**Diabetes mellitus:**

It is a state of chronic hyperglycemia due to an absolute/relative deficiency of

insulin.

**Definition:**

After at least 8 hrs. of fasting, the venous glucose is measured. Now the patient

is fed 75g glucose (300 kCal)—For children or anybody below 50 kg, the dose

is adjusted as 1.75 g/kg. After 2 hrs. of the meal, a postprandial venous glucose is

measured.

**Condition Venous plasma glucose:**

Diabetes mellitus Fasting > 126

Or postprandial > 200

Or random > 200

Impaired fasting glucose Fasting 110 –125

Impaired glucose tolerance PP 140 –199

**Types:**

1. **Type I**—Autoimmune, early onset, fatal without treatment, ketosis is common.

2. **Type II**—Insulin resistant, slow process, associated with the metabolic

syndrome, complicated by other diseases.

3. **Gestational** (diabetes in pregnancy)—Due to growth hormone, progesterone,

cortisol and prolactin excess.

4. **Maturity onset diabetes in the young** (MODY)—Genetic β cell malfunction.

5. **Lipotropic**—Genetic defect in insulin action.

6. Diseases of exocrine pancreas.

7. **Endocrine diseases**—Pheochromocytoma, Cushing syndrome, acromegaly,

hyperthyroidism.

8. **Drug-induced**—Steroids, thiazides, phenytoin, niacin.

9. **Congenital diseases**—Down’s, Klinefelter’s, Turner syndrome, Friedreich's

ataxia.

**Type I diabetes mellitus:**

Type I diabetes has a rapid, discernible onset, usually before 30 years of age. It

is usually autoimmune in origin, insulin dependent, and develops ketoacidosis if

untreated.

**Agent:**

1. Infections—Mumps, coxsackie, CMV, rubella

2. Nutrition—Early introduction of cow's milk

3. Autoimmunity—Against islet cells, glutamic acid decarboxylase

4. Genetic—Associated with HLA DR3 and DR4.

**Type II diabetes mellitus:**

**Agent:**

Insulin resistance seems to be the primary pathology. Up to a certain age, this

resistance is countered by increasing secretion, until β cell dysfunction sets in

and insulin production drops. Insulin resistance is brought on in the first place by

**rapid rise of blood glucose** after regular meals of refined sugars (burgers, colas,

candies) along with obesity.

**Host:**

1. Age—Type II diabetes usually comes to light only after 30, and many people

in the world live with it without being aware of it.

2. Sex—Type II diabetes is male dominant.

3. Genetic—Family history of Type II diabetes is a risk factor.

4. Central obesity—Waist circumference > 107 cm in men and > 88 cm in women

carries a high risk of Type II diabetes.

5. Habitual physical inactivity.

6. Maternal diabetes.

**Environment:**

1. **Diet**—Excess of saturated fatty acids, refined carbohydrates, inadequate

intake of dietary fibers and polyunsaturated fatty acids; children (and

teenagers) have acquired all the bad food habits of the west since the last two decades, and Type II diabetes is on the rise in children.

2. **Stress**—Physical (trauma, surgery) or mental stress may bring out latent

diabetes.

**Differences between Types I and II diabetes mellitus:**

Onset < 30 > 40

Ketosis Common Uncommon

Body weight Weight loss Occurs more in obese

Genetic 90% 50%

Circulating antiislet cell antibody + -

Associated autoimmune diseases + -

Treatment Insulin OAD and insulin

Insulin level ↓↓ ↓/ ↑

Insulin resistance Uncommon Usual

**Complications:**

1. Diabetes increases the risk of **heart disease and stroke**. 50% of people with

diabetes die of cardiovascular disease (primarily heart disease and stroke).

2. Combined with reduced blood flow, neuropathy in the feet increases the chance

of foot ulcers and eventual limb amputation.

3. **Diabetic retinopathy** is an important cause of blindness, and occurs as a result

of long-term accumulated damage to the small blood vessels in the retina.

After 15 years of diabetes, approximately 2% of people become blind, and

about 10% develop severe visual impairment.

4. Diabetes is among the leading causes of **kidney failure**. 10–20% of people

with diabetes die of kidney failure.

5. Diabetic neuropathy is damage to the nerves as a result of diabetes, and affects

up to 50% of people with diabetes. Although many different problems can occur

as a result of diabetic neuropathy, common symptoms are tingling, pain,

numbness, or weakness in the feet and hands.

The overall risk of dying among people with diabetes is at least *double* the risk

of their peers without diabetes.

**Primary prevention:**

**Population strategy:**

Type I diabetes is not practically preventable, except for genetic counseling. We

can only prevent Type II diabetes through a sea change in our lifestyle, habits and

activity. The 'dietary goals' as enunciated by WHO must be followed. But diabetes

cannot only be controlled by dieting. The cosmopolitan India must rethinks its

priorities of life, allow himself to slow down, if just a little, in the rat race, and

destress once every often.

**High risk strategy:**

Concentrate to eliminate risk factors in:

1. First degree relatives of a diabetes patient

2. Obese

3. Those with impaired glucose tolerance.

**Secondary prevention:**

**Screening:**

Target population for screening for diabetes:

1. People > 40

2. Family history

3. Obese

4. Women with babies > 4.5 kg at birth

5. Excess weight gain in pregnancy

6. Premature atherosclerosis.

**There are two methods for screening:**

1. **Glycosuria** after 2 hrs. Of a meal—This test is neither very sensitive nor

specific.

2. **Oral glucose tolerance test**.

**Oral glucose tolerance tests:**

The patient should take good carbohydrate meal three days prior to the test, and should

avoid all kind of drugs/exercise that will affect blood glucose level. Further, he should

consume 30–50 g of carbohydrate the prior evening. Next, he should do at least 8 hrs.

fasting before the test.

↓

Blood is collected at the fasting state from a vein and the glucose is estimated. Normally,

it should be 75–110 mg/dl

↓

The subject is fed 75 g pure (anhydrous) glucose in 250 –300 ml of water.

↓

Blood and urine are collected at ½ hr. intervals for next 2 ½ hrs. After 2 ½ hrs., blood glucose in a normal person should be 110–140 mg/dl.

**Treatment of diabetes:**

**Monitoring of therapy**

**•** Blood sugar

**•** Urine for sugar / protein / ketone

**•** Blood pressure.

**•** Visual acuity.

**•** Weight.

**•** Examination of vascular supply of feet (diabetic gangrene is very common in

lower limbs).

**•** Glycosylated hemoglobin—Indicator of long-term glycemic control.

Most of the monitoring can be done by the patients themselves through appropriate

self-care devices.

**Tertiary prevention:**

Save the 'diabetic foot', eyes and prevent other disabilities (ischemic heart disease,

renal failure). The patient can still be lead a good life.

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