Diabetes Mellitus

Definition: Diabetes Mellitus is defined as a chronic metabolic disease characterized by increased levels of BG as a result of defects in insulin secretion and insulin action

Cost of Diabetes

Over $1 of every $5 spend on US health care is for a person with diabetes

Annual cost of diabetes in US medical expenses is over $116 billion

People with diabetes spend over $12,000 /yr

Incidence of Diabetes

Nearly 24 million children & adults have DM in US
Almost 8% of the US population
Incidence of DM increases yearly
1.6 million new cases of DM last yr
DM is the 7th leading cause of death

Discussion Question

Why has the incidence of diabetes increased in recent years?

Implications for Nursing

The key to decreasing the incidence of DM is prevention.
type 2 diabetes can be prevented with appropriate changes in lifestyle.
For Persons at high risk for type 2 diabetes standard - intensive program lifestyle modifications can lower risk by 58% for both genders and all racial and ethnic groups. type 2 diabetes can be prevented or delayed in persons at high risk for the disease.

The key to limiting cost is containment of complications.

The challenge for RN and pt is achieving & maintaining meticulous BG control so that long term complications can be prevented

Development and progression of complications such as retinopathy, nephropathy, and neuropathy.

ASSESSMENT

Clinical Manifestations

depend on the patient's level of hyperglycemia.

Classic clinical manifestations of all types of diabetes:

Polyuria and polydipsia due to excess loss of fluid associated with osmotic diuresis

polyphagia that results from the catabolic state induced by insulin deficiency and the breakdown of proteins and fats.

Other symptoms include :

fatigue and weakness, sudden vision changes, tingling/numbness in hands or feet, dry skin, skin lesions/wounds that are slow to heal, & recurrent infections.

onset of type 1 diabetes may also be associated with sudden weight loss or nausea, vomiting, or abdominal pains, if DKA has developed.

Assessment and Diagnostic Findings

abnormally high blood glucose level is the basic criterion for the diagnosis of diabetes.

Fasting plasma glucose (FPG), random plasma glucose, and glucose level 2 hours after receiving glucose (2-hour postload) may be used.

OGTT and the IV glucose tolerance test are no longer recommended for routine clinical use.

DIAGNOSIS

1. Symptoms of diabetes (the 3 Ps + obesity or recent wt lost) and casual plasma glucose concentration = or > 200 mg/dL.

or

2. Fasting plasma glucose greater than or equal to 126 mg/dL

Fasting is defined as no caloric intake for at least 8 hours.

or

3. Two-hour postload glucose equal to or greater than 200 mg/dL during an oral glucose tolerance test. (not usually used for routine clinical))

In the absence of unequivocal hyperglycemia with acute metabolic decompensation, these criteria should be confirmed w/repeat testing on a different day.

History

Symptoms related to the diagnosis of diabetes:

- Symptoms of hyperglycemia
- Symptoms of hypoglycemia - Frequency, timing, severity, and resolution

Results of blood glucose monitoring

Status, symptoms, and management of chronic complications of diabetes:

- Eye; kidney; nerve; genitourinary and sexual, bladder, and gastrointestinal. Cardiac; peripheral vascular; foot complications associated with diabetes

Adherence to/ability to follow prescribed dietary management plan, exercise regimen, follow prescribed pharmacologic treatment

Use of tobacco, alcohol, and prescribed and over-the-counter medications/drugs

Lifestyle, cultural, psychosocial, and economic factors that may affect diabetes treatment

Effects of diabetes or its complications on functional status (eg, mobility, vision)

Physical Examination

- Blood pressure (sitting and standing to detect orthostatic changes)
- Body mass index (height and weight)
- Fundoscopic examination and visual acuity
- Foot examination (lesions, signs of infection, pulses)
- Skin examination (lesions and insulin-injection sites)
- Neurologic examination
    - Vibratory and sensory examination using monofilament
    - Deep tendon reflexes
- Oral examination

Laboratory Examination

- $HgbA_{1C}$ (A1C)
- Fasting lipid profile
- Test for microalbuminuria
- Serum creatinine level
- Urinalysis
- Electrocardiogram

Need for Referrals

- Ophthalmology
- Podiatry
- Dietitian
- Diabetes educator
- Others if indicated

Gerontological

- ↑BG levels appear to be age related and occur in both men and women throughout the world.
- commonly appear in the 5^{th} decade and increase in frequency with advancing age.
- Approximately 10% to 30% of elderly people have age-related hyperglycemia, not counting those with overt diabetes.
- What causes age-related changes in carbohydrate metabolism is not known.
- Possibilities include:

poor diet, physical inactivity, ↓in lean body mass (ie decreased glucose storage), altered insulin secretion, and ↑in fat tissue, which ↑'s insulin resistance

Risk Factors for Diabetes Mellitus

Family history of diabetes (ie, parents or siblings with diabetes)
Obesity (ie, ≥20% over desired body weight or BMI ≥27 kg/m2)
Race/ethnicity (eg, African Am., Hispanics,
Native Americans, Asian Americans, Pacific Islanders)
Age ≥45 y
Prev. identified impaired fasting glucose or impaired glucose tolerance
Hypertension (≥140/90 mm Hg)
HDL cholesterol level ≤35 mg/dL (0.90 mmol/L)
and/or triglyceride level ≥250 mg/dL (2.8 mmol/L)
History of gestational diabetes or delivery of babies over 9 lb

Pancreatic islet cells (Islets of Langherhorn's) Influences CHO metabolism, indirectly affects PRO and FAT metabolism
Insulin - Beta Cells Lowers BG by facilitating glucose transport across cell membranes of muscle, liver, & adipose tissue ↓ **Insulin:** DM - Type I, Type II, LADA or Type 1.5 In Blood: Hyperglycemia, Lipidemia, Ketoacidosis In urine: Glycosuria, Ketonuria, Na+ & K+ loss
Glucagon – Alpha Cells - Stim'd by low BG ↑BG conc by stim of glycogenolysis & glyconeogenesis
Somatostatin – Delta Cells Delays intestinal absorption of glucose Suppresses insulin and glucagon release

Anatomy & Physiology of Pancreas

Endocrine gland - produces hormones; insulin, glucagon, somatostatin
Exocrine gland – releases sodium bicarbonate and pancreatic enzyme

Insulin is secreted by beta cells, which are one of four types of cells in the islets of Langerhans in the pancreas. Insulin is an anabolic, or storage, hormone. When a person eats a meal, insulin secretion increases and moves glucose from the blood into muscle, liver, and fat cells. In those cells, insulin
Transports and metabolizes glucose for energy
Stimulates storage of glucose in the liver and muscle (in the form of glycogen)
Signals the liver to stop the release of glucose
Enhances storage of dietary fat in adipose tissue
Accelerates transport of amino acids (derived from dietary protein) into cells
Insulin also inhibits the breakdown of stored glucose, protein, and fat.
During fasting periods (between meals and overnight), the pancreas continuously releases a small amount of insulin (basal insulin); another pancreatic hormone called glucagon (secreted by the alpha cells of the islets of Langerhans) is released when BG levels decrease and stimulates the liver to release stored glucose. Insulin & glucagon together maintain a constant level of glucose in the blood by stimulating the release of glucose from the liver.

Pathophysiology of Diabetes Mellitus - Classification of Diabetes Mellitus - research findings suggest many differences among individuals within each category. Second, except for people with type 1 diabetes, patients may move from one category to another.

Type 1 a metabolic disorder characterized by an absence of insulin production & secretion from autoimmune destruction of the beta cells

Type 2 a metabolic disorder characterized by the relative deficiency of insulin production and a decreased insulin action and increased insulin resistance;

Metabolic Syndrome up trigycerides, up LDL, down HDL, Abd obesity, high fasting BG, HTN (any of three)

Gestational Diabetes is any degree of glucose intolerance w/ onset during preg. Hyperglycemia develops during pregnancy because of the secretion of placental hormones, which causes insulin resistance. Occurs in as many as 14% of pregnant women and ↑their risk for HTN disorders during pregnancy, high risk pts should be screened and rescreened at 24-28 wks if neg. initial tx includes diet mod and BG monitoring. If continues then insulin.
Goal is 105 mg/dL or less before meals and 130 mg/dL or less 2 hours after meals. After birth can return to normal but many progress to type 2 later in life.

Prediabetes or IGT (impaired glucose tolerance) classified as impaired glucose tolerance (IGT) or impaired fasting glucose (IFG) and refers to a condition in which BG concentrations fall between normal levels and those considered dx for diabetes. Can progress to DM, stay the same, or return to normal levels

Type 1 (5–10% of all diabetes)

Onset any age, but usually young (<30 y)
Usually thin at diagnosis; recent weight loss
Etiology includes genetic, immunologic, and environmental factors (eg virus)
Often have islet cell antibodies
Often have antibodies to insulin even before insulin treatment
Little or no endogenous insulin
Need insulin to preserve life
Ketosis prone when insulin absent
Acute complication of hyperglycemia: diabetic ketoacidosis

beta cell destruction results in:
decreased insulin production,
unchecked glucose production by the liver,
and **fasting hyperglycemia**.
Unstored glucose from food remaining in the bloodstream
Glycosuria If BG exceeds renal threshold (180-200 mg/dL)
osmotic diuresis: excess glucose is excreted, big loss of F & E follows it
unrestrained glycogenolysis (breakdown of stored glucose) **and gluconeogenesis** (production of new glucose from amino acids and other substrates), contributing further to hyperglycemia.
In addition, **fat breakdown** occurs, resulting in an increased production of ketone bodies, which are the byproducts of fat breakdown.
diabetic ketoacidosis (DKA) may cause S&S: abdominal pain, nausea, vomiting, hyperventilation, a fruity breath odor, and, if left untreated, altered level of consciousness, coma, and death.
Insulin treatment, along with F&E as needed, is essential TX of hyperglycemia and DKA and rapidly improves the metabolic abnormalities.

LADA or Type 1.5

Usually leaner, thinner & responsive to insulin
New Classification of DM
Latent Autoimmune Diabetes
10% of people with Type 2 DM
Genetically has components of Type 1 & 2
Criteria for DX:
Adult onset
Presence of auto antibodies in blood
No insulin needed for initial 6 months
Still being researched

Type 2 (90%)

30-40% eventually require meds
Onset any age, usually over 30 y
Usually obese at diagnosis
Causes include obesity, heredity, and environmental factors
No islet cell antibodies
Decrease in endogenous insulin, or increased with insulin resistance
Most patients can control BG through weight loss if obese
Oral antidiabetic agents may improve BG if diet and exercise are unsuccessful
May need insulin on a short-term or long-term basis to prevent hyperglycemia (common for these pts to be on both PO meds and inj insulin)
DKA uncommon, unless stress or infection
Acute complication: hyperglycemic hyperosmolar nonketotic syndrome

insulin resistance and impaired insulin secretion mechanisms in type 2 diabetes are unknown, genetic factors are thought to play a role.
In an effort to overcome insulin resistance and to prevent the buildup of glucose in the blood, increased amounts of insulin are secreted to maintain the glucose level at a normal or slightly elevated level.
This is called metabolic syndrome, which includes hypertension, hypercholesterolemia, and abdominal obesity.
If the beta cells cannot keep up with the increased demand for insulin, the glucose level rises and type 2 diabetes develops.

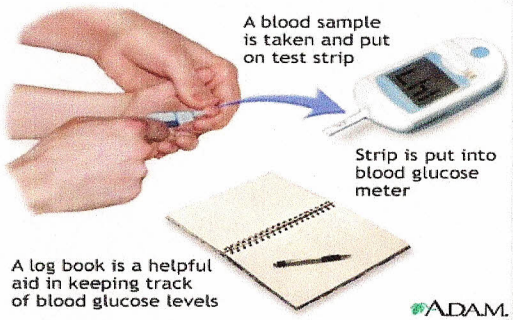
slow, progressive glucose intolerance is associated w/ Type 2 diabetes, its onset may go undetected for many years. If pt experiences symptoms, they are frequently mild and may include fatigue, irritability, polyuria, polydipsia, poorly healing skin wounds, vaginal infections, blurred vision (if BG very ↑).
It is critical to screen at risk pts to avoid long term complication like eye disease, peripheral neuropathy, PVD that can develop prior to dx

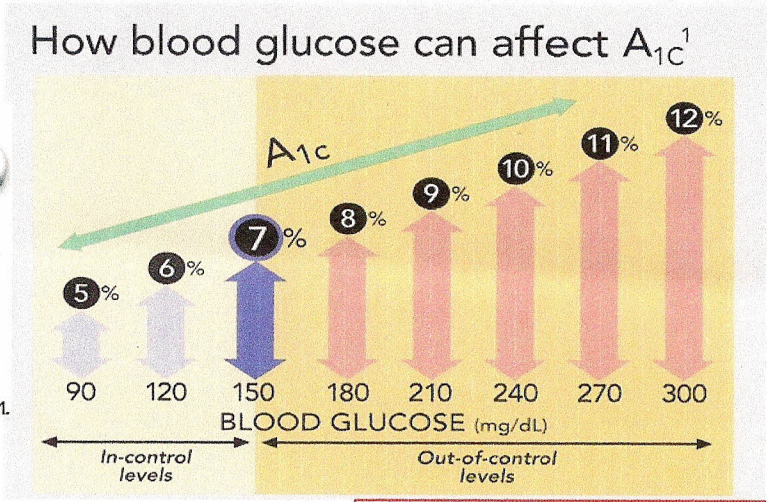
Diabetes mellitus associated with other conditions or syndromes

(Previously classified as secondary diabetes)
Accompanied by conditions known or suspected to cause the disease: pancreatic diseases, hormonal abnormalities, meds like corticosteroids & estrogen-containing preps.
Depending on pancreas ability to make insulin, pt may need TX w/ PO antiDM meds or insulin

Diagnostic Screening for DM (done twice)

ADA criteria for DM diagnosis:
Symptoms of DM (hyperglycemia) plus
Casual (random) Plasma Glucose (PG) >200 mg/dL
Fasting Plasma Glucose (FPG) >126 mg/dL
HgbAC1 contributes to dx if > 6.5





Hyperglycemia / Hypoglycemia

Normal BG 60-110mg/dL
HYPERGLYCEMIA Fasting BG greater than 110mg/dL
2hr after meal BG greater than 140 mg/ dL
HYPOGLYCEMIA Low BG level
Less than 60 mg/dL

MONITORING for DM pts: BG, Hemoglobin A1C, Ketones, Other

Advantages of BG monitoring:
BG key to DM management
Control promotes more normal BG
Normal BG reduces complications
SMBG allows adjustment of tx.
Limits hypoglycemic episodes

BG Monitoring Frequency:
2-4 times daily (HO orders) – finger is most accurate –arm is 20 min old
If hypo or hyperglycemia suspected
Insulin patients; at least 3 X daily, before each meal
Monitor occasionally, after fasting, 2 hours after a meal, & before bed
Increase monitoring with illness or changes in meds, activity or diet.
Continuous can be implanted or wireless

Cornerstone of DM Mgmt is:
BG Monitoring
SMBG affected by visual acuity, fine motor skill, cognitive ability, comfort w/ tech & willingness to use it, & cost.
SMBG risks: pt may obtain incorrect BG due to: incorrect techniques, improper application of blood (eg, drop too small), damage to reagent strips by heat or humidity, outdated strips, improper meter cleaning & maintenance.

BG Levels for DM: studies showed that lowering these values closer to normal ↑ hypoglycemic episodes & mortality rates
Normal Fasting BG (FBG): 60-110mg/dL
FBG w/ DM: <126mg/dL
Pre-prandial: before meal: 70-130 mg/dL
Post-prandial: after meals: less than 180 mg/dL

HgbA1c - Glycated Hemoglobin
Measures all RBC and bound glucose for 2-3 mo period
Definition: blood test that reflects the average BG over 2-3 months.
done Q3 months in new DM until control of BG is achieved.
Test is done Q 6months for controlled DM
Goal is less than 7.0% (closer to normal is better) Normal: 5.5-6.0

Implications of Ketones
Tests that are + with small - medium amounts need to be monitored.
Tests that are + with lg amts need to be treated and correlated with the BG.
Large volumes can lead to DKA - Diabetic Ketoacidosis

Other:
Serum Triglycerides & Cholesterol :
Lipid Panel – 1X per year
Total cholesterol of <200
Triglyceride of < 150
HDL >40 mg/dl
LDL < 100
Urine test for protein
proteinuria can indicate early nephropathy.
Kidney function: Q 6 month.
Urine for microalbumin: 1X per yr.
Test will show changes in kidney function prior to blood tests
Normal is < 30 mg
Liver Function Tests: PO meds esp effect liver or if pt drinks
LFTs are done Q3- Q6 months for certain medications

Abnormalities in BG

Dawn Phenomena (complication of long term)

AM BG Elevation caused by release of either GH, Cortisol and/or endogenous insulin abnormalities.

Diagnosis is made by checking BG at 2 and 4 AM ; high levels = DP

Treatment: adjustment of insulin doses from dinner to bedtime or increase HS insulin

Somogyi's Effect (complication of long term)

Wide differences in early morning (low) and fasting (high) BG.

BG drops below normal due to too much insulin at night.

Caused by counter-regulatory response to night-time low BG.

Hypoglycemia is seen in the middle of the night

Problem is AM BG may be high and therefore the provider or patient may add more PM insulin.

The low middle of night BG is missed.

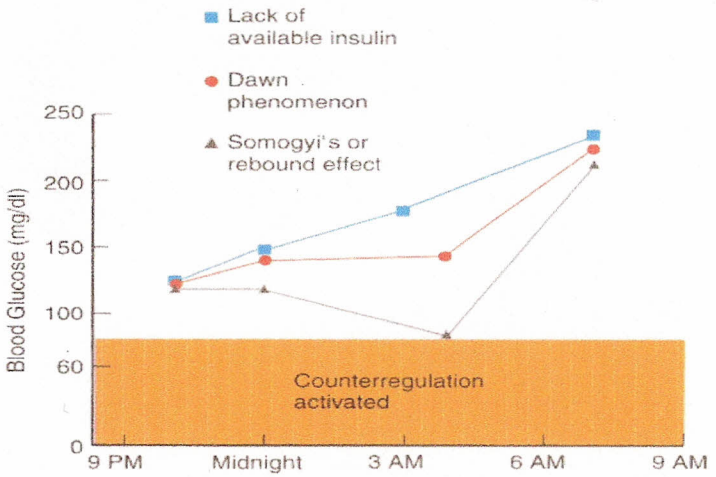


Figure 68–8. Three blood glucose phenomena in diabetic clients.

To diagnoses this the patient must awaken at 2 and 4 AM and check the BG. AM BG must also be checked.

If these test values are low (60 mg/dl) add more dietary intake at bedtime and give less insulin.

Morning Hyperglycemia and Hypoglycemia	
Characteristic	Treatment
Insulin Waning Progressive rise in blood glucose from bedtime to morning	Increase evening (predinner or bedtime) dose of intermediate-acting or long-acting insulin, or institute a dose of insulin before evening meal if one is not already part of tx regimen.
Dawn Phenomenon Relatively normal BG until about 3 AM, when the level begins to rise	Change time of injection of evening intermediate-acting insulin from dinnertime to bedtime.
Somogyi Effect Normal or elevated BG at bedtime, a decrease at 2–3 AM to hypoglycemic levels, and a subsequent increase caused by the production of counterregulatory hormones	Decrease evening (predinner or bedtime) dose of intermediate-acting insulin, or increase bedtime snack.

Sick Day Management (when stressed BG ↑ even if not eating)

Want BG to be <126 but at least < 150

Prevent Hyperglycemia and progression to DKA

Prevent dehydration

Promote Nutrition

Monitor BG every 3-4 hrs during illness

Continue meds based on BG

Sip 8-12 oz. of clear fluid hourly

Substitute easily digested foods

Call healthcare provider if not eating for 24 hrs

Call healthcare provider if vomiting or diarrhea for 6 hrs

Sick Day Guidelines for pts

Take insulin or oral antidiabetic agents as usual.

Test blood glucose and test urine ketones every 3 to 4 h.

Report ↑ BG (>300 mg/dL or as otherwise specified) or urine ketones to HO

If on SQ take insulin, may need supplemental doses of regular insulin q3-4h

If cannot follow usual meal plan, substitute soft foods (eg, 1/3 cup regular gelatin, 1 cup cream soup, ½ cup custard, 3 squares graham crackers) 6-8 serv/d.

If V&D, or fever persists, take liquids (eg, ½ cup regular cola or orange juice, ½ cup broth, 1 cup Gatorade) every ½ to 1 hour to prevent dehydration and to provide calories.

Report N, V&D to HO, because extreme fluid loss may be dangerous.

If unable to retain oral fluids, may require hospitalization to avoid DKA and possibly coma.