PT IN OTHER CONDITIONS

DIABETES MELLITUS

It refers to a group of common metabolic disorders that share the phenotype of hyperglycemia. Several distinct types of DM exist and are caused by a complex interaction of genetics and environmental factors. Depending on the etiology of the DM, factors contributing to hyperglycemia include reduced insulin secretion, decreased glucose utilization, and increased glucose production. The metabolic dysregulation associated with DM causes secondary pathophysiologic changes in multiple organ systems that impose a tremendous burden on the individual with Diabetes and on the health care system. In the United States, DM is the leading cause of end-stage renal disease (ESRD), non traumatic lower extremity amputations, and adult blindness. It also predisposes to cardiovascular diseases. With an increasing incidence worldwide, DM will be a leading cause of morbidity and mortality for the foreseeable future.

Etiological Classification

I. Type 1 Diabetes (-cell destruction, usually leading to absolute insulin deficiency)
A. Immune-mediated
B. Idiopathic
II. Type 2 Diabetes(may range from predominantly insulin resistance with relative insulin deficiency to a predominantly insulin secretory defect with insulin resistance)

Gestational DiabetesMellitus

Glucose intolerance may develop during pregnancy. Insulin resistance is related to the metabolic changes of late pregnancy, and the increased insulin requirements may lead to IGT.

Criteria for the Diagnosis

- Symptoms of Diabetesplus random blood glucose concentration 11.1 mmol/L (200 mg/dL) or
- Fasting plasma glucose 7.0 mmol/L (126 mg/dL) or
- Two-hour plasma glucose 11.1 mmol/L (200 mg/dL) during an oral glucose tolerance test
- Random is defined as without regard to time since the last meal.
- Fasting is defined as no caloric intake for at least 8 h.
- The test should be performed using a glucose load containing the equivalent of 75 g anhydrous glucose dissolved in water; not recommended for routine clinical use.

  Risk Factors for Type 2 DiabetesMellitus

  Family history of Diabetes (i.e., parent or sibling with type 2 diabetes)

  Obesity (BMI 25 kg/m^2)

  Habitual physical inactivity

  Race/ethnicity (e.g., African American, Latino, Native American, Asian American, Pacific Islander)

  Previously identified IFG or IGT

  History of GDM or delivery of baby >4 kg (>9 lb)

  Hypertension (blood pressure 140/90 mmHg)

  HDL cholesterol level <35 mg/dL (0.90 mmol/L) and/or a triglyceride level >250 mg/dL (2.82 mmol/L)

  Polycystic ovary syndrome or acanthosis nigricans

  History of vascular disease

Acute Complications of DM

Diabetic ketoacidosis (DKA) and hyperglycemic hyperosmolar state (HHS)

Manifestations of Diabetic Ketoacidosis

Symptoms	Physical findings
Nausea/vomiting	Tachycardia
Thirst/polyuria	Dehydration / hypotension
Abdominal pain	Tachypnea / Kussmaul respirations/respiratory distress
Shortness of breath	Abdominal tenderness (may resemble acute pancreatitis or surgical abdomen)
Precipitating events	Lethargy /obtundation / cerebral edema / possibly coma
Inadequate insulin administration	
Infection (pneumonia/UTI/gastroenteritis/sepsis)	
Infarction (cerebral, coronary, mesenteric, peripheral)	
Drugs (cocaine)	
Pregnancy	

Chronic Complications of Diabetes Mellitus

Microvascular
Eye disease
Retinopathy (nonproliferative/proliferative)
Macular edema
Neuropathy
Sensory and motor (mono- and polyneuropathy)
Autonomic
Nephropathy
Macrovascular
Coronary artery disease
Peripheral arterial disease
Cerebrovascular disease
Other
Gastrointestinal (gastroparesis, diarrhea)
Genitourinary (uropathy/sexual dysfunction)
Dermatologic
Infectious
Cataracts
Glaucoma
Periodontal disease

Prevention

Type 2 DM is preceded by a period of IGT, and a number of lifestyle modifications and pharmacologic agents prevent or delay the onset of DM. Intensive changes in lifestyle (diet and exercise for 30 min/day five times/week) in individuals with IGT prevents or delays the development of type 2 DM.

Individuals with a strong family history of type 2 DM and individuals with IFG or IGT should be strongly encouraged to maintain a normal BMI and engage in regular physical activity.

Patient Education About DM, Nutrition, and Exercise

Education topics important for optimal care include self-monitoring of blood glucose; urine ketone monitoring (type 1 DM); insulin administration; guidelines for diabetes management during illnesses; management of hypoglycemia; foot and skin care; diabetes management before, during, and after exercise; and risk factor–modifying activities.

Nutritional Recommendations for Adults with Diabetes

Fat
20–35% of total caloric intake
Saturated fat < 7% of total calories
<200 mg/day of dietary cholesterol
Two or more servings of fish/week provide -3 polyunsaturated fatty acids
Minimal trans fat consumption
Carbohydrate
45–65% of total caloric intake (low-carbohydrate diets are not recommended)
Amount and type of carbohydrate important[b]

Sucrose-containing foods may be consumed with adjustments in insulin dose
Protein
10–35% of total caloric intake (high-protein diets are not recommended)
Other components
Fiber-containing foods may reduce postprandial glucose excursions
Nonnutrient sweeteners

Exercise

Exercise has multiple positive benefits including cardiovascular risk reduction, reduced blood pressure, maintenance of muscle mass, reduction in body fat, and weight loss. For individuals with type 1 or type 2 DM, exercise is also useful for lowering plasma glucose (during and following exercise) and increasing insulin sensitivity. In patients with diabetes, the ADA recommends 150 min/week (distributed over at least 3 days) of aerobic physical activity

In patients with type 2 DM, the exercise regimen should also include resistance training.

Despite its benefits, exercise presents challenges for individuals with DM because they lack the normal glucoregulatory mechanisms (normally, insulin falls and glucagon rises during exercise). Skeletal muscle is a major site for metabolic fuel consumption in the resting state, and the increased muscle activity during vigorous, aerobic exercise greatly increases fuel requirements. Individuals with type 1 DM are prone to either hyperglycemia or hypoglycemia during exercise, depending on the preexercise plasma glucose, the circulating insulin level, and the level of exercise-induced catecholamines. If the insulin level is too low, the rise in catecholamines may increase the plasma glucose excessively, promote ketone body formation, and possibly lead to ketoacidosis. Conversely, if the circulating insulin level is excessive, this relative hyperinsulinemia may reduce

hepatic glucose production (decreased glycogenolysis, decreased gluconeogenesis) and increase glucose entry into muscle, leading to hypoglycemia.

To avoid exercise-related hyper- or hypoglycemia, individuals with type 1 DM should: (1) monitor blood glucose before, during, and after exercise; (2) delay exercise if blood glucose is >14 mmol/L (250 mg/dL) and ketones are present; (3) if the blood glucose is <5.6 mmol/L (100 mg/dL), ingest carbohydrate before exercising; (3) monitor glucose during exercise and ingest carbohydrate to prevent hypoglycemia; (4) decrease insulin doses (based on previous experience) before exercise and inject insulin into a nonexercising area; and (5) learn individual glucose responses to different types of exercise and increase food intake for up to 24 h after exercise, depending on intensity and duration of exercise. In individuals with type 2 DM, exercise-related hypoglycemia is less common but can occur in individuals taking either insulin or insulin secretagogues.

Because asymptomatic cardiovascular disease appears at a younger age in both type 1 and type 2 DM, formal exercise tolerance testing may be warranted in diabetic individuals with any of the following: age >35 years, diabetes duration >15 years (type 1 DM) or >10 years (type 2 DM), microvascular complications of DM (retinopathy, microalbuminuria, or nephropathy), PAD, other risk factors of CAD, or autonomic neuropathy. Untreated proliferative retinopathy is a relative contraindication to vigorous exercise, as this may lead to vitreous hemorrhage or retinal detachment.

HYPERTENSION

Hypertension is defined as systolic blood pressure >= 140mm Hg or diastolic blood pressure >= 90 mmHg

JNC-VII Classification of blood pressure

Blood Pressure Classification	**Systolic (mmHg)**	**Diastolic (mmHg)**
Normal	<120	And <80
Pre hypertension	120-139	Or 80-89
Stage 1Hypertension	140-159	Or 90-99
Stage 2 hypertension	>=160	Or >=100

Pathophysiology

B P = CO x TPR

Factors contributing to these hemodynamic alterations include:

- Susceptibilty to renal retention of excess sodium
- Sympathetic system hyperactivity
- Renin Angiotensin Activating System
- Endothelial cell dysfunction

Risk factors

- Genetics
- Behavioral
- Enviornmental factors

Causes

A) Essential hypertension (primary or idiopathic)

B) Secondary Causes

- Drug-induced or related causes
- Chronic kidney disease
- Primary aldosteronism
- Chronic steroid therapy and Cushing's syndrome
- Pheochromocytoma
- Coarctation of the aorta
- Obstructive sleep apnea

Other variants

- White coat hypertension- transient & persistent elevation in BP when it is taken in physician's office
- Isolated systolic hypertension- SBP>=140 & DBP>=90mm Hg

Why is hypertension a problem

- Increased risk for CVD
- Heart

• Left ventricular hypertrophy

• Heart failure

- Brain

• Stroke or transient ischemic attack

- Chronic kidney disease
- Peripheral arterial disease

- Retinopathy

Treatment

GOALS

- Treating SBP and DBP to targets that are <140/90 mmHg is associated with a decrease in complications.
- In patients with hypertension and Diabetesor renal disease, the BP goal is <130/80 mmHg.

Lifestyle Modifications

- Weight reduction - BMI < 25 kg/m2
- Adopt DASH eating plan -Diet rich in fruits, vegetables, and low-fat dairy products with reduced content of saturated and total fat
- Dietary sodium reduction - < 6 g NaCl/d
- Cessation of alcohol consumption

Medical Management

Anti hypertensive drugs

- Thiazide diuretics
- Potassium-sparing diuretics
- Aldosterone receptor blocker
- Barbiturates
- ACE inhibitors
- Angiotensin II antagonist

Pre exercise screening

Medical evaluation

- Blood pressure monitoring
- Individual & family history
- Physical examination
- Assessment of major risk factors
- Target organ damage
- CVD complications

Exercise testing

- For diagnosing and managing hypertension
- GXT is usually recommended for adults over 40 yrs of age
- Borderline resting BPs
- Normal response- progressive increase in SBP typically 8 to 12 mm Hg/MET, DBP usually decreases or remains unchanged
- Termination – 250/115mm Hg

Exercise prescription

- Frequency : endurance training frequency between 3 to 7 days/week
- Intensity : aerobic training :moderate-40 to 70 % VO_{2max}

  resistance training at 30 to 50 %of 1 RM
- Duration : 30 to 60 min continuous or intermittent exercise
- Type : primarily aerobic activity suplemented by resistance exercise

- Aerobic training- any activity that uses large muscle groups, e.g. walking, jogging, running, or cycling
- Resistance training-light weight lifting
- This leads to a decrease of 6 to 8 mm Hg in BP in SBP & DBP

Precautions

- Monitor BP before, during and after exercise
- Extend the warm up and cool down period to prevent hypotension upon cessation of activity
- Use borg scale
- Adequate fluid replacement
- For individual with documented episodes of ischemia during exercise, the exercise intensity should be set (≥10 beats·min-1) below the ischemic threshold.
- Avoid the Valsalva maneuver during resistance training

Hypertension in pregnancy

It is a chronic medical problem & is associated with increased maternal and perinatal mortality and morbidity

It can be:

- Gestational hypertension
- Pre-eclampsia
- Chronic hypertension

Treatment

ACE inhibitors & angiotensinII receptor blocker are moderately terotogenic & fetotoxic – Contraindicated

Labetolol- Ist choice

Methyldopa, nifidipine – Alternative

low-dose aspirin, reduce the risk of pre-eclampsia by 10% in high risk women

- Dietary supplementation
- Lifestyle modification.

OBESITY

Obesity is often defined simply as a condition of abnormal or excessive fat accumulation in adipose tissue, to the extent that health may be impaired or Obesity is defined as the abnormal growth of adipose tissue due to enlargement of fat cell size (hypertrophic obesity) or an increase in fat cell number (hyperplastic obesity) or a combination of both.

Obesity is a heterogeneous disorder; obese individuals vary in their body fat distribution, their metabolic profile and degree of associated cardiovascular and metabolic risk. Overweight and obesity are the fifth leading risk for global deaths.

The graded classification of overweight and obesity; (a) permits meaningful comparisons of weight status with in between population; (b) makes it possible to identify individuals and groups at increased risk of morbidity and mortality; (c) enables priorities to be identified for intervention at individuals and community levels; and (d) provides a firm basis for the evaluation of interventions.[9]

BODY MASS INDEX

Body Mass Index (BMI) provides the most useful, albeit crude, population level measure of obesity. It can be used to estimate the prevalence of obesity with a population and the risks associated with it. However, BMI does not account for the wide variation in body fat distribution, and may not corresponding to the same degree of fatness or associated health risk in different individuals and population.

BMI is a simple index of weight-for-height that is commonly used to classify underweight, overweight and obesity in adults. It is defined as the weight in kg by the square of the height in meters (kg/m^2).

The classification of overweight and obesity, according to BMI, is shown in table 1.1. Obesity is classified as a BMI >_30.0. The classification shown in table 1.1 is in agreement with that

recommended by WHO, but includes an additional subdivision at BMI 35.0-39.9 in recognition of the fact that management options for dealing with obesity differ above a BMI and mortality.

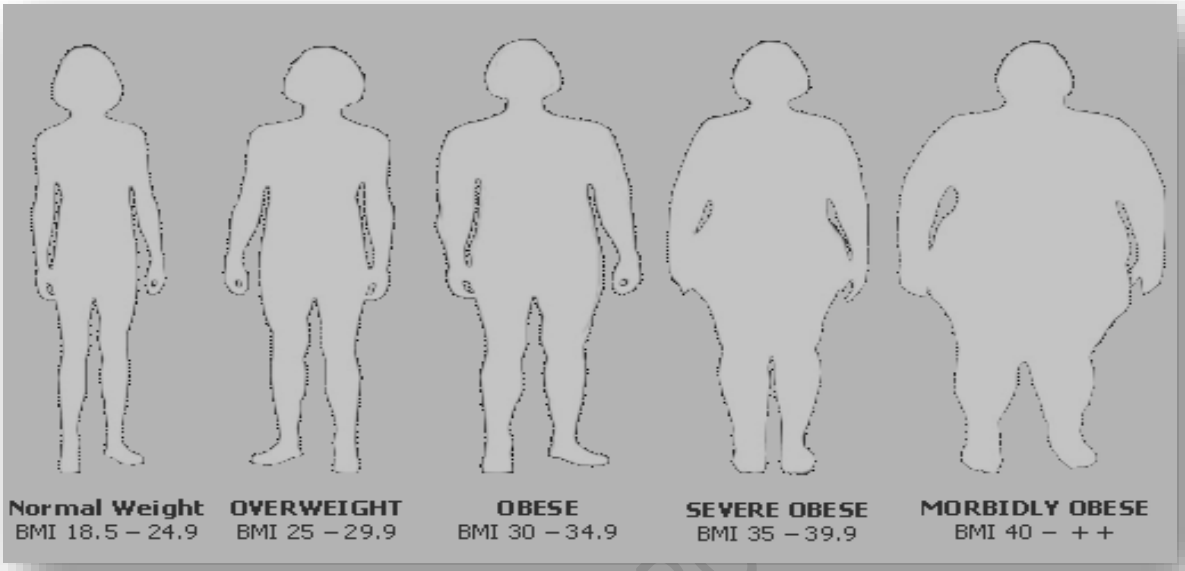


Figure 1: Classification of adults according to BMI

Table 1: Classification of adults according to BMI

Classification	**BMI**	**Risk of comorbidities**
Underweight	<18.5	Low(but risk of other clinical problems increased)
Normal range	18.5-24.99	Average
Overweight:	>25.00	
Preobese	25.0-29.99	Increased
Obese class 1	30.0-34.99	Moderate
Obese class 2	35.0-39.99	Severe
Obese class 3	>40.00	Very severe

WAIST CIRCUMFERENCE AND WAIST-HIP RATIO

Abdominal fat mass can vary dramatically within a narrow range of total body fat or BMI. Indeed, for any accumulation of total body fat, men have on average twice the amount of abdominal fat

than is generally found in premenopausal women. Other methods in addition to the measurement of BMI would therefore be valuable in identifying individuals at increased risk from obesity related illness due to abdominal fat accumulation.

A high WHR (WHR>0.90 in men and >0.85 in women) indicates abdominal fat accumulation.

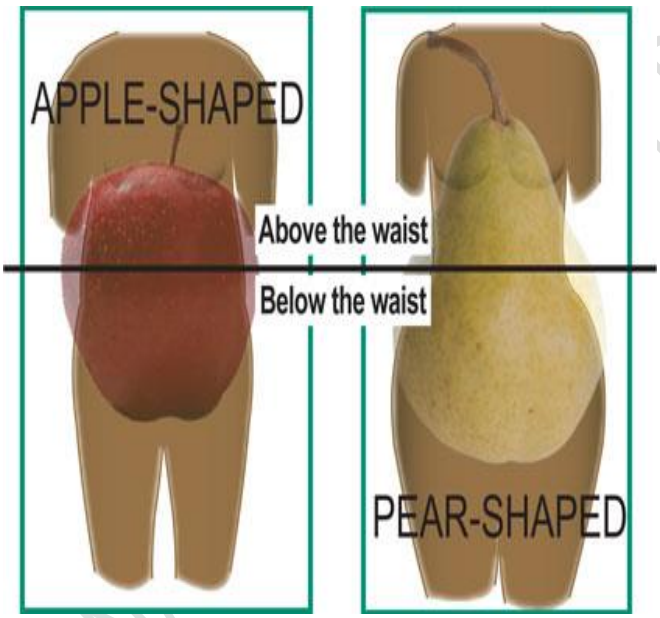
REGIONAL FAT DISTRIBUTION

CENTRAL OR ANDROID TYPE

In men twice as compared to women

PERIPHERAL OR GYNOID TYPE

More in women



PATHOPHYSIOLOGY

- Control mechanism affecting the hunger and satiety center- Leptin and Coupling Protein 2
- Dysfunctioning gene – ob gene, B- adrenoreceptor gene, D_2 dopamine receptor gene

EFFECTS OF OBESITY

- Each year, obesity contributes to an estimated 112,000 preventable deaths.
- Hypertension
- Coronary heart disease
- Hypercholestrelemia
- Type 2 Diabetes
- Stroke
- Gout, OA
- low back pain
- Obstructive sleep apnea
- Obesity hypoventilation syndrome
- ↑ complications during GA[2]
- Asthma
- Depression
- Social stigmatization
- ONCOLOGY: Breast, Ovarian, Esophageal, Colorectal, Liver, pancreatic, Gallbladder, Stomach, Endometrial, cervical, Prostate, Kidney
- Chronic renal failure

MANAGEENT

- Life style modification
- Behaviour modification
- Diet

✓ LCD [800-1500kcal]

✓ VLCD [≤ 800kcal]

- Pharmacologic agents

✓ Orlistat

✓ Sibutramine

Bariatric Surgery.

- BMI ≥40 kg/m^2
- BMI of 35 kg/m^2 - 40 kg/m^2 and other significant disease that could be improved if weight is lost

EXERCISE TESTING

- Exercise protocols that increase workloads at 0.5-1 MET each minute or ramp protocol.
- **AEROBIC TRAINING**
- Ideal exercise consists of continuous large muscle activity with high to moderate calorie cost such as circuit training, running, walking, skipping, stair climbing, cycling and swimming.
- 30–60 min·d-1 to total 150 min/week, progressing to 300 min/week of moderate physical activity; 150 min/week of vigorous physical activity; or an equivalent combination of both.
- **RESISTANCE TRAINING**
- This exercise mode burns substantial calories during a typical 30-60min workout.
- Resistance training of each major muscle group 2–3 d·wk-1 with at least 48 hours separating the exercise training sessions for the same muscle group.

RENAL FAILURE

Acute Renal Failure

ARF is characterized by a rapid decline in glomerular filtration rate (GFR) over hours to days. Retention of nitrogenous waste products, oliguria (urine output <400 mL/d contributing to

extracellular fluid overload), and electrolyte and acid-base abnormalities are frequent clinical features. ARF is usually asymptomatic and diagnosed when biochemical monitoring of hospitalized patients reveals a new increase in blood urea and serum creatinine concentrations.

For purposes of diagnosis and management, causes of ARF are generally divided into three major categories: (1) diseases that cause renal hypoperfusion, resulting in decreased function without frank parenchymal damage (prerenal ARF, or azotemia) (~55%); (2) diseases that directly involve the renal parenchyma (intrinsic ARF) (~40%); and (3) diseases associated with urinary tract obstruction (postrenal ARF) (~5%).

ARF is often considered to be reversible, although a return to baseline serum creatinine concentrations postinjury might not be sufficiently sensitive to detect clinically significant irreversible damage that may ultimately contribute to chronic kidney disease. ARF is associated with significant in-hospital morbidity and mortality, the latter in the range of 30–60%, depending on the clinical setting and presence or absence of nonrenal organ system failure

CHRONIC RENAL FAILURE

The term chronic renal failure applies to the process of continuing significant irreversible reduction in nephron number, and typically corresponds to CKD stages 3–5. The dispiriting term end-stage disease represents a stage of CKD where the accumulation of toxins, fluid, and electrolytes normally excreted by the kidneys results in the uremic syndrome. This syndrome leads to death unless the toxins are removed by renal replacement therapy, using dialysis or kidney transplantation

SYMPTOMS OF RENAL FAILURE

- Vomiting and/or diarrhea
- Shortness of breath

- Lethargy
- Confusion
- Blood in urine
- Abnormal heart rhythm
- Muscle cramps
- Swelling of legs, ankle, feet, face
- Darkening of skin

Medical Management

Control Hypertension

Restrict Dietary protein

Renal replacement Therapy

- Hemodialysis
- Peritoneal Dialysis
- Kidney Transplantation

Physiotherapy Management

Physiotherapy management has bee found to be effective in dialysis patients.

Warm period of light exercises and stretching

Aerobic exercises

Treadmill or arm ergometry for about 15-30 minutes

Resistance Exercises

For major muscle group of lower limb for about 10-20 minutes

Cool Down

Regular breathing exercises mainly diaphragmatic breathing.

Active range of motion exercises for bed ridden patients

Incentive Spirometry