India and China rank next only to China Globally.

Diabetes Prevalence

Where are we in India today with respect to Epidemiology and Screening

ABSTRACT

K Chathrapathy Muthy, Mohan

Optimizing Outcomes
Best Practices in Diabetes:

**Table 1: Indian Diabetes Risk Score**

<table>
<thead>
<tr>
<th>Score</th>
<th>Particulars</th>
</tr>
</thead>
<tbody>
<tr>
<td>20</td>
<td>Males</td>
</tr>
<tr>
<td>10</td>
<td>Females</td>
</tr>
<tr>
<td>0</td>
<td>Both sexes</td>
</tr>
</tbody>
</table>

- **How to identify the "High-risk" group?**

Box 4: list the criteria for screening Indian populations.

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**Examples of high-risk factors**

- People with Type 2 diabetes
- People with Type 1 diabetes
- People with gestational diabetes
- People with a family history of diabetes
- People with high blood pressure
- People with high cholesterol levels
- People with a history of heart disease
- People with a personal history of diabetes
- People with a history of gestational diabetes
- People with a history of Type 1 diabetes
- People with a history of Type 2 diabetes

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**Who to screen?**

- People with a high risk of developing diabetes
- People with a family history of diabetes
- People with a history of gestational diabetes
- People with a history of Type 1 diabetes
- People with a history of Type 2 diabetes

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**New elevation of our country.**

- Children of prediagnosis are at increased risk of diabetes
- Diseases are teaching and early stages in asymptomatic
Prevention is key: The earlier diabetes prevention begins, the more effective it is. Lifestyle changes such as healthy eating and regular exercise can help to reduce the risk of developing diabetes.

Evidence that diabetes can be prevented (Fig. 4.1)

The number of diabetes cases in children 10-14 (childhood diabetes) is increasing, and prediabetes (pre-diabetes) is becoming more common in children due to obesity, physical inactivity, and poor diet. The American Diabetes Association (ADA) has developed guidelines to help identify and manage prediabetes.

Pathogenesis of diabetes

The发病过程 involves a complex interplay of genetic, environmental, and lifestyle factors. Categorization of diabetes is primarily based on age of onset: Type 1 diabetes occurs before age 30, Type 2 diabetes occurs after age 30, and gestational diabetes occurs during pregnancy.

Diagnosis and classification of diabetes

Table 4.2 shows the criteria for diagnosis of diabetes and prediabetes.

Diabetes prevention program (DP) components

The diabetes prevention program (DP) includes lifestyle changes such as improved diet, weight loss, physical activity, and medication. The program is designed to help prevent or delay the onset of Type 2 diabetes in people who are at high risk.

Can diabetes be reversed?

Yes, diabetes can be reversed in some cases. This is especially true in cases of prediabetes where lifestyle changes can help to normalize blood sugar levels.

Fig. 4.1: Levels of diabetes prevention, and primary, secondary, and tertiary prevention of diabetes.

Fig. 4.2: Prevention of diabetes progression with improved prevention.
**Classification of Diabetes**

- **Type 1 Diabetes**
  - Onset usually before age 30
  - Usually severe
  - Insulin dependence
  - Genetic predisposition
  - Autoimmune destruction of β-cells
  - Other specific types of diabetes mellitus
  - Congenital diabetes mellitus
  - Type 2
  - Type 1

**Value of 1-hour Value in OGTT**

- **OGTT (oral glucose tolerance test)**
  - Measurement of plasma glucose levels after ingestion of a glucose solution
  - 2-hour post-glucose value (mg/dL) vs. 1-hour post-glucose value (mg/dL)
  - Values above 140 mg/dL indicate abnormal glucose tolerance

**Table 4.2: Criteria for Diabetes and Prediabetes**

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Normoglycemia</th>
<th>Impaired Glucose Tolerance</th>
<th>Prediabetes</th>
<th>Diabetes</th>
</tr>
</thead>
<tbody>
<tr>
<td>2-hour post-glucose glucose (mg/dL)</td>
<td>&lt;140</td>
<td>&gt;140</td>
<td>&gt;140</td>
<td>&gt;200</td>
</tr>
<tr>
<td>Fasting plasma glucose (mg/dL)</td>
<td>&lt;100</td>
<td>&lt;140</td>
<td>&gt;140</td>
<td>&gt;200</td>
</tr>
<tr>
<td>2-hour post-meal glucose (mg/dL)</td>
<td>&lt;140</td>
<td>&gt;140</td>
<td>&gt;140</td>
<td>&gt;200</td>
</tr>
</tbody>
</table>

**Table 4.3: Diagnostic Sensitivity and Specificity of OGTT**

<table>
<thead>
<tr>
<th>Sensitivity</th>
<th>Specificity</th>
</tr>
</thead>
<tbody>
<tr>
<td>100%</td>
<td>99%</td>
</tr>
</tbody>
</table>

**Graphic:**

- Description of glucose levels and their implications for diagnosis.

**Text:**

- Detailed explanation of the importance of OGTT in evaluating glucose levels.
- Discussion on the criteria for normoglycemia, impaired glucose tolerance, prediabetes, and diabetes.
- Emphasis on the 1-hour value in OGTT for early detection of potential issues.
Maturity Onset Diabetes of the Young (MODY) Criteria for

1. Genetic testing for a Family History of MODY is recommended for all MODY.
2. The diagnosis of MODY is based on the following criteria:
   - Phenylalanine at birth is >5 mg/dL
   - Birth weight is <15th percentile
   - Presence of at least one first-degree relative with MODY
   - Absence of any other known diabetes predisposition syndromes

Gestational Diabetes Mellitus

- Chort and decaturization (CDI) antibodies are present
- Daily insulin requirement is >50 units
- Any abnormality detected by ultrasound
- Age >35 years
- Sustained features of LADA

How to Diagnose LADA from Type 1 Diabetic Patients

1. Presence of intra-islet cell antibodies
2. Presence of anti-GAD antibodies
3. Presence of anti-insulin antibodies
4. Presence of anti-glutamic acid decarboxylase antibodies
5. Presence of anti-IA-2 antibodies

Table 4.1 Types of maturity onset diabetes of the young (MODY).

<table>
<thead>
<tr>
<th>MODY Type</th>
<th>Clinical Features</th>
</tr>
</thead>
<tbody>
<tr>
<td>MODY 1</td>
<td>Fasting hyperglycemia</td>
</tr>
<tr>
<td>MODY 2</td>
<td>Fasting hyperinsulinemia</td>
</tr>
<tr>
<td>MODY 3</td>
<td>Familial overlap with MODY</td>
</tr>
<tr>
<td>MODY 4</td>
<td>Autosomal dominant inheritance</td>
</tr>
<tr>
<td>MODY 5</td>
<td>Autosomal recessive inheritance</td>
</tr>
</tbody>
</table>

Table 4.2: Diagnosis of gestational diabetes mellitus (GDM) using International Association of Diabetes and Pregnancy Study Groups (IADPSG) criteria.

<table>
<thead>
<tr>
<th>GDM Diagnosis</th>
<th>Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>2-hour post-glucose</td>
<td>&gt;155 mg/dL</td>
</tr>
<tr>
<td>1-hour post-glucose</td>
<td>&gt;140 mg/dL</td>
</tr>
<tr>
<td>30-minute post-glucose</td>
<td>&gt;95 mg/dL</td>
</tr>
</tbody>
</table>

Table 4.3: Association of diabetes and pregnancy study groups (IADPSG) criteria.
Lifestyle Modification

- Weight loss should be achieved with diet and physical activity.
- Regular physical activity should be encouraged.
- A regular meal plan should be promoted.
- Physical activity.
- Medication of choice.
- Medication therapy (Fig. 4.3).

TREATMENT OF DIABETES

Approach to Diagnosis of Types of Diabetes in Youth

2. Type 2 diabetes.
3. An ideal meal plan.
4. Effect of medication on glycemic control.
5. C-peptide levels.
6. Insulin resistance.
7. C-reactive protein.
8. Fasting plasma glucose.
10. HbA1c levels.

Flowchart 4.1: Algorithm for differential diagnosis of diabetes in youth.
### Table 4.6: Pharmacological treatment which includes the following classes of drugs.\(^{16}\)

<table>
<thead>
<tr>
<th>Class</th>
<th>Compounds</th>
<th>Primary physiological actions</th>
<th>Advantages</th>
<th>Disadvantages</th>
<th>Cost</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Biguanides</strong></td>
<td>Metformin</td>
<td>Activates AMP-kinase</td>
<td>Rare hypoglycemia</td>
<td>GI side effects</td>
<td>Low</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hepatic glucose production</td>
<td>↓ CVD events (UKPDS)</td>
<td>Relatively higher A1c efficacy</td>
<td></td>
</tr>
<tr>
<td><strong>Sulfonylureas (SU)</strong></td>
<td>Glyburide</td>
<td>KATP channels antagonists</td>
<td>↑ insulin secretion</td>
<td>Hypoglycemia</td>
<td>Low</td>
</tr>
<tr>
<td></td>
<td>Glipizide</td>
<td></td>
<td>↓ microvascular risk (UKPDS)</td>
<td>↑ weight</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Gliclazide</td>
<td></td>
<td>Relatively higher A1c efficacy</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Meglitinides (glitazones)</strong></td>
<td>Repaglinide</td>
<td>KATP channels antagonists (non-SU)</td>
<td>↑ insulin secretion</td>
<td>Hypoglycemia</td>
<td>High</td>
</tr>
<tr>
<td></td>
<td>Nateglinide</td>
<td></td>
<td>↓ postprandial glucose excursions</td>
<td>↑ weight</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Dosing flexibility</td>
<td>Frequent dosing schedule</td>
<td></td>
</tr>
<tr>
<td><strong>Thiazolidinediones (TZD)</strong></td>
<td>Pioglitazone</td>
<td>PPAR-γ activator</td>
<td>↑ insulin sensitivity</td>
<td>↑ weight edema/heart failure</td>
<td>Low</td>
</tr>
<tr>
<td></td>
<td>Rosiglitazone</td>
<td></td>
<td>Rare hypoglycemia</td>
<td>Bone fractures</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Relatively higher A1c efficacy</td>
<td>Bladder cancer</td>
<td></td>
</tr>
<tr>
<td><strong>α-glucosidase inhibitors</strong></td>
<td>Acarbose</td>
<td>Intestinal α-glucosidase</td>
<td>Rare hypoglycemia</td>
<td>Gastrointestinal side effects (flatulence, diarrhea)</td>
<td>Low to moderate</td>
</tr>
<tr>
<td></td>
<td>Voglibose</td>
<td>inhibition and Slows intestinal carbohydrate digestion/absorption</td>
<td></td>
<td>Generally modest A1c efficacy</td>
<td></td>
</tr>
<tr>
<td><strong>Dipeptidyl peptidase 4 inhibitors (DPP4)</strong></td>
<td>Sitagliptin</td>
<td>Increased postprandial incretin (GLP-1, GIP) concentrations</td>
<td>Rare hypoglycemia</td>
<td>Angioedema/urticarial and other immune-mediated dermatological effects</td>
<td>High</td>
</tr>
<tr>
<td></td>
<td>Saxagliptin</td>
<td></td>
<td>Well-tolerated</td>
<td>Acute pancreatitis</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Linagliptin</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Alorglaptin</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Contd...**

<table>
<thead>
<tr>
<th>Class</th>
<th>Compounds</th>
<th>Primary physiological actions</th>
<th>Advantages</th>
<th>Disadvantages</th>
<th>Cost</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sodium glucose co-transporter 2 inhibitors (SGLT2)</strong></td>
<td>Empagliflozin</td>
<td>Inhibit SGLT2 in the proximal nephron Block glucose reabsorption by the kidney increasing glucosuria</td>
<td>Rare hypoglycemia</td>
<td>Genitourinary infections Polyuria, volume depletion/hypotension/dizziness</td>
<td>High</td>
</tr>
<tr>
<td></td>
<td>Canagliflozin</td>
<td></td>
<td></td>
<td>↑ LDL-C,</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Dapagliflozin</td>
<td></td>
<td></td>
<td>↑ Creatinine (transient) DKA Urinary tract infections leading to urosepsis, pyleonephritis</td>
<td></td>
</tr>
<tr>
<td><strong>Glucagon-like polypeptide 1 (GLP1) analogs</strong></td>
<td>Exenatide</td>
<td>Activate GL-1 receptors insulin secretion (glucose-dependent)</td>
<td>Rare hypoglycemia</td>
<td>Gastrointestinal side effects (nausea/vomiting/diarrhea)</td>
<td>High</td>
</tr>
<tr>
<td></td>
<td>Exenatide-extended release</td>
<td></td>
<td></td>
<td>↑ Heart rate</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Liraglutide</td>
<td>↓ glucagon secretion (glucose-dependent)</td>
<td></td>
<td>Acute pancreatitis</td>
<td>Injectable</td>
</tr>
<tr>
<td></td>
<td>Albiglutide Lixisenatide</td>
<td>Slow gastric emptying</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Dulaglutide</td>
<td>↑ Satety</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Insulin</strong></td>
<td>Rapid-acting analogs:</td>
<td>Activate insulin receptors</td>
<td>Nearly universal response Theoretically unlimited efficacy</td>
<td>Hypoglycemia, weight gain Patient and provider reluctance</td>
<td>High</td>
</tr>
<tr>
<td></td>
<td>• Lispro</td>
<td>↑ glucose disposal</td>
<td></td>
<td>Injection site lipohypertrophy</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Aspart</td>
<td>↓ hepatic glucose production</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Glulisine</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Long-acting analogs:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Gliargine</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Detemir</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Degludec</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

(CVD: Cardiovascular; GI: Gastrointestinal; LDL-C: Low-density lipoprotein cholesterol; DKA: Diabetic ketoacidosis; SGLT2: Sodium glucose co-transporter 2).
TYPE 2 DIABETES

Indian Consensus Guidelines for Treatment of Type 2 Diabetes

Guidelines for Stage-Wise Management

The following points are to be taken into consideration while choosing the right option among the different classes of drugs:

- Age
- Side effect profile
- Cardiovascular risk
- Cost
- Weight

1. Oral hypoglycemic agents (Table 4.1)

Hypoglycemia: Hypoglycemia can occur after meals with certain drugs, especially when used in combination with other drugs. It can be severe and require immediate medical attention.

- Sulfonylureas
- Metformin
- Thiazolidinediones
- DPP-4 inhibitors
- GLP-1 receptor agonists

2. Insulin therapy

- Basal insulin
- Multiple daily injections
- Continuous subcutaneous insulin infusion (CSII)
- Insulin pump

3. Lifestyle modifications

- Nutrition counseling
- Physical activity
- Weight loss
- Stress management

4. Other therapies

- Pancreatic islet cell transplantation
- Artificial pancreas
- Stem cell therapy
- Gene therapy

Fig. 4.4: A model of the American Diabetes Association (ADA) guidelines for type 2 diabetes 2017

Fig. 4.4: Achievement of glycemic control by different strategies

- Basal insulin + CSII
- Multiple daily injections (MDI)
- Insulin pump

Fig. 4.4: American Diabetes Association (ADA) guidelines 2016

Fig. 4.4: Achievingglycemic control by different strategies

- Basal insulin + CSII
- Multiple daily injections (MDI)
- Insulin pump
Initiation and Titration of Premixed Insulin

- Higher carbohydrate meals
- Appropriate in type 2 and insulin-dependent patients
- Usually by 0.25 units/kg or 5 units/day
- Consider adding short-acting insulin

Guidelines for Use of Insulin

<table>
<thead>
<tr>
<th>Insulin</th>
<th>Initial Dose</th>
<th>Increasing Dose</th>
<th>Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Melli</td>
<td>0.5 units/kg</td>
<td>0.5 units/kg</td>
<td>OADs + NGBs</td>
</tr>
<tr>
<td>NPH</td>
<td>0.5 units/kg</td>
<td>0.5 units/kg</td>
<td>OADs + NGBs</td>
</tr>
<tr>
<td>Insulin</td>
<td>0.5 units/kg</td>
<td>0.5 units/kg</td>
<td>OADs + NGBs</td>
</tr>
<tr>
<td>GLP-1</td>
<td>0.5 units/kg</td>
<td>0.5 units/kg</td>
<td>OADs + NGBs</td>
</tr>
</tbody>
</table>

For management of diabetes in primary care, the ADA guidelines for insulin initiation and continuation guidelines are followed.

Program X outlines the ADA guidelines for insulin initiation of therapy in order to have a good compliance with insulin therapy. Insulin use in some cases of diabetes is inevitable.
Chronic Kidney Disease

Insulin should be properly transported to preserve its efficacy.

Storage and Cold Chain

- The disposal of insulin needs and ancillaries should be ensured.
- Needle should not be re-used. Insulin pens, cartridges, and vials should be stored at least one year before the expiry. If ILT is detected, it should be disposed of.
- Information sheets should be inspected and performed by pharmacies and other professionals.
- Insulin should be distributed and performed by pharmacies and other professionals.
- Insulin should be inspected and dispatched by pharmacies and other professionals.
- The insulin should be cleaned and disposed of properly.
- Correct indication: Indications for the use of insulin, treatment of diabetes.

Special Considerations

Insulin

If insulin is prescribed, it should be used exclusively at the time of diagnosis and in the following cases:

- In patients with diabetes.
- In patients with insulin resistance.
- In patients with hyperglycemia.
- In patients with uremic patients.
- In patients with severe hyperglycemia.
- In patients with severe hypoglycemia.
- In patients with severe hyperglycemia.
- In patients with severe hypoglycemia.
- In patients with severe hyperglycemia.
- In patients with severe hypoglycemia.
- In patients with severe hyperglycemia.
**Follow-up of Patients (Tables 4.9 and 4.10)**

In addition to the other complications, other complications include:
- Hypertension
- Hyperlipidemia
- Coronary artery disease
- Peripheral arterial disease
- Stroke
- Nephropathy
- Retinopathy
- Microvascular disease

<table>
<thead>
<tr>
<th>Table 4.10: Complications of Diabetes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nephropathy</td>
</tr>
<tr>
<td>Diabetic neuropathy</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Table 4.9: Frequency and Duration of Follow-up Visit</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frequency</td>
</tr>
<tr>
<td>Evaluation</td>
</tr>
<tr>
<td>One in 3 months</td>
</tr>
<tr>
<td>Hemoglobin A1C (HbA1c)</td>
</tr>
<tr>
<td>One in 3 months</td>
</tr>
<tr>
<td>Glycosylated hemoglobin (HbA1c)</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Investigation</td>
</tr>
</tbody>
</table>

**Figure 4.4: Ambulatory Glucose Profile (AGP)**

- Nephropathy
- Retinopathy
- Hypertension
- Hyperlipidemia
- Diabetic neuropathy
- Coronary artery disease
- Peripheral arterial disease
- Stroke
- Nephropathy
- Retinopathy
- Macular edema

**Monitoring of Diabetes**

- **HbA1c (%)**
  - Less than 8%
  - 8% or higher

<table>
<thead>
<tr>
<th>Table 4.5: Glycemic Targets</th>
</tr>
</thead>
<tbody>
<tr>
<td>HbA1c (%)</td>
</tr>
<tr>
<td>--------------------------------------------------</td>
</tr>
<tr>
<td>6.5-7.5</td>
</tr>
<tr>
<td>8.0-10.0</td>
</tr>
<tr>
<td>10.0-110</td>
</tr>
<tr>
<td>PPG (mg/dl)</td>
</tr>
<tr>
<td>PPG (mg/dl)</td>
</tr>
</tbody>
</table>

**Note:** These targets are not applicable to pregnant women.
REFERENCES

multidisciplinary approach to achieve "best practices" in diabetes.


MULTI-DISCIPLINARY APPROACH TO ACHIEVE "BEST PRACTICES" IN DIABETES:


CONCLUSION