

# UPDATE ON DIABETES

## I. DEFINITIONS

- A. Diabetes—"to siphon"; Mellitus—"sweet"
- B. "Taste thy patient's urine"—Dr. Thomas Willis (16<sup>th</sup> century)
- C. Diabetes Mellitus—a chronic disorder of carbohydrate, fat, and protein metabolism characterized in its fully expressed clinical form by an absolute deficiency of insulin (Type 1 diabetes) or a relative insulin deficiency (Type 2 diabetes).

## II. DIAGNOSIS

- A. High risk groups at any age with symptoms (polyuria, polydipsia) and all individuals at age 45 should be screened for diabetes

High risk groups for Type 1—family history, sibling or parent with Type 1  
High risk groups for Type 2—1<sup>st</sup> degree relative with diabetes, BP greater than 140/90; history of impaired glucose tolerance, including gestational diabetes and having a baby weighing greater than 9 pounds; high risk ethnic group; obesity greater than 120% of desirable body weight or a body mass index (BMI) greater than 27 kg/m<sup>2</sup> (with a BMI of 30 kg/m<sup>2</sup> or greater, the risk of diabetes 10-20 times normal); undesirable lipid levels (HDL less than 35 mg/dL or triglycerides greater than 250 mg/dL); family history of early onset coronary artery disease; history of hypoglycemia; polycystic ovary syndrome (PCOS)

1. FPG (fasting plasma glucose) – blood drawn after 8 hours without caloric intake)—morning specimens greater than or equal to 126 mg/dL (previous recommendation for diagnosis was an FPG of 140 mg/dL); an FPG of 140 mg/dL is the equivalent of a postprandial glucose (PPG) of greater than 200 mg/dL, which is associated with microvascular complications; afternoon fasting blood sugars of greater than 114 mg/dL are considered positive
2. Hemoglobin A<sub>1c</sub> (glycosylated hemoglobin)----not presently recommended for the diagnosis of diabetes; However, it is used as the gold standard measure of long-term glycemic control in persons with diagnosed diabetes.

- a. normal range is 4-6% ; each percent equals 20-22 mg/dL of plasma glucose, therefore a HbA<sub>1c</sub> of 4% is the equivalent of a FPG of 80-88 mg/dL; a HbA<sub>1c</sub> of 10% is the equivalent of a FPG of 200-220 mg/dL.

### III. CLASSIFICATIONS

- A. Type 1 diabetes mellitus—primarily diagnosed in pre-teens or teenagers; onset prior to age 40 in the majority of patients; Caucasians greater than African Americans or Hispanic Americans; accounts for 5-10% of all diabetics in the U.S.; Finland is #1 country in the world for Type 1 DM (1 in every 150 kids develops diabetes by age 15)
  - 1. an absolute lack of effective insulin
  - 2. 80% of the 1-1.5 million Islet cells in the pancreas secrete insulin. Type 1 diabetes presents after 90% of the beta cells have been destroyed
  - 3. Associated with an abnormal immune response; genetic predisposition to autoimmune associated events with antibodies to insulin or islet cells; the antibodies appear three to ten years prior to the overt onset of the disease
    - a. What triggers the autoimmune response?
      - 1. virus? Protein in cow's milk?
- B. Type 2 diabetes mellitus—approximately 800,000 new cases per year; approximately 20 million in U.S. Usual onset over age 40 however in the last decade the prevalence of diabetes jumped 70% a
- C. Among people in their 30s. Overall, diabetes increased 33% from 1990-1998. The increase was greatest among Hispanics, for whom the rate increased 38%, compared to Caucasians with a 29% increase in rate, and African Americans with a 26% increase; obesity and lack of physical activity as well as genetics play a major role in the development of Type 2 diabetes
  - 1. Ethnic prevalence—percent of total population that has been diagnosed with diabetes
    - a. Caucasian (4.6% in 1990; 5.9% in 1998)
    - b. African-American (7.0% in 1990; 8.9% in 1998)
    - c. Hispanic (5.6% in 1990; 7.7% in 1998)
    - d. Native American Indians have a prevalence rate that is 5 times that of Caucasians

\*\* Within a single ethnic group, diet and lifestyle play a huge role in the development of obesity and accompanying insulin resistance and DM. Among Pima Indians of Arizona, obesity is the norm and DM occurs in more than 50% of the adult population. However, Pima Indians living a

more traditional lifestyle in northern Mexico do not develop obesity and DM is rare

2. Genes also play a role—having a first degree relative with Type 2 diabetes increases the risk; lifestyle increases the risk—obesity, high calorie/high fat diet, sedentary lifestyle
- D. Pre-diabetes—asymptomatic; fasting blood sugars between 100-125; 1-5% of Caucasians develop diabetes within 5-10 years, 25% of African-Americans develop diabetes within 5-10 years; majority remain in this class or return to normal glucose tolerance; the state of impaired glucose tolerance is considered a risk factor for developing coronary artery disease

#### IV. TYPE 2 DIABETES MELLITUS

- A. Metabolic derangements aren't usually as severe as Type 1; with insulin resistance, serum glucose levels gradually increase; with increasing glucose levels the pancreas responds by increasing insulin output resulting in hyperinsulinemia
1. may have few, if any symptoms initially; 2 P's instead of 3 (polyuria and polydipsia without significant polyphagia); weight gain due to the hyperinsulinemic state
  2. This process may continue for a full decade as a "silent" disease; unfortunately it isn't so "silent" in the tissues
- B. COMPLICATIONS OF TYPE 2 DIABETES –ATHEROSCLEROSIS (may manifest before the diabetes is diagnosed)
1. Atherosclerosis—Coronary artery disease, cerebrovascular disease, peripheral vascular disease
    - a. coronary artery disease and angina
      1. estrogen does NOT protect the premenopausal diabetic woman from heart disease
    - b. peripheral vascular disease and intermittent claudication
      1. risk of amputation is 15-40 times higher in the diabetic; 50 percent die within 3 years of amputation
    - c. cerebrovascular disease and transient ischemic attacks
  2. LDL-cholesterol is the bad guy and is considered to be the major atherogenic lipoprotein. Triglycerides are also atherogenic, however

LDL-C is the predominant cholesterol carrying lipoprotein and is the foremost contributor to the increased risk associated with high serum cholesterol.

1. decrease LDL-C by reducing intake of trans and saturated fats; increase monosaturated fats (olive oil and canola oil); increase whole grains and fiber
2. ASA to prevent platelet aggregation—low dose; clopidogrel (Plavix)
3. “STATINS” to reduce the production of LDL-C and triglycerides in the liver
  - a. the “STATIN” sisters—lova (Mevacor), prava (Pravastatin), simva (Zocor), atorva\*(Lipitor), fluva (Lescol), rosuva (Crestor) keep LDL-C at 70 mg/dl and triglycerides less than 150 mg/dl; HDL-C above 40mg/dl in men, above 50 mg/dl in women
  - b. Other ways to reduce cholesterol--Green tea, flaxseed, Take Control, Benecol, Garlic, B vitamins to reduce homocysteine, booze, soy products, almonds, viscous fiber

For every 1% decrease in glycosylated hemoglobin (hemoglobin A<sub>1c</sub>)—which corresponds to a 20-22 mg/dl decrease in plasma glucose levels—yields a 25% reduction in the risk of diabetic complications.

### C. DIABETIC PERIPHERAL NEUROPATHY (DPN)

1. Screening for sensory loss in the feet of patients with diabetes currently offers the best chance for early detection of DPN—small fiber sensory loss resulting initially in the loss of pain, light touch, and temperature followed by the loss of vibratory sensation as degeneration of the large myelinated fibers progresses.
2. Neuropathic pain can range from uncomfortable tingling or burning to disabling pain in the legs and feet; “tightness or constriction”, “walking on hot coals”, “walking on shards of glass”
3. Treatment goals—improve blood sugar control, relieve pain
  - a. OTC analgesics for mild pain
  - b. TCA’s—amitriptyline (Elavil) or nortriptyline (Aventyl, Pamelor); venlafaxine (Effexor)
  - c. Anticonvulsants—gabapentin (Neurontin), topiramate (Topamax)
  - d. Mexiletine (Mexitol)—an antiarrhythmic agent and local anesthetic

- e. Acupuncture, TENS, PNT (percutaneous neuromodulation therapy), magnet therapy
  - f. Narcotics
4. Autonomic neuropathy—should be evaluated and followed by a cardiologist
- a. Orthostatic hypotension—BP falls when getting up
  - b. Gastroparesis—consider in patients who exhibit wide swings in blood sugar, as their slowed digestion interferes with the timing of insulin injections relative to meals; symptoms include early satiety, chronic nausea and vomiting, of food ingested hours before
  - c. Erectile dysfunction—many causes
    - 1. atherosclerosis and neuropathy are the two major causes
    - 2. sildenafil (Viagra) is an effective treatment in 50% of the cases (contraindications – heart disease, nitrates, hypertension, macular degeneration
    - 3. injections, implants, suction devices
  - d. Impaired bladder emptying with hydroureter, hydronephrosis, and chronic infection
    - 1. urecholine, DuVoid
- D. DIABETIC NEPHROPATHY—check kidney function at every visit; monitor urine for “microalbuminuria”
- 1. Use of ACE inhibitors to decrease angiotensin 2 and subsequently dilate the efferent arteriole and decrease intraglomerular hypertension;
    - a. THE “PRILS”—captopril (Capoten), enalapril (Vasotec), ramipril (Altace), lisinopril (Zestril, Prinivil), etc.
    - b. THE “SARTANS”—angiotensin 2 blockers; losartan (Cozaar), valsartan (Diovan), candesartan (Atacand)
  - 2. reduce animal protein in the diet, reduce serum glucose, treat systemic hypertension (if present)

E. DIABETIC RETINOPATHY—refer to ophthalmologist; the diabetic has a 25x greater risk of blindness and partial loss of vision than the nondiabetic; risk of cataracts is 4-6 times greater; 2x greater risk of glaucoma; EARLY detection is imperative

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