT BASICS

- Treat the underlying cause
- Taper exogenous steroids when appropriate
- Surgery for pituitary, adrenal, or ectopic source if indicated



8 QUICK COMPARE

4 CUSHING CLUES

WEIGHT GAIN



HYPERTENSION



HYPOKALEMIA

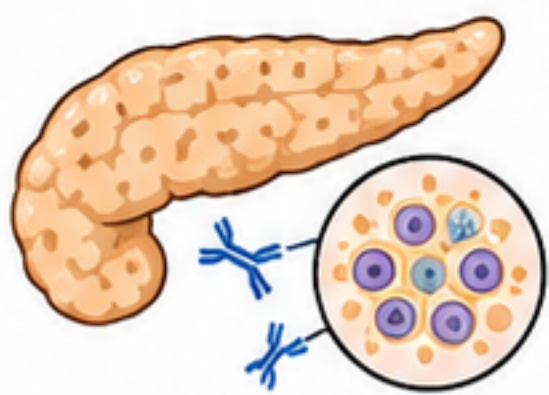


HYPERGLYCEMIA



9 EXAM PEARLS

- ★ Most common cause = steroid therapy
- ★ Cushing disease = pituitary ACTH adenoma
- ★ Moon face + purple striae
- ★ Cortisol excess causes Na retention and K loss



TYPE 1 DIABETES MELLITUS

High-Yield Exam Summary



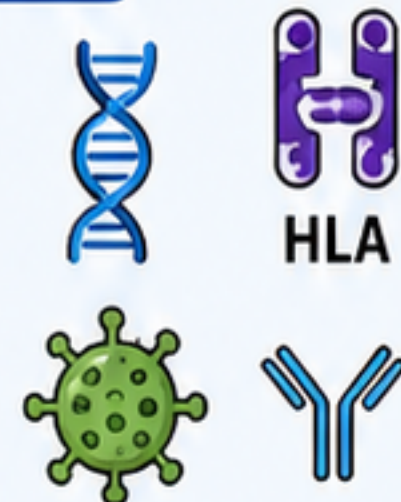
1 DEFINITION

Autoimmune pancreatic beta-cell destruction causing absolute insulin deficiency.



2 RISK FACTORS / PATHOGENESIS

- Genetic susceptibility
- HLA association (e.g., DR3-DQ2, DR4-DQ8)
- Environmental trigger (viral infection, etc.)
- Autoimmune-mediated beta-cell destruction



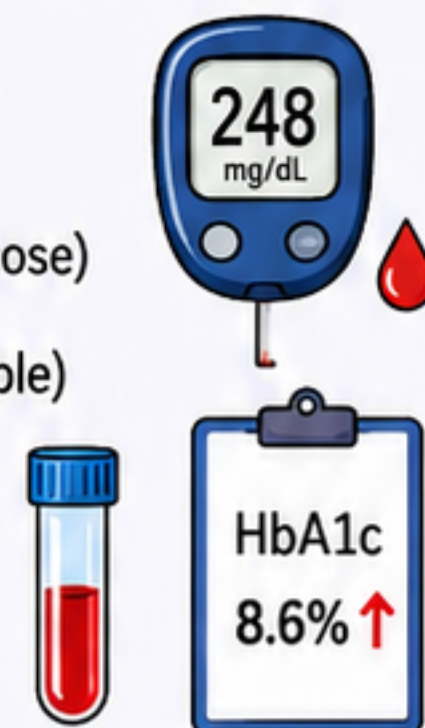
3 KEY SYMPTOMS & SIGNS

- Polyuria
- Polydipsia
- Polyphagia
- Weight loss
- Fatigue
- May present with DKA



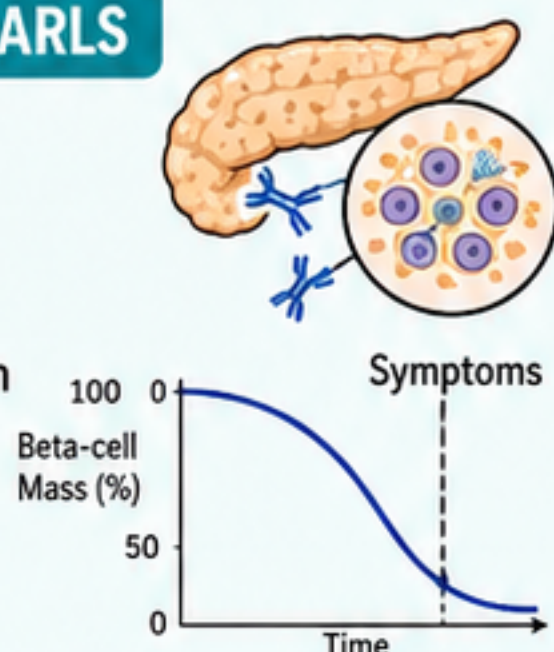
4 LABS / DIAGNOSIS

- Hyperglycemia (↑ fasting glucose, ↑ random glucose)
- Low C-peptide (low or undetectable)
- Autoantibodies may be present (GAD65, IA-2, ZnT8, ICA)
- HbA1c elevated



5 DISEASE COURSE PEARLS

- Preclinical immune phase (autoantibodies present)
- Symptoms appear after 80–90% beta-cell destruction
- Honeymoon phase can occur after diagnosis (transient partial remission)



6 COMPLICATIONS / IMPORTANT POINTS

- DKA is the major acute complication
- Lifelong insulin dependence



7 MANAGEMENT BASICS

- Insulin therapy (basal-bolus regimen)
- Glucose monitoring (SMBG / CGM)
- Medical nutrition therapy (carb counting)
- Sick-day rules and ketone monitoring



8 QUICK COMPARE TYPE 1 DIABETES CLUES

	Younger onset (childhood / adolescence)
	Lean habitus
	Autoimmune
	Insulin required (from diagnosis)

9 EXAM PEARLS



Absolute insulin deficiency



Low C-peptide



Autoimmune beta-cell destruction



DKA common



DIABETIC KETOACIDOSIS (DKA)

High-Yield Emergency Summary



1 DEFINITION

- Acute diabetic emergency characterized by hyperglycemia, ketosis, and high anion gap metabolic acidosis.



2 COMMON TRIGGERS

- Insulin omission
- Infection / illness
- First presentation of type 1 diabetes



3 PATHOPHYSIOLOGY

Severe insulin deficiency
+ excess glucagon

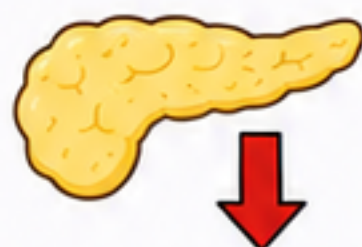
↑ Gluconeogenesis
and ↑ Lipolysis

Free fatty acids (FFAs)

Ketone bodies

(acetoacetate, β -hydroxybutyrate, acetone)

High anion gap
metabolic acidosis



4 KEY FEATURES



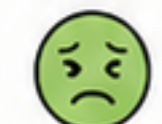
Polyuria



Dehydration



Abdominal pain



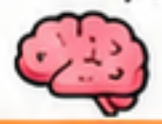
Nausea / vomiting



Kussmaul respirations
(deep, rapid breathing)



Fruity (acetone) breath



Altered mental status

5 DIAGNOSTIC LABS



- Glucose: often 500–700 mg/dL
- Ketonemia / ketonuria: positive
- pH: < 7.30 (acidosis)
- Serum bicarbonate (HCO_3^-): < 18 mEq/L
- Anion gap: > 12 mEq/L
- Total body potassium deficit (K^+ low or normal, but stores are depleted)

6 IMPORTANT POINTS / COMPLICATIONS

- Severe dehydration
- Cerebral edema (especially in children)
- Coma
- Death if untreated



7 MANAGEMENT BASICS



- IV fluids: restore circulation and perfusion
- Insulin: regular insulin IV after fluids
- Potassium: monitor closely and replace as needed
- Treat precipitating cause (e.g., infection, MI, missed insulin)

8 QUICK COMPARE: 4 DKA CLUES

KETONES



Ketones present
(blood / urine)

ACIDOSIS



Low pH,
low HCO_3^-

KUSSMAUL BREATHING



Deep, rapid
respirations

FRUITY BREATH



Acetone causes
fruity odor

9 EXAM PEARLS



Most common
in type 1 DM



Insulin omission
is a classic trigger



High anion
gap acidosis



Acetone causes
fruity odor



TYPE 2 DIABETES MELLITUS



High-Yield Exam Summary

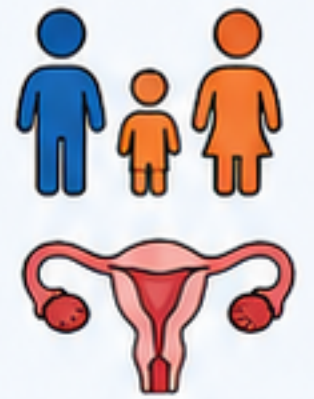
1 DEFINITION

- Insulin resistance with progressive beta-cell dysfunction and relative insulin deficiency.



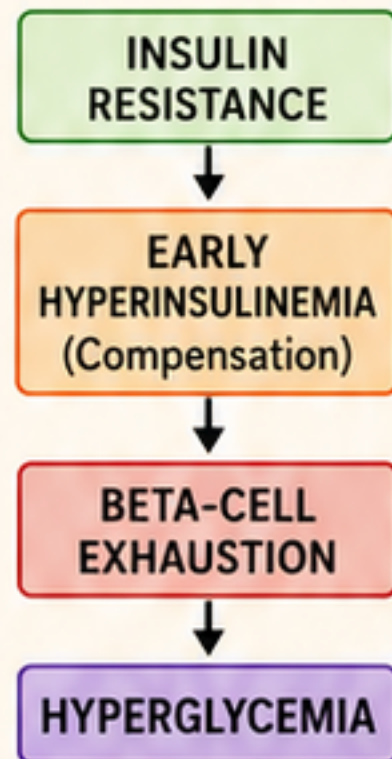
2 RISK FACTORS

- Obesity (especially visceral)
- Family history
- Physical inactivity
- Older age
- Gestational diabetes history
- PCOS



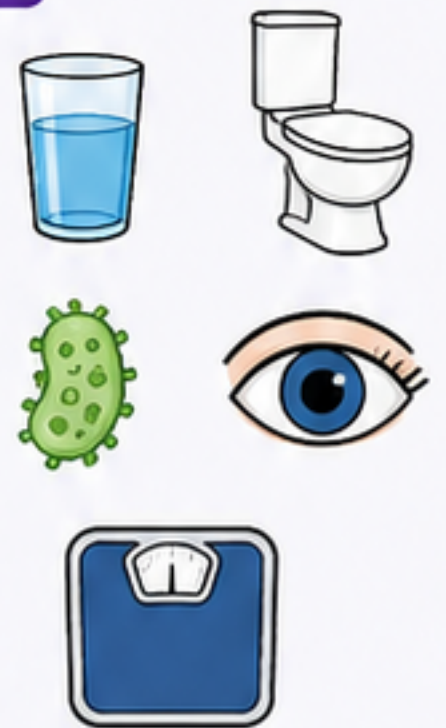
3 PATHOGENESIS

- Insulin resistance develops first.
- Early phase: pancreas compensates with hyperinsulinemia.
- Later phase: beta-cell exhaustion → decreased insulin secretion.
- Result: hyperglycemia.



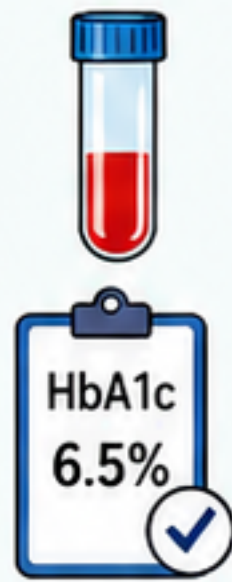
4 KEY CLINICAL FEATURES

- Often asymptomatic initially
- Polyuria
- Polydipsia
- Recurrent infections (e.g., UTIs, skin infections)
- Blurred vision
- Associated with obesity



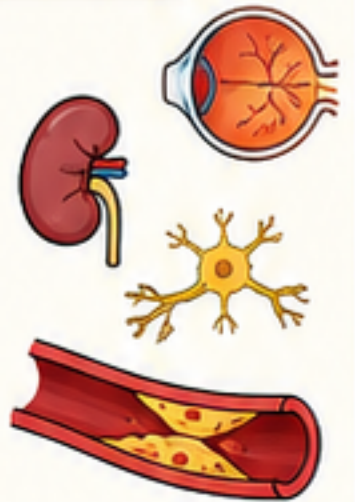
5 LABS / DIAGNOSIS

- Fasting plasma glucose ≥ 126 mg/dL
- HbA1c $\geq 6.5\%$
- 2-hour OGTT glucose ≥ 200 mg/dL
- C-peptide: often normal or high early (due to insulin resistance)



6 COMPLICATIONS / IMPORTANT POINTS

- Microvascular: retinopathy, nephropathy, neuropathy
- Macrovascular: CAD, stroke, peripheral arterial disease
- Hyperosmolar hyperglycemic state (HHS) is more typical than DKA.



7 MANAGEMENT BASICS

- Lifestyle modification: healthy diet, regular exercise, stop smoking
- Weight loss improves insulin sensitivity
- Metformin is first-line therapy in most patients (no contraindications)
- Add additional agents as needed (GLP-1 RA, SGLT2 inhibitor, DPP-4 inhibitor, TZD, sulfonylurea)
- Insulin if glycemic goals not met or with advanced disease



8 QUICK COMPARE

TYPE 2 DIABETES CLUES

	Obesity common
	Insulin resistance (first)
	Later onset (usually > 40 years)
	DKA uncommon; HHS more typical

9 EXAM PEARLS



90–95% of diabetes cases.



Visceral obesity is a major risk factor.



Hyperinsulinemia occurs early.



Beta-cell failure occurs later.





TYPE 2 DM RISK FACTORS



High-Yield Exam Summary

1 BODY WEIGHT / LIFESTYLE

- BMI ≥ 25
- Physical inactivity
- Central obesity



2 FAMILY / DEMOGRAPHIC RISK

- First-degree relative with diabetes
- High-risk ethnic groups (South Asian, African, Hispanic/Latino, Native American, Pacific Islander)



3 PREDIABETES / METABOLIC MARKERS

- Impaired fasting glucose (IFG)
- Impaired glucose tolerance (IGT)
- Hypertension
- Low HDL cholesterol
- High triglycerides
- Cardiovascular disease



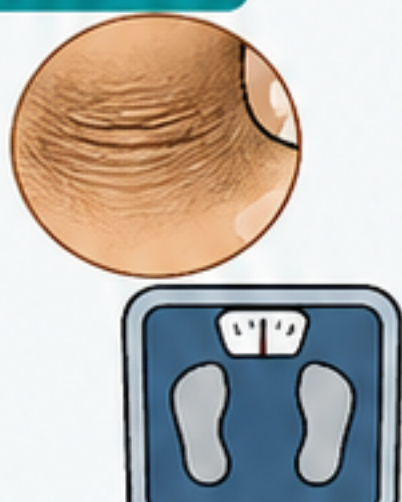
4 REPRODUCTIVE / FEMALE RISK

- History of gestational diabetes
- Delivery of baby >9 lb (4.1 kg)
- Polycystic ovary syndrome (PCOS)



5 INSULIN RESISTANCE CLUES

- Acanthosis nigricans (dark, velvety skin in neck, axilla, or groin)
- Severe obesity



6 WHY THESE MATTER



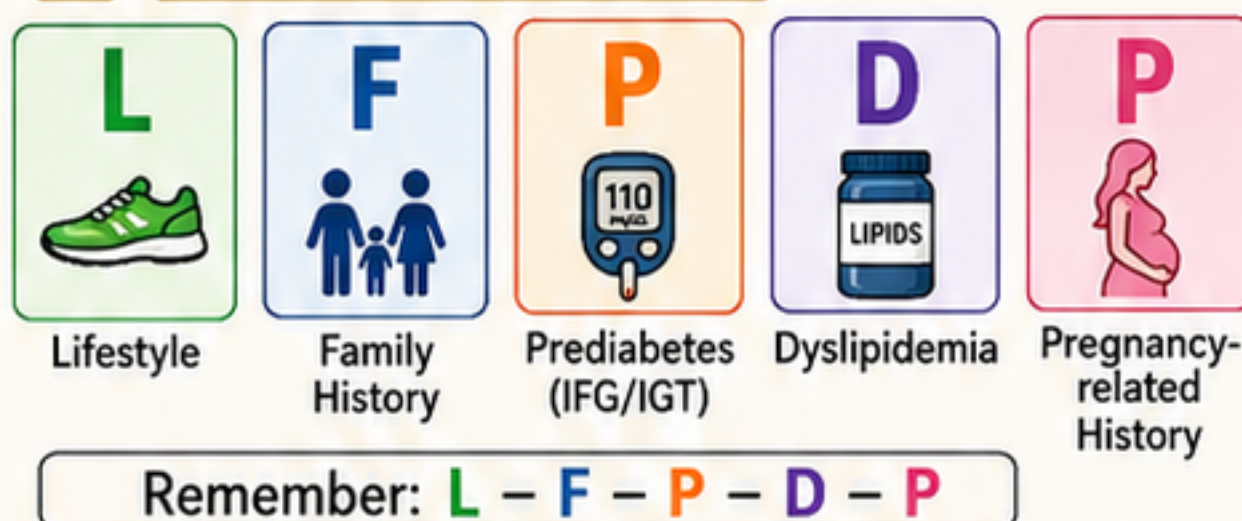
All increase insulin resistance and future risk of type 2 diabetes.

7 PREVENTION PEARLS

- Weight reduction
- Regular physical activity
- Blood pressure & lipid control
- Screen high-risk patients



8 QUICK MEMORY BOX



9 EXAM PEARLS



Obesity is the strongest risk factor.



Acanthosis nigricans suggests insulin resistance.



Prior GDM increases future type 2 risk.



IFG/IGT = high-risk state.



PREDIABETES

High-Yield Exam Summary



1 DEFINITION



Glucose levels above normal but below diabetes range.

2 DIAGNOSTIC CUTOFFS



FASTING PLASMA GLUCOSE (FPG)

● Normal	<100 mg/dL
● Impaired Fasting Glucose (IFG)	100–125 mg/dL
● Diabetes	≥126 mg/dL



ORAL GLUCOSE TOLERANCE TEST (OGTT, 2-HOUR)

● Normal	<140 mg/dL
● Impaired Glucose Tolerance (IGT)	140–199 mg/dL
● Diabetes	≥200 mg/dL

3 WHY IT MATTERS



Prediabetes is an intermediate stage before overt type 2 diabetes.

4 RISK OF PROGRESSION



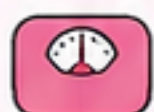
- Increased risk of progression to diabetes
- Roughly 5–10% per year

5 CARDIOVASCULAR RISK



- Increased CVD risk
- Often associated with low HDL, hypertriglyceridemia, insulin resistance

6 COMMON FEATURES / ASSOCIATIONS



Overweight



Central obesity



Sedentary lifestyle



Family history of type 2 diabetes

7 PREVENTION / MANAGEMENT BASICS



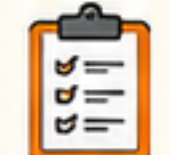
Healthy diet
(e.g., whole foods, less sugar, more fiber)



Regular exercise
(≥150 min/week moderate activity)



Weight loss
(5–10% of body weight)



Regular screening
(especially if high risk)

8 QUICK COMPARE

CLUE	NORMAL	PREDIABETES	DIABETES
● Fasting Glucose (mg/dL)	<100	100–125 (IFG)	≥126
● 2-Hour OGTT (mg/dL)	<140	140–199 (IGT)	≥200
● Future Diabetes Risk	Low	Increased (5–10%/yr)	High
● Cardiovascular Risk	Baseline	Increased	High

9 EXAM PEARLS

- ★ IFG = 100–125
- ★ IGT = 140–199
- ★ Prediabetes is reversible with lifestyle change
- ★ CVD risk is already increased





DIABETES COMPLICATIONS

High-Yield Exam Summary



1 BIG PICTURE

Chronic hyperglycemia causes both acute and chronic complications.



2 ACUTE COMPLICATIONS

- **DKA (Diabetic Ketoacidosis)**
More common in type 1 diabetes
- **HHS (Hyperosmolar Hyperglycemic State)**
More common in type 2 diabetes
- **Hypoglycemia**
From insulin or other glucose-lowering therapy

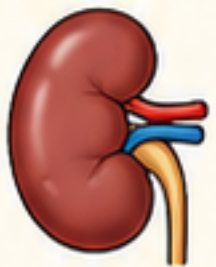


3 MICROVASCULAR COMPLICATIONS



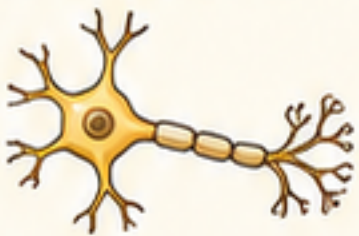
Retinopathy

Leads to vision loss and blindness



Nephropathy

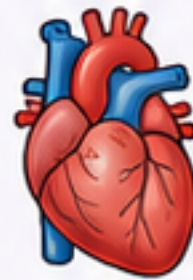
Leads to proteinuria, CKD, and ESRD



Neuropathy

Peripheral neuropathy → numbness, pain, loss of sensation

4 MACROVASCULAR COMPLICATIONS



Coronary Artery Disease / Myocardial Infarction

Accelerated atherosclerosis in coronary arteries



Stroke

Increased risk of ischemic and hemorrhagic stroke



Peripheral Vascular Disease

Claudication, poor wound healing, increased risk of amputation

5 FOOT PROBLEMS



Neuropathy + poor circulation

↓
Ulcers → Infection

↓
Possible amputation

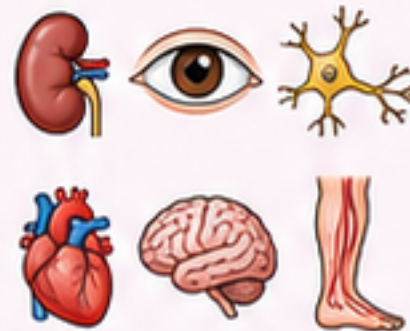
6 PREVENTION PEARLS

- ✓ Glucose control (A1C goal individualized)
- ✓ Blood pressure control
- ✓ Lipid control (statin as indicated)
- ✓ Foot care (daily inspection, proper footwear)
- ✓ Eye screening (annual dilated exam)
- ✓ Kidney monitoring (urine albumin, eGFR)



7 IMPORTANT POINTS

- Kidney, eye, and nerve damage are classic **microvascular** complications.
- Heart, brain, and peripheral vessels are **macrovascular** complications.



9 EXAM PEARLS

- ★ Retina, kidney, nerve = microvascular
- ★ MI, stroke, PVD = macrovascular
- ★ Chronic hyperglycemia drives complications
- ★ Foot care prevents ulcers and amputation



8 QUICK COMPARE

MICROVASCULAR (Small Vessel)		MACROVASCULAR (Large Vessel)	
	Retinopathy		Coronary Artery Disease / MI
	Nephropathy		Stroke
	Neuropathy		Peripheral Vascular Disease



Think: **SMALL VESSELS** (micro) affect kidneys, eyes, nerves.
BIG VESSELS (macro) affect heart, brain, and legs.