Module 12 Ch. 68 Diabetes

Normal Blood Glucose Levels 60-150 mg/dL

1. Diabetes Mellitus
- A group of metabolic diseases characterized by hyperglycemia
- Results from a defect in insulin secretion or absorption
- Hyperglycemia from an insulin deficiency, resistance to insulin action, or both
- Relative or absolute lack of insulin or inadequate function of insulin
- Chronic hyperglycemia is associated with long-term damage, dysfunction and failure of numerous organs

Hyperglycemia Symptoms:
- Polyuria (excessive urination)
- Polydipsia (excessive thirst, drinking)
- Polyphagia (excessive hunger, eating)
- Glycosuria (sugar in the urine) ketones
- Weight loss
- Weakness
- Blurred vision
- Frequent or recurrent infections, increased susceptibility to infections
- Impaired growth
- Slow healing
- Tingling, numb, cramps (neuropathy)
- Fatigue
- Eye, kidney, cardiovascular problems,
- Impotence (erectile dysfunction)
- Diabetic Coma (metabolic acidosis) ketoacidosis

8% US population is diagnosed with diabetes. 1/3rd have not yet been diagnosed.
Aging population with increase in obesity = increased prevalence
Most costly health condition: prevalence, cost of treatment, lost productivity

2. Insulin
Hormone produced by the pancreas beta cells (Isles of Langerhans). Insulin is released into the blood in proportion to the blood glucose level. Insulin allows glucose transport into cells for cell energy, which results in decreased blood glucose levels and energy for the cells.
Without sufficient insulin, hyperglycemia results
Pancreas secretes the hormones insulin and glucagon into the bloodstream. The release of insulin into the blood lowers the level of blood glucose by enhancing glucose by cells, where it is metabolized. If blood glucose gets too low, the pancreas secretes glucagon to stimulate the release of glucose from the liver.

2. Description of Insulin:
- Food ingested and converted into glucose

- Increase in blood glucose level stimulates the pancreas to release insulin into the blood stream
- Insulin enables glucose transport into cells to use as energy
- Blood glucose level then decreases

2. Functions of Insulin:
- Facilities glucose uptake from blood into tissues
- Speeds the oxidation of glucose within the cells to use for energy
- Speed the conversion of glucose to glycogen to store in the liver and skeletal muscles and prevents the reverse condition
- Facilitates conversion of glucose to fat in adipose tissue

3, 5. Type I Diabetes Mellitus
- Used to be called Juvenile onset, 5-10%
- Absolute insulin deficiency due to destruction of beta cells from the islets of Langerhans in the pancreas
- Insulin dependent
- Prone to ketoacidosis
- Common in childhood or puberty and Caucasians

Effects of Decreased Insulin
- Less glucose transmitted through cell walls into the cells
- Glucose increases in circulating blood (hyperglycemia) until a threshold is reached and glucose spills over into the urine (glycosuria)
- Increased glycosuria induces osmotic diuresis with excretion of large amounts of urine (polyuria) water and electrolytes are lost
- Fluid loss signals excessive thirst to the brain (polydipsia)
- Cell starving for glucose may cause pt. to increase food intake (polyphagia), but weight loss may still occur
- Without glucose for energy, the body metabolizes fat for energy. End products of fat metabolism are harmful ketones that accumulate in the blood. Ketones are acidic, accumulate, neutralized in the blood, when the quantity is large, neutralizing effect is depleted rapidly and an acidic condition (metabolic acidosis) results.
- Metabolic acidosis leads to diabetic coma (ketoacidosis) if not treated promptly

3,5. Type II Diabetes Mellitus

<u>Former names:</u>
- Non-insulin dependent diabetes mellitus (NIDDM)
- Adult – onset diabetes
- Maturity onset diabetes
- Ketosis – resistant diabetes

- Same symptoms as Type 1
- Most prevalent type 90-95%
- Acute complications are rare, chronic long-term complications are common.
- Ketoacidosis is rare
- More stable and easy to manage

- Chronic manifestations develop slowly at later stages
- Insulin secretion may be high or low but the patient is resistant to the utilization of insulin
- Relative insulin resistance – the inability of the tissues to respond to the insulin
-Slow progression of disease over years. This has an early mild severity but progressively serious.
- Usually develops after age 30

Risk Factors: (lots)

- African American, Native American, Hispanics
- More frequent occurrence in families
- Obesity, sedentary lifestyles, high-fat diets

3. Effects of decreased Insulin

- Cell surface insulin receptors develop defects; glucose cannot be transmitted into the cell
- Blood glucose level increases as the insulin resistance of cells increases. Stimulates more insulin to be released
- Overtime, insulin secretion may also decline and lead to both decrease of insulin in blood and increased insulin resistance of cells.

4. Insulin Complications * Understand these*****

Hypoglycemia/ Insulin Shock

- Too much insulin (hyperinsulinism) lowers level of blood glucose (hypoglycemia)
- Hypoglycemia would occur more likely in the dental setting
- Sudden onset

Hyperglycemia Reaction/ Diabetic Coma (Ketoacidosis)

- Too little insulin (hypoinsulinism) with increased levels of blood glucose (hyperglycemia)
- Develops over long time

6. Gestational Diabetes Mellitus

Effects on Mother

- Could have any degree of glucose intolerance with pregnancy
- Related to genetics, obesity and hormones causing insulin resistance usually in 3rd Trimester
- Occurs in about 4% US pregnancies
- Diagnosis is reclassified 6 or more weeks post-pregnancy. Maternal dangers are reduced by insulin adjustment, supervised prenatal care and improved obstetric practices.

Effects on Infant

- Larger infants
- More frequent premature births
- High incidence of congenital malformations and perinatal death
- Tendency to develop Type 2 diabetes later in life