

# DIABETES MELLITUS

	<b>TYPE I</b> (5%)	<b>TYPE II</b> (90%)
<b>DEFECT</b>	Autoimmune destruction of pancreatic $\beta$ -cell • Severe glucose intolerance; Requires insulin to live	Obesity $\rightarrow$ Insulin resistance $\rightarrow$ pancreatic $\beta$ -cell failure • Mild glucose intolerance
<b>ONSET</b>	Sudden onset • < 20 years (youth); can occur at any age • Associated with HLA-DR3 & HLA-DR4	Gradual onset • > 40 years; strong genetic predisposition • Associated with obesity; $\uparrow$ age $\rightarrow$ $\downarrow$ insulin production
<b>SYMPTOMS</b>	Polydipsia and polyuria (osmotic diuresis) Polyphagia and weight loss (unopposed glucagon)	Often clinically silent (may have polyuria & polydipsia)
<b>SCREENING</b>	All adults > 45 every 3 years	

## DIAGNOSIS

HbA <sub>1c</sub>	$\geq 6.5\%$	Reflects blood glucose over RBC lifespan (~120 days) <5.7% = normal; diabetics should aim to keep their HbA <sub>1c</sub> < 7%
Fasting plasma glucose (x2)	$\geq 126$	Fasting for > 8 hours < 100 = normal
2 hour oral glucose tolerance test	$\geq 200$	Most sensitive test, but expensive and inconvenient Preferred test in gestational diabetes, PCOS and CF related diabetes < 140 = normal
Random plasma glucose	$\geq 200$	Symptoms must also be present <140 = normal

<b>TREATMENT</b>	<b>MECHANISM</b>	<b>ADVANTAGES</b>	<b>SIDE EFFECTS</b>
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Diet & exercise			
Metformin	$\uparrow$ Insulin sensitivity in liver	Weight loss. Doesn't cause hypoglycemia	GI upset, lactic acidosis Contraindicated in renal failure
Sulfonylureas	Stimulate pancreatic insulin production	Inexpensive	Weight gain, hypoglycemia
Acarbose	$\downarrow$ GI glucose absorption		GI upset
Thiazolidinediones	$\downarrow$ Insulin resistance in fat & muscle	$\downarrow$ insulin levels	Hepatotoxicity (monitor LFTs)

## CHRONIC COMPLICATIONS & MANAGEMENT

	Tight blood glucose control $\rightarrow$ $\downarrow$ microvascular complications (nephropathy, retinopathy); uncertain effect on microvascular complications (MI, stroke) Diabetic sensorimotor polyneuropathy affecting: Small nerve fibers $\rightarrow$ "positive" sx's (pain, paresthesia, allodynia) Large nerve fibers $\rightarrow$ "negative" sx's (numbness, loss of proprioception & vibration)
Heart	Check cholesterol levels every year; if LDL > 100 $\rightarrow$ statin Check BP at every visit; if >130/80 $\rightarrow$ ACEi or ARB MI is most common cause of death in diabetics
Kidney	Screen for microalbuminemia (30-300 in 24hrs) every year; if present $\rightarrow$ ACEi or ARB Check BUN/Creatinine every year
Eye	Annual screening for diabetic retinopathy Complications: retinopathy (hemorrhage, microaneurysms, vessel proliferation), glaucoma, cataracts
Nerves	Annual pediatric exam; Monofilament testing is used to determine presence of peripheral neuropathy Complications: peripheral neuropathy, erectile dysfunction, gastroparesis (tx: metoclopramide)
Misc.	All diabetics > 30 $\rightarrow$ daily aspirin (81mg) All diabetics should receive the pneumococcal vaccine

ACUTE COMPLICATIONS	DKA	HHS
Patient	Type I DM; younger age	Type II DM; older age
Presentation	Kussmaul respiration, blurred vision, altered mentation	Altered mentation
Glucose (mg/dL)	250-500	> 600 (often > 1,000)
Bicarbonate (mEq/L)	< 18	> 18
Anion Gap	↑, ⊕ serum ketones	Normal, ⊖ serum ketones
Serum Osmolality (mOsm/kg)	< 320	> 320

### MANAGEMENT OF DKA & HHS

IV Fluids	0.9% normal saline; once glucose $\leq$ 200, add dextrose 5%
Insulin	Continuous IV insulin infusion; switch to SQ insulin once glucose $\leq$ 200, Bicarb > 18, or AG < 12
Potassium	If serum K <sup>+</sup> $\leq$ 5.2 → IV potassium If serum K <sup>+</sup> < 3.3 → hold insulin Nearly all patients are K <sup>+</sup> depleted, even with 'hyperkalemia'
Bicarbonate	Consider for patients with pH < 6.9
Phosphate	Consider for serum phosphate < 1, cardiac dysfunction, or respiratory depression Monitor serum calcium frequently

Serum anion gap and  $\beta$ -hydroxybutyrate are the best markers for indication of DKA resolution