# Diabetes mellitus

Health & Medicine, Diabetes

DIABETES MELLITUS * Chronic multisystem dz , abnormal insulin production / impaired utilization * Disorder of glucose metabolism related to absent/ insuff insulin supply or poor utilization of inslin that’s available * 7th leading cause of death * leading cause of blindness, ESRD, lower limb amputation * contributing factor for heart dz/ stroke risk 2-4 x higher than without DM * INSULIN — hormone produced by cells in islets of Langerhans of pancreas. Normal — continously into bloodstream ( basal rate), or increased w/ meals (bolus) * Normal glucose range 70-120 mg/dL, average insulin secreted daily 40-50 U 0. 6 U/kg * Glucagon, epinephrine, GH, cortisol oppose effects of insulin counterregulatory hormones they blood glucose lebels, stimulate glucose production by liver, movement of glucose into cells. * Insulin released from cells — as precursor / proinsulin thru liver enzymes form insulin & C-peptide ( C-peptide in serum & urine indicator of cell function) * in plasma insulin after meal storage of glucose as glycogen in liver/ muscle, inhibits gluconeogenesis, fat deposition, protein synthesis * Nl overnight fasting release of stored g; ucose from liver, protein from muscle, fat from adipose tissue * Skeletal muscle & adipose tissue receptors for insulin insulin-dependent tissues Type I Diabetes * Juvenile onset, insulin-dependent, s/s abrupt but dz process present for several yrs, 5-10%, absent or minimal insulin production, virus/toxins, under 40, 40% before 20 yr * s/s thirst( polydipsia), polyuria, polyphagia ( hunger), fatigue, wt loss, Kussmaul respirations * immune mediated dz; T-cells attack & destroy cells * genetic predisposition & exposure to virus * Idiopathic diabetes — not atoimmune, strongly inherited, in small # pt w/ type I DM , African/Asian * Predisposition HLAs human leukocyte antigens when exposed to viral infection cells

destroyed * Long preclinical period, s/s develop when pancreas can no longer produce sufficient insulin to maintain nl glucose levels * Req. insulin from outside source exogenous insulin eg. injection * No insulin diabetic ketoacidosis (DKA) life threatening, results in metabolic acidosis * “ honeymoon period" — newely diagnosed pts, tx initiated pt experience remissions req little insulin because cells produce suff amount of insulin lasts 3-12 mths then req permanent insulin Prediabetes * risk for developing diabetes * glucose levels high but not high enough for diabetes diagnosis * impaired fasting glucose IGF 100-125 mg/dL * 2 hr oral glucose tolerance test OGTT 140-199 mg/dL * HgB A1C — 5. 7%-6. 4% risk for diabetes * Increased risk for developing DM type II — if no preventive measures develop DM in 10 yrs * Long term damage to body heart, blood vessels occur in prediabetes * Usually no symptoms * Maintain healthy weight, exercise regularly, healthy diet risk of developing diabetes Type II Diabetes * Adult onset, non-insulin dependent, 90% * > 35, overweight, tendency to run n families * African Am, Asian, Hispanics, Amerian Indians * Some insulin is produced but either insufficient for body needs / poorly utilized * Gradual onset, many yrs undetected hyperglycemia, 500-1000mg/dL * Early usu. asymptomatic; high risk pt screen annually * Fatigue, recurrent inf, vaginal yeast inf, candida inf, prolonged wound healing, visual changes * Risk factor obesity ( abdominal/ visceral ) * 4 major metabolic abnormalities * insulin resistance > tissue no response to insulin / unresp receptors — receptors are located on skeletal muscles, fat & liver * ability of pancreas to produce insulin — fatigued from compensatory prod of insulin, cell mass lost * inappropriate glucose by liver — too much glucose for body needs — type II *

altered prod. of hormones & cytokines by adipose tissue ( adipokines) role in glucose & fat metabolism — type II. Two adipokines ( adiponectin & leptin ) affect insulin sensitivity altered mechanism in type I & I * Metabolic syndrome > risk for type II & cardio dz, cluster of abnormalities, insulin resistance, insulin levels, triglycerides, HDLs, LDLs, HTN * Risk factors for metabolic syndrome central obesity, sedentary lifestyle, urbanization, westernization Gestational Diabetes * During pregnancy, 7% of pregnancies * High risk — severe obesity, prior hx of gestational DM, glycosuria, polycystic ovary syndrome, family hx of DM II screened at 1st prenatal visit * Average risk OGTT at 24-28 wks of gestation * Higher risk of cesarean delivery, perinatal death, neonatal complications * Will have nl glucose levels within 6 wks postpartum but risk of DM II in 5-10 yrs * Nutritional therapy — 1st line , if doesn’t work insulin therapy Other specific types of diabetes * Due to other medical condition or treatment causes abn blood glucose levels * Damage , injury, destruction of cell function * Cushing’s, hyperthyroidism, pancreatitis, cystic fibrosis, hemochromatosis, TPN * Meds > corticosteroid (prednisone), thiazides, phenytoin(Dilantin), antipsychotics — clozapine * Tx underlying condition, stop meds Diagnostic studies * A1C > 6. 5 % ; greater convenience, no fasting req, less day to day alterations during stress/ illness * FPG > 126 — no caloric intake for 8 hrs prior testing ; confirmed by repeated testing another day; if has s/s and FPG> 126 further testing OGTT not req * 2 hr OGTT > 200, glucose load 75g accuracy depends on pt preparation, and factors that influence results. False negative > impaired GI absorption, falsely elevated> severe restrictions of carbs, acute illness, meds corticosteroids, contraceptives, bed rest * IFG impaired fasting glucose & IGT

> prediabetes, 100-125 mg/dL, IGT 2 hr > 140-199 * Glycosylated HgB — HgB A1C > amount of glucose attached to HgB molecules over lifespan ( RBC 90-120 days ) DM pts should check it regularly, done to monitor success of tx / make changes to tx < 6. 5 % - risk of retinopathy, nephropathy, neuropathy dz affecting RBCs — can affect A1C results Treatment * Goals > s/s, promote well being, prevent acute complications, prevent/ delay onset/ progression; met when pt maintain glucose level as near to nl, daily decisions about food intake, blood glucose testing meds, exercise * Rapid acting insulin - lispro (Humalog), aspart (NovoLog) — onset 0-15 min, peak 60-90 min, dur. 3-4 hrs , clear, give 15 min before meals ; bolus * Short acting — Regular (Humulin R, Novolin R) onste ½ -1 hr, peak 2-3hr, dur 3-6 hrs, injected 30-45 min before meals; bolus * Intermediate acting — NPH, basal insulin, onset 2-4hrs, peak 4-10hrs can result in hypoglycemia, dur. 10-16 hrs, can be mixed w/ short & rapid, cloudy, must be agitated before adm. * Long acting — glargine (Lantus), detemir ( Levemir) addition to mealtime insulin, type I, to control glucose between meals & overnight, without it risk of developing DKA, no peak — risk of hypoglycemia , not diluted or mixed, clear; onset 1-2 hrs, dur. 24hrs +, basal * Combination > pt don’t want 2 separate injections, 2 type of insulin mixed together, not same control of glucose levels as with basal-bolus; ahort/rapid mixed w/ intermediate provide both mealtime & basal coverage * Storage > vials room temperature 4 wks, heat & freezing alter insulin, between 32-86 F; avoid direct exp to sunlight, extra insulin in fridge/ traveling-thermos, Prefilled syringes — sight impaired, manual dexterity; syringes w/ c; udy solution in vertical position needle up to avoid clumping of suspension, rolled gently, warm before injection. * Injection > abdomen

fastest absorption arm, thigh, buttock, rotate within 1 particular site; never into site that’s about to be exercised (heat = absorption & onset), vial 1ml= 100U, SQ 90 degrees * Needles ½- 5/16 inch (short — children, thin adults); gauges 28, 29, 30, 31 — higher gauge = smaller diameter = more comfortable injection * Recapping done only by person using syringe, never recap syringe used by pt; alcohol swabs in health care facility before inj to HAI, at home soap & water * Insulin pump — continuous subq insulin infusion 24 hr/d basal rate , loaded w/ rapid acting insulin via plastic tubing to catheter in subq tissue. At meal time — bolus . (+) tight glucose control, similar to nl physiologic pattern, nl lifestyle, more flexibility (-) infection at site, risk of DKA, cost Problems w/ insulin therapy * Hypoglycemia * Allergic rxn - itching, erythema, burning around inj. site, may improve w/ low dose antihistamine ; rxns to Zinc, protamine, latex , rubber stoppers on vials * Lipodystrophy - atrophy of subq tissue if same inj site used * Somogyi effect — rebound effect, overdose of insulin induces undetected hypoglycemia in hrs of sleep, produces glucose decline in response to too much insulin s/s headaches, night sweats, nightmares ; if in morning glucose — adcised to check glucose levels at 2-4am if hypoglycemia present at that time. If it is insulin dosage in affecting morning blood glucose is reduced TX : less insulin * Dawn phenomenon — hyperglycemia on awakening in the morning due to release counterregulatory hormones in predawn hrs ( possibly GH/cortisol) adolescence/ young; TX: adjustment in timing of insulin adm. or in insulin. Predawn fasting glucose levels insulin production from pancreas , s. eff > wt gain, hypoglycemia * Meglitinides repaglinide(Prandin) insulin prod, less likely cause hypoglycemia because more rapidly absorbed/eliminated, cause

wt gain, take 30 min before meal, not if skipped * Biguanides — Metformin > glucose lowering, first choice DM II/prediabetes, obese & “ starch blockers" slow down carbs absorption, taken with “ first bite", effectiveness> check 2 hr postprandial glucose levels * Thiazolidinediones — Avandia > “ insulin sensitizers", for pts w/ insulin resistance, don’t insulin Production, not cause hypoglycemia; risk of MI, stroke , not for pt w/ HF * DPP4 inhibitor — Januvia > new class, slow inactivation of incretin hormones; DDP4 inh are glucose dependent = risk of hypoglycemia, no wt gain * Incretin mimetics — exenatide (Byetta) > stimulate incretin horm which are in DM II, stim. of insulin, Suppress glucagon, satiety = caloric intake, slows gastric emptying; prefilled pen * Amylin analog > Amylin hormone secreted by cells, co secreted w/ insulin Pramlintide (Symlin) is Synthetic , type I & II when glucose level not achieved w/ insulin at mealtimes , subq thigh or abdomen NOT arm , not mixed w/ insulin — cause severe hypoglycemia ! * blockers --- masks s/s of hypoglycemia, prolong hypoglycemic effects of insulin * Thiazide / loop diuretic --- hyperglycemia, K Nutrition * Type I > meal planning, exercise, developed w/ pt’s eating habits & activity pattern in mind, day to day consistency in timing & amount of food eaten * Type II > wt loss = improved insulin resistance, total fats & simple sugars = calorie & carbs intake; Spacing meals , wt loss 5-7% = glycemic control, regular exercise * Carbohydrates > sugar, starches, fiber whole grains, fruits, veggies, low fat milk included min 130g/d * Glycemic index GI > describe blood glucose levels 2 hrs after carb meal , GI of 100 = 50g glucose * Fiber intake 14g/1000 kcal * Fats 7% of total calories , < 200mg/d cholesterol & trans fats * Protein same for diabetes / normal renal function / gen.

population, high proein diet not recommended * Alcohol > inhibits gluconeogenesis ( breakdown of glycogenglucose) by liver; severe hypoglycemia in pt on insulin / oral hypoglycemic dx. Moderate alcohol consumption < 2 drinks men, track carbs w/ each meal & daily, set limit for max amount ( depends on age, wt, activity level) usu. 45-60g /meal ; also My Pyramid & plate method ( ½ nonstarchy veggies, ¼ starch, ¼ protein, nonfat milk & fruit * Exercise > 150 min/wk moderate intensity aerobic; DM II resistance training 3 x wk, most adults should 30 min moderate intensity activity 5 x most days * Exercise > insulin resistance, blood glucose, wt loss which insulin resistance ( may need less meds), triglycerides, LDL, HDL, BP, circulation * Start slowly w/ progression. Insulin, sulfonylureas, meglitinides > risk of hypoglycemia with increase physical activity esp if exercise at peak of dx or no food intake. Effect may last 48 hrs post exercise Exercise 1 hr after meal, have 10-15g carb snack every 30 min. during exercise (prevent hypoglycemia). Before exercise glucose immediate info about glucose levels — can make adjustments diet, activity, meds * Recomm. for all insulin-treated pts * Multiple insulin injections — 3 or more x day, done before meals, before & after exercise esp in type I, whenever hypoglycemia suspected, when ill (stress), 2 hrs after start of meal — if effective Pancreas transplantation * For pt w/ ESRD, plan to have kidney transplant * Pancreas transplanted following kidney transplant, pancreas alone —rare * Pancreas alone only if hx of severe metabolic complications, emotional problems w/ exogenous insulin, failure of insulin-based management * Improve quality of life, no exogenous insulin need, no dietary restrictions * Only partially able to reverse renal & neurologic complications * Need lifelong immunosuppression

to prevent rejection * Pancreatic islet cell transplantation in experimental stage, islets from deceased pancreas via catheter into abdomen portal vein Nursing management * Pt active participant in management of diabetes regimen * Few/no episodes of acute hyper/hypoglycemic episodes, maintain glucose level near nl * Prevent/ delay chronic complications * Adjust lifestyle to accommodate DM regimen w/ min. stress Nursing assessment * Past hx mumps, rubella, viral inf, recent trauma, stress, pregnancy, infant> 9lbs, Cushing, acromegaly, family hx of DM * Meds > compliance w/ insulin, OA; corticosteroids, phenytoin, diuretics * Eyes > sunken eyeballs, vitreal hemorrhages, cataract * Skin > dry, warm, inelastic, pigmented lesions on legs, ulcers(feet), loss of hair on toes * Respiratory > Kussmaul — rapid, deep * Cardio > hypotension, weak rapid pulse * GI > dry mouth, vomiting, fruity breath * Neuro > altered reflexes, restlessness, confusion, coma * MS > muscle wasting * Also electrolyte abnormalities, fasting glucose level > 126, tolerance test> 200, leukocytosis, BUN, creatinine, triglycerides, cholesterol, LDL, HDL, A1C 45yrs without risk factors for diabetes Acute intervention * Hypoglycemia, DKA, HHS — hypersmolar hyperglycemic syndrome * Stress of acute illness/ surgery > counterregulatory hormones > hyperglycemia ( even minor upper resp infection or flu can cause this) * Continue regular diet, noncaloric fluids (broth, water, diet gelatin, decaffeinated), take OA/insulin as prescribed, monitor glucose Q4H * Acutely ill DM I , glucose> 240 test urine for ketones Q3-4H , medium/large report to MD * Ill > eat than normal > continue OA meds/ insulin as prescribed + carbohydrate containing fluids (soup, juices, decaffeinated) * Unable to keep fluids/ food down MD * Don’t stop insulin when ill counterregulatory

mechanisms will glucose level * Food intake important body needs extra energy to deal w/ stress Extra insulin may be needed to meet this demand, prevent DKA in DM I * Intraoperative > IV fluids & insulin before, during, after sx when there’s no oral intake In DM II w/ OA — explain it’s temporary measure, doesn’t mean worsening of DM * If contrast medium (w/iodine) > Metformin discontinued 1-2 days before sx, resumed 48 hrs after sx risk of acute renal failure. Resume after kidney function nl ( creatinine checked & is nl) * Insulin adm > teach proper administration, adjustments, side effects, assess response to insulin tx, if new to insulin assess ability to manage tx safely, cognitive status, ability to recognize/ tx hypoglycemia, if cognitive skill another responsible person must be assigned; diff to self inject/ afraid of needles * Follow ups > inspect injection sites ( lipodystrophy ) * Short term memory deficit > OA or short acting OA cuz doesn’t cause hypoglycemia * OA w/ diet & activity, not take extra pill when overeating * Diligent skin care & dental > aily brushing/ flossing, inform dentist about DM * Foot care !!! scrapes, burns treated promptly & monitored > nonirritating antiseptic ointment > dry sterile pad> not start to heal in 24 hrs or infection > MD * Regular eye exams * Travel — sedentary > walk Q2H to prevent DVT & prevent glucose , carry snacks, extra insulin COMPLICATIONS Diabetic Ketoacidosis DKA * Diabetic coma * Profound deficiency of insulin > hyperglycemia, ketosis, acidosis, dehydration * Most likely in DM I pts, but sometimes in DM II ( severe illness/ stress) * Causes > illness, infection, undiagnosed DM I, inadeq insulin dosage, poor self management, neglect * Insulin - glucose cant be properly used for energy fat broken for fuel ketones (by product) serious when excessive in blood alter pH, cause metabolic

acidosis ketonuria (in urine) & electrolyes depleted; impaired protein synthesis, nitrogen lost from tissues * Untreated depletion of Na, K, Cl, Mg, phosphate hypovolemiarenal failure/ retention of ketones & glucose shockcoma (result of dehydration, lytes & acidosis)death * s/s > dehydration, poor turgor, dry mm, HR, orthostatic hypotension, Kussmaul , abdominal pain, sunken eyeballs, acetone fruity odor, early s/s > lethargy, weakness * blood glucose > 250, arterial blood pH IV access begin fluid/ electrolyte replacement NaCL 0. 45% or 0. 9% to restore urine output 30-60 ml/hr & BP * glucose level approach 250 5% dextrose added * Incorrect fluid repl > sudden Na & cerebral edema * Obtain K level before insulin started — insulin > further K * Insulin withheld until fluid resuscitation & K> 3. 5 * Too rapid IV fluids & rapid lowering of glucose cerebral edema Hypersmolar hyperglycemic syndrome HHS * Life threatening, able to produce insulin to prevent DKA but not enough to prevent severe hyperglycemia, osmotic diuresis, ECF depletion * Less common than DKA * Often > 60, in DM II * Causes > UTI, pneumonia, sepsis, acute illness, new DM II * Asymptomatic in early stages > so glucose can rise very high > 600mg/dL * The higher glucose > in serum osm > neurologic manifestations somnolence, coma, seizures, hemiparesis, aphasia * Resemble CVA (stroke) determine glucose level for correct dx * Ketones absent in urine * Tx similar to DKA * First IV 0. 45% or 0. 9% NS, regular insulin given after fluid replacement * Glucose fall to 250 — add glucose 5% dextrose * Hypokalemia not as significant as in DKA * HHs require greater fluid replacement * Assess VS, I&O, turgor, labs, cardiac / renal monitoring related to hydration & electrolyte levels, mental status, serum osm Hypoglycemia * Low blood glucose glucagon &

epinephrine > defense against hypoglycemia * s/s of epinephrine > shaking, palpitations, nervousness, diaphoresis, anxiety, hunger, pallor * brain req constant supply of glucose > when > affect mental functioning > LOC, diff speaking, visual disturbances, confusion, coma, death * Hypoglycemis unawareness > no warning signs until glucose reach critical point > incoherent, combative, LOC > often elderly w/ beta blocker meds * When very high glucose level falls too rapidly, too vigorous management of hyperglycemia * Mismatch in timing of food intake & peak of isulin/ OA * Can be quickly reversed * Check glucose levels, if contain fat that glucose absorption; check glucose in 15 min * Still 70 eat regular meal/snack low peanut butter, bread, cheese, crackers, check glucose in 45 min * No significant imptovement after 2-3 doses of 15g carb MD * Pt not alert to swallow 1mg glucagon IM in deltoid muscle ( nausea, vomiting rebound hypoglycemia) * Hospital setting > 20-50ml of 50% dextrose IV push * CHRONIC COMPLICATIONS OF DM Angiopathy * end organ dz from damage to blood vessels (angiopathy) 2nd to chronic hyperglycemia * leading cause of diabetes-related deaths, 68% deaths due to cardio, 16% strokes * causes: accumul. Of glucose metabolism by products (sorbitol) damage to nerve cells, abnormal glucose molecules in basement membrane of small blood vessels (eye, kidney), derangement in RBCs - oxygenation to tissues * DM I > keep blood glucose levels near to normal - retinopathy & nephropathy (complications of microvascular complications) Macrovascular complications * Dz of large, medium size blood vessels , earlier onset in pt w/ diabetes * W > 4-6x risk of cardiovascular dz, M > 2-3 x * risk factors > obesity, smoking, HTN, fat intake & sedentary lifestyle * Smoking injurious to pt w/DM, risk for

blood vessel dz, CV dz, stroke, lower extremity amputations * Maintain BP control — prevention of CV / renal dz Microvascular complication * Thickening of vessel membranes in capillaries/ arterioles in response to chronic hyperglycemia * Are specific to diabetes * Eyes ( retinopathy ), kidneys ( nephropathy ), skin (dermopathy ) * Some changes present w/DM II at time of dx, but s/s not appear until 10-20 yrs after onset of DM * Diabetic retinopathy — microvascular damage to retina, most common cause of blindness 20-74 yrs old. Nonproliferative> most common, partial occlusion of small blood vesselin retina microaneurysms, Proloferative> most severe, involves retina & vitreous neovasculization ( form new blood vessels to compensate) if macula involved vision is lost * DM II > dilated eye exam at time of diagnosis & annually, DM I within 5 yrs after DM onset * Laser photocoagulation * Virectomy * Glaucoma Nephropathy — microvascular complication, damage to small blood vessels that supply glomeruli / kidney. Leading cause of ESRD in US; same risk for DM I & II > HTN, smoking, genetic predisposition, chronic hyperglycemia * Screen for nephropathy annually w/ measurement albumin / creatinine ratio * If micro/macroalbuminuria > ACE inh ( lisinopril ) or angiotensin II rec antagonist ( Cozaar ) tx HTN & delay progression of nephropathy * Aggressive BP management & tight glucose control Neuropathy Sensory neuropathy (PNS)— loss of protective sensation in lower extremities amputations * Hyperglycemia > sorbitol & fructose accumulate in nerves damage * Distal symmetric polyneuropathy > hand/ feet bilaterally * Loss of sensation — to touch/ temperature * Pain > burning, cramping, crushing, tearing , at night * Paresthesias > tingling , burning, itching * At times skin

too sensitive (hyperesthesia) * Foot injury & ulcerations without having pain * TX : blood glucose control, topical creams capsaicin ( Zostrix ) 3-4 X/d pain in 2-3 wks, selective serotonin, norepinephrine reuptake inh ( Cymbalta ), pregabali ( Lyrica ), gabapentin Autonomic neuropathy — can affect all body systems & lead to hypoglycemic unawareness, bowel incontinence, diarrhea, urinary retention Complications : * Delayed gastric emptying ( gastroparesis ) anorexia, n/v, reflux, fullness, can trigger hypoglycemia by delaying food absorption * Cardiovascular abnormalities , postural hypotension assess change from lying, sitting, standing, painless MI, resting tachycardia HR * Risk for falls * Sexual dysfunction > ED in diabetic men > 1st s/s of autonomic failure * Neurogenic bladder > urinary retention, diff. voiding, weak stream empty bladder Q3H in sitting position, Crede maneuver ( massage lower abdomen) * Cholinergic agonists > benthanechol Feet & lower extremities * Risk for foot ulcerations & lower extremity amputations * Sensory neuropathy > major rosk for amputations due to loss of protective sensations LOPS * Unaware of foot injury, improper footwear, stepping on objects w/ bare feet * Screening using microfilament > insensitivity to 10g Semmes-Weinstein > risk for ulcers * Proper footwear, avoid injuries, diligent skin care, inspect feet daily * PAD risk for amputations due to blood flow to lower extremities * PAD s/s > intermittent claudication, pain at rest, cold feet, loss of hair, cap refill, dependent rubor ( redness when extr in dependent position ) * DX : ankle brachial index ABI & angiography * Casting to redistribute weight on plantar surface * Wound control > debridement, dressings, vacuum, skin grafting etc. * Charcot’s foot > ankle & foot changes joint deformity need fitted footwear * Acanthosis nigricans — dark, coarse,

thickened skin in flexures & neck * Necrobiosis lipoidica diabeticorum — DM I, red-yellow lesions w/ atrophic skin , shiny & transparent revealing blood vessels under the surface — young women * Granuloma annulare — DM I, autoimmune, partial rings of papules, dorsal surface of hands/ feet Infection Candida albicans, boils, furuncles, bladder infections (glycosuria) antibiotics Gerentologic * reduction in cells, insulin sensitivity, altered carbohydrate metabolism * 20 % > 65 YO * # of conditions treated w/ meds that impair insulin action (