

Diabetes Mellitus.

Introduction

- Heterogenous group of diseases.
- State of chronic hyperglycemia-due to various aetiologies-environmental and genetic.
- Main cause- defective production or action of insulin.
- Many complications-Cardiovascular, renal, neurological , ocular, and infections.

Classification

- 1. Diabetes Mellitus (DM).
 - a) IDDM-(Type 1).
 - b) NIDDM(Type-2).
 - c) Malnutrition related -Diabetes Mellitus.
 - d) Other types-(secondary to pancreatic, hormonal drug induced, genetic etc)
- 2. Impaired glucose tolerance.
- 3. Gestational Diabetes Mellitus.

- **IDDM**-Abrupt onset, seen in people < 30.
- Immune mediated - 90 % ,idiopathic-10%.
- Rate of destruction of beta pancreatic cell variable. Fail to respond to all insulin related stimuli.
- asso. with ketosis if untreated, exogenous insulin needed .

➤ **NIDDM**: more common, in elderly.

- Mild, slow to ketosis & compatible with long survival if treated adequately.
- Complicated by other disease processes.

SYNDROME X-Insulin resistance syndrome.

- In obese patients with type 2 DM, asso of hyperglycemia, hyperinsulinemia dyslipidemia & HT →CHD & stroke.
- Due to genetic defect, -insulin resistance exaggerated due to obesity.

Problem Statement

- Iceberg disease. More in industrialized countries. Nos. ↑ in India & China.
- 150 million cases globally. No. expected to double by 2025 (Prevalence-5.4%).
- Previously- Ds. of elderly but now age is ↓
- In developing countries- ↑ prevalence due to rapid urbanisation & industrialization.
- No. of cases more in urban than rural.
- 20% of current diabetic population in SEA.

INDIA

- Indians show ↑ susceptibility to DM.
- Rates of DM in migrants higher than those of local population.
- Prevalence-2.4 % in rural and 4-11% in urban areas.
- In coming decades, prevalence expected to rise.
- In Indians renal complications more common.

Natural history-Epidemiological determinants

- AGENT-Insulin deficiency- absolute in IDDM & partial in NIDDM.
- Due to:
- a)pancreatic disorders- neoplastic, inflammatory, etc.
 - b) Defects in insulin formation- in synthesis.
 - c) Destruction of beta cells-e.g-viral infecns,
 - d)Decreased insulin sensitivity- ↓ receptors.
 - e) Genetic defects.
 - f) Auto immunity.

HOST FACTORS

a) Age.

b) Sex: In Asia, males more than females.

c) Genetic factors: In NIDDM-90%.

-In IDDM-50%

d) Genetic Markers: IDDM-HLA-B8 & B15.

and HLA-DR3 and DR4. NIDDM-no asso.

e) Immune Mechanisms: evidence of cell mediated and humoral immunity.

f) Obesity

g) Maternal Diabetes

Environmental Risk Factors

- a) Sedentary lifestyle: lack of exercise alters interaction between insulin & its receptors.
- b) Diet: High fat intake-higher risk of impaired glucose tolerance.
- c) Dietary Fibre: high intake ↓ blood glucose levels.
- d) Malnutrition: partial failure of β cells.
- e) Alcohol: excessive intake ↑ risk .

Environmental Risk Factors

f) Viral infections: Rubella, mumps, human coxsackie virus-B4.

g) Chemical agents: Alloxan, streptozotocin, Valcor,-toxic effects on β cells.

h) Other factors: Occupation, marital status, religion, economic status, education, urbanisation etc.

Screening for diabetes

URINE EXAMINATION:

- 2 hrs after meals.
- All having glycosuria- diabetic unless proved otherwise by oral GTT.
- Glucosuria found in most severe cases .
- Test yields too many false negatives. Not a very sensitive test.
- not appropriate for epidemiological surveys or case- finding.

Screening (contd)

- **BLOOD SUGAR TESTING**: Standard oral glucose tolerance test.
- fasting, post prandial or random samples maybe used.
- For epidemiological purposes, 2 hr value after 75 gm oral glucose to be used.

		Glucose mg/dl	
	Venous	capillary	
Fasting value	>120	>120	
2 hrs after glucose load	>180	>200	
Impaired glucose tolerance-fasting	<120	<120	
2 hrs after glucose	120-180	140-200	

Target populations

- age group 40 or over,
- those with a family history of diabetes.
- Obese people
- Women having baby weighing more than 4.5 kg
- Women who gain excess weight in pregnancy.
- Patients with premature atherosclerosis.

Prevention and care

- PRIMARY PREVENTION:

A **POPULATION STRATEGY**: need for *primordial prevention*- prevention of emergence of risk factors.

B.HIGH RISK STRATEGY:

- Correction of sedentary lifestyle, eating habits, obesity.
- Avoid alcohol, OCs, smoking, HT, elevated cholesterol.

Secondary prevention

Adequate treatment based on:

- a) diet alone.
- b) Diet & oral antidiabetic drugs.
- c) diet and insulin.

Glycosylated haemoglobin: 6 monthly.

Provides index of long term glucose control.

- Self care.
- Home blood glucose monitoring.

Tertiary Prevention

- To organize specialized clinics (diabetic clinics) and units capable of providing diagnostic and management skills of high order.
- need to establish these clinics in small towns.
- tertiary care centres to do research.

National Programme

- For control of diabetes:

3 components:

- A. Health promotion for general population.*
- B. Disease prevention for the high risk group.*
- C. Assessment of prevalence of risk factors.*

THANK YOU!!!