

DIABETES MELLITUS

	TYPE I (5%)	TYPE II (90%)
DEFECT	Autoimmune destruction of pancreatic β -cell • Severe glucose intolerance; Requires insulin to live	Obesity \rightarrow Insulin resistance \rightarrow pancreatic β -cell failure • Mild glucose intolerance
ONSET	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
SYMPTOMS	Polydipsia and polyuria (osmotic diuresis) Polyphagia and weight loss (unopposed glucagon)	Often clinically silent (may have polyuria & polydipsia)
SCREENING	All adults > 45 every 3 years	

DIAGNOSIS

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

TREATMENT

	MECHANISM	ADVANTAGES	SIDE EFFECTS
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 ≤ 200 , add dextrose 5%
Insulin	Continuous IV insulin infusion; switch to SQ insulin once glucose ≤ 200 , Bicarb > 18 , or AG < 12
Potassium	If serum K ⁺ ≤ 5.2 → IV potassium If serum K ⁺ < 3.3 → hold insulin Nearly all patients are K ⁺ 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 β -hydroxybutyrate are the best markers for indication of DKA resolution