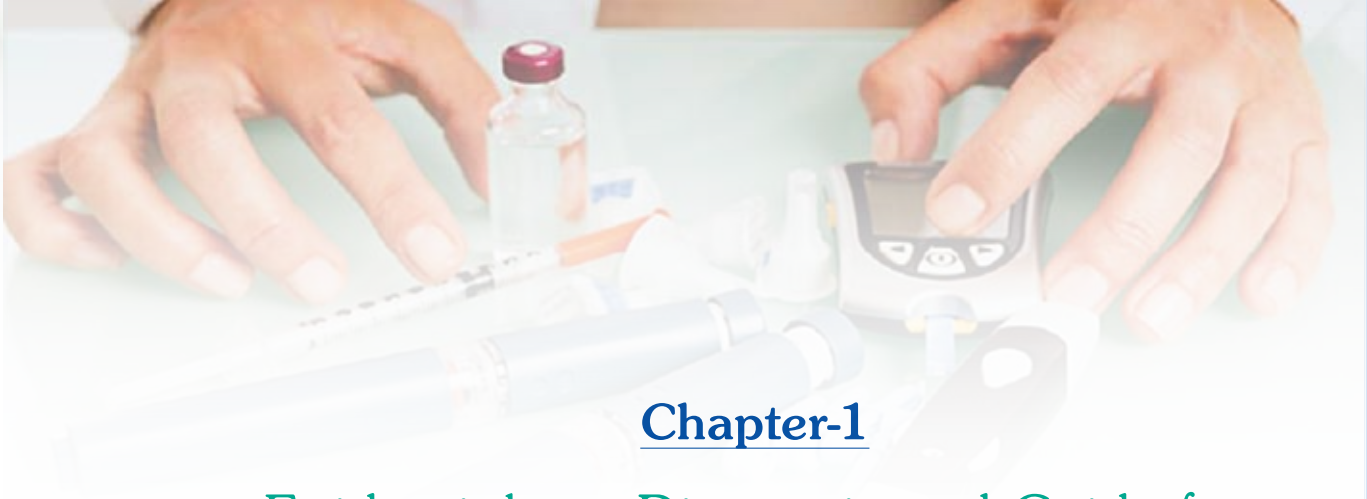
# Guidelines for Management of Type 1 Diabetes

# Chapter-1

# Epidemiology, Diagnosis and Guide for Differential Diagnosis

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## Definition and diagnosis

Type 1 Diabetes mellitus (T1DM) is an autoimmune disease characterized by insulin deficiency and hyperglycemia in people with underlying genetic susceptibility[1,2]. The diagnosis is established based on the tests, fasting plasma glucose (FPG), oral glucose tolerance test (OGTT), glycated hemoglobin (HbA1c) or random plasma glucose (along with characteristic signs and symptoms of hyperglycemia), using cut-offs recommended by American Diabetes Association[1,3].

## Epidemiology of T1DM

There are nearly 1.1 million people below 20 years of age estimated to be affected by T1DM globally and 0.13 million are diagnosed with the disease each year, as per the International Diabetes Federation (IDF) Atlas, 9$^{th}$ edition[4]. A recent study found an incidence of 4.9 cases/100000/year in India, which is much lower than the incidence of 21.2 cases/100,000/year observed in the SEARCH registry of USA[5]. The peak incidence for T1DM is seen between 10-14 years of age, though it can affect an individual at any age[6].

## Pathogenesis of T1DM

Genetic factors play a significant role in the etiology of T1DM. The concordance rate in monozygotic twins is around 30%[7,8]. The risk of T1DM is 3%, 5%, and 8%, respectively, when mother, father, and sibling have T1DM[9]. The HLA haplotypes with a strong association with T1DM are DR3-DQ2 and DR4-DQ8, present in 30-40% patients with T1DM compared with 2.4% in the general population[10,11]. There are more than 50 non-HLA loci, which increases the susceptibility for T1DM, though the association is weaker than for the HLA region[1,6,7]. The identified genes play a significant role in immune function or pancreatic β-cell function. Though the environmental risk factors have been proposed in the etiopathogenesis of T1DM, understanding is still low. The factors with the most substantial evidence include enteroviral infections, older maternal age, rapid weight gain in early life, and β-cell stress[12].

## Natural history of disease evolution

In a genetically susceptible individual, T1DM is preceded by the autoimmunity phase without clinical diabetes. A joint statement from professional organizations proposed a staging classification system that defines the early stages of T1DM with prognostic significance[13]. The recommended classification is mainly for research rather than for clinical practice.

### *Staging*

The various stages are defined as follows[13]:

i) Stage 1 (pre-symptomatic): Presence of β-cell autoimmunity with normoglycemia. This stage is evident by the presence of two or more islet cell autoantibodies.

ii) Stage 2 (pre-symptomatic): β-cell autoimmunity is present along with dysglycemia.

iii) Stage 3 is the onset of symptomatic disease.

A study found that in children with two or more islet autoantibodies, 43.5%, 69.7%, and 84.2% developed symptomatic type 1 diabetes at 5, 10, and 15 years of follow- up[14]. T1DM occurred in 12.7%, 61.6%, and 79.1% of children with a single, two, and three autoantibodies, respectively, after 15 years of follow-up. The progression was faster in those who developed autoantibodies at less than three years of age and had HLA DR3- DQ2/DR4-DQ8 genotype[14]. The magnitude of the autoimmunity titer, the autoantibody's affinity, and the type of autoantibody also determine the rates of progression[6]. Higher titers of insulin and IA-2 autoantibodies are associated with earlier onset of disease. The presence of IA-2 or ZnT8 autoantibodies is associated with faster progression to T1DM[15]. The two and five year risk for T1DM is approximately 60% and 75%, respectively, for those who have reached stage 2 (prediabetes)[13]. The β-cell function is reduced at presentation and shows partial recovery with control of hyperglycemia. Patients may go in a so-called honeymoon period after diagnosis and may have minimal or no exogenous insulin requirement. However, residual cells are subsequently lost. The beta-cell damage may not be complete in certain individuals. With sensitive C-peptide measurements, 30–80% of people with long-term T1DM are insulin micro-secretors[1].

## Classification of diabetes

World Health Organization came out with a recent classification system in 2019. The WHO 2019 classification system has removed the subtypes for T1DM and T2DM. A new entity called 'hybrid forms of diabetes' is introduced, including a) slowly evolving immune-mediated diabetes of adults and b) ketosis-prone type 2 diabetes. WHO has also introduced a category called 'unclassified diabetes' which is a temporary label, when it is difficult to make a precise diagnosis regarding the type of diabetes, especially close to the time of diagnosis. 'Diabetes mellitus in Pregnancy' is included as a distinct entity, and 'Gestational Diabetes Mellitus' is placed under 'Hyperglycemia first detected during pregnancy'[16].

## Spectrum of T1DM

T1DM is associated with autoantibodies in 70-90% of the individuals with the disease. In the classification system of the American Diabetes Association, this entity is called immune-mediated T1DM. T1DM without evidence of autoimmunity is known as idiopathic T1DM[3]. In 2019, WHO removed these subtypes, as insulin need is determined clinically rather than based on autoimmunity.

T1DM can have variable presentations[17-21]. Children often present acutely, with severe symptoms of polyuria, polydipsia, and ketonemia, and approximately a third of them present with diabetic ketoacidosis (DKA). Older children may have a hyperacute presentation so-called fulminant T1DM. In them, autoimmunity is absent, and there is a complete loss of beta-cell function. Japan and Korea have predominantly reported cases of fulminant T1DM. Adults usually present with a more gradual onset, and the initial clinical presentation may

appear consistent with type 2 diabetes mellitus (T2DM). However, they may have an acute presentation similar to children. The reasons for different clinical presentations seen in children and adults may be due to the more severe nature of the disease in childhood T1DM and less frequent blood testing for unrelated indications. WHO has renamed 'Latent autoimmune diabetes in adults' (LADA) as 'slowly evolving immune-mediated diabetes of adults' in their new classification. It is characterized by presence of islet antibody (predominantly GAD65), in individual older than 35 years at diagnosis, and with no insulin requirement for the first 6-12 months after diagnosis. The individuals retain greater beta-cell function and have higher chances of having metabolic syndrome features than individuals with T1DM[16,22]. We have tabulated the salient differences between T1DM and slowly evolving immune-mediated diabetes of adults in Table I.

**Table I: Comparison of features between slowly evolving immune-mediated diabetes of adults and Type 1 Diabetes mellitus**

| Characteristic | T1DM | Slowly evolving immune- mediated diabetes of adults |
|---|---|---|
| Age at diagnosis | Childhood to adolescence, rarely in adulthood | > 35 years |
| Body mass index | Underweight to normal | Normal to overweight |
| Onset | Acute | Rarely acute |
| Autoimmunity | Severely increased | Increased |
| Ketosis | Frequent | Rare |
| Insulin dependence | At onset | > 6 months after onset |
| Insulin resistance | No change | Increased or no change |

Abbreviation: T1DM-Type 1 diabetes mellitus

## Differentiation from other types of diabetes

The age group of 20-40 years has the highest heterogeneity and is at risk of misclassification[23]. In this regard, the category of 'unclassified diabetes' in the new WHO 2019 classification is a significant addition. The precise label for a type of diabetes can be given at a later stage, once the diagnosis is clear. T2DM and monogenic diabetes are two common entities from which T1DM needs to be differentiated. The other forms of diabetes seen in the younger age group and from which T1DM needs to be differentiated upon include neonatal diabetes, mitochondrial diabetes, and fibrocalculous pancreatic diabetes (FCPD). There is a vast list of conditions that can give rise to or are associated with diabetes. Diabetes can be seen in context to diseases of the exocrine pancreas, endocrinopathies, infections, and drugs. It may also result in the context of other genetic syndromes or due to genetic defects in insulin action. Given their rarity in youth, these conditions are not discussed in detail but are essential to recognize, as they may be the first clue to underlying syndrome or disease[3].

## Differentiation from T2DM

T2DM has emerged as a new type of diabetes in childhood due to an increase in obesity[24]. The incidence and prevalence of youth onset T2DM are increasing. In the Registry of people with diabetes with young age at onset (YDR) from India, 25.3% of individuals developing diabetes under the age of 25 years had a diagnosis of T2DM[24]. Dabelea et al. provided an etiologic approach for the classification of recently diagnosed diabetes

in youth less than 20 years[25]. They described four categories using autoimmunity and insulin sensitivity (IS). Most individuals (54.5%) had autoimmune markers and were insulin sensitive. This group had characteristics of T1DM. There were 15.9% of individuals who did not have autoimmunity and were insulin resistant. This group had characteristics of T2DM. The third group (19.5%), who had autoimmunity and were insulin resistant had similar prevalence and titers of diabetes autoantibodies, and distribution of HLA risk genotypes to those in the autoimmune plus insulin sensitive group. This suggests that it includes individuals with T1DM who are obese. The last group classified as nonautoimmune plus IS (10.1%) may include those with monogenic diabetes. Nearly 50% of insulin-sensitive individuals had diabetes onset before the age of ten years, compared to 5 to 11% for insulin-resistant individuals. The mean age of diabetes onset reported in YDR is 10.5 years for T1DM and 16.5 of T2DM. There were 15.7%, 30.7%, 32.7% and 20.9% individuals with T1DM in age groups 0-4, 5-9, 10-14 and 15-19 years. The corresponding figures for T2DM were zero, 4.4%, 25.6%, and 70%. The individuals with T1DM who were overweight/obese in YDR were 11.9% compared to 58.2% with T2DM[26]. DKA at diagnosis was present in 28.7% of individuals with T1DM and 6.6% with T2DM[27]. There were 30.6% of individuals with T2DM who were on insulin alone or in combination with oral glucose-lowering drugs in YDR[28]. These data suggest that discriminating T1DM from T2DM on isolated features based on the age of onset, overweight/obesity, autoantibody positivity, insulin requirement, or presentation with ketoacidosis is difficult[23]. The initial differentiation between T1DM and T2DM requires considering multiple factors rather than relying on a single parameter. It is relatively easy to make a diagnosis of T1DM for a patient who is younger than ten years, is normal weight, has evidence of autoimmunity, and presents with DKA[23]. Key features are tabulated that may help in differentiating between T1DM and T2DM in Table II.

## Differentiation from Maturity Onset Diabetes in the Young (MODY)

MODY is due to a single gene defect resulting in compromised insulin secretion[29-31]. The mutations in the genes encoding the enzyme glucokinase (GCK), the nuclear transcription factors hepatocyte nuclear factor 1α (HNF1A), and hepatocyte nuclear factor 4α (HNF4A) are the most common causes of MODY30. There are more than ten different types of MODY described so far. It should be considered in a patient without obesity, who develops diabetes before 25 years of age and has two or three generations of diabetes in the family[23,30,31]. MODY's other features are insulin independence (although insulin may be needed for optimal control), absence of features of insulin resistance, and absence of β cell autoimmunity.

Patients with MODY may be misdiagnosed as T1DM or T2DM. The diagnosis may be revised in those who show persistent C peptide production, low insulin dose (<0.5 units/kg/day), and with no tendency for DKA on insulin omission, especially three to five years after diagnosis of apparent T1DM[31]. Patients with T2DM who are less than 45 years, and who do not have features of insulin resistance (acanthosis nigricans, central obesity, hypertension, and dyslipidemia) may have MODY[30]. Genetic testing can help in the precise diagnosis of MODY. There is a 50% probability for a first degree relative to have the genetic mutation and 95% probability of getting the disease in those having the genetic mutation.

## Neonatal Diabetes Mellitus

The term neonatal diabetes is used for cases presenting with diabetes within the first six months of life. Full-term infants usually present around six weeks of age. Preterm infants

**Table II: Differential diagnosis between Youth onset T1DM, T2DM and MODY**

| Key features | T2DM | T1DM | MODY |
|---|---|---|---|
| Age at diagnosis | Rarely younger than 10 years | Peak in presentation occur between 5-7 years of age and at or near puberty | Younger than 25 years |
| Causes and genetic factors | Obesity; genetic and ethnic predisposition | Autoimmune; genetic predisposition {HLA and other genes} | Autosomal dominant: HNF1A, HNF4A, GCK are predominantly affected genes |
| Body mass index | Usually overweight / obese at onset | Lean or weight loss at diagnosis | Lean or weight loss at diagnosis |
| Associated features | Acanthosis nigricans, polycystic ovarian syndrome, hypertension, hyperlipidemia, fatty liver disease | Thyroid autoimmunity; coeliac disease | No such features as seen in T1DM or T2DM are associated with MODY |
| Diabetic ketoacidosis at presentation | Yes; 5-10% | Yes; about 25% | No |
| Family history | Strong family history | An individual presenting without a first degree relative is generally suggestive of T1DM | Strong family history with autosomal dominant inheritance |
| Insulin deficiency | Mild to moderate | Severe | Moderate |
| Insulin resistance | Moderate to severe | None or mild | None or mild |

Abbreviations: T1DM-Type 1 diabetes mellitus, T2DM- Type 2 diabetes mellitus, MODY-Maturity onset diabetes of the young , HLA-human leukocyte antigen, HNF1A-Hepatocyte nuclear factor 1-Alpha, HNF4A-Hepatocyte nuclear factor 4-Alpha, GCK-Glucokinase

can present around one week of age[32,33]. Neonatal diabetes mellitus (NDM) occurs in approximately 1 in 90,000 to 160,000 live births. There are more than 20 known genetic causes for NDM[32]. Mutation in KCNJ11, INS and ABCC8 accounts for more than 50% of NDM[33]. NDM can be transient or permanent and can be isolated or part of a syndromic presentation. The data of 11 infants reported by Jain et al. found permanent neonatal diabetes in eight infants[34].

*When should neonatal diabetes be suspected?*

There may be stress hyperglycemia in neonates, especially in those who are premature or had very low birth weight. Therefore, consider neonatal diabetes (and genetic testing) in cases[32]:

- With persistent hyperglycemia (glucose > 250 mg/dl) without an alternative explanation.
- When true serum glucose levels exceed 300 mg/dl, regardless of the time course.
- Requiring insulin before 6 to 12 months of age.

*Why is it crucial to differentiate neonatal diabetes from T1DM?*

Differentiating neonatal diabetes from T1DM is helpful, as individuals with mutations in KCNJ11 and ABCC8 may be treated with oral sulfonylureas. They account for about 40% of these patients[32,33]. In a large genetic screening study from India, 12 mutations were

identified (seven in ABCC8, three in KCNJ11, and two in the INS gene) in 33 neonatal diabetes cases. This study also reported some novel mutations. The patients carrying the KCNJ11 (Cys42Arg, Arg201Cys) and ABCC8 (Val86Ala, Asp212Tyr) were successfully treated with sulfonylurea[35]. Early sulfonylurea treatment in responsive patients, in contrast to insulin, may improve neurodevelopmental outcomes, quality of life, is safer, and also more effective in achieving good glycemic control. Another Indian study has reported the mechanism behind the effectiveness of sulfonylurea[36]. Early diagnosis helps explain additional clinical features in syndromic forms, and guide appropriate management for patients. The proportion of monogenic diabetes was 85% in infantile-onset diabetes (n=40) if onset was in the first half of infancy and 55% if it was in the second half of infancy[37]. Genetic testing for NDM is suggested for all cases of diabetes diagnosed in infancy. Wolcott-Rallison syndrome (WRS) is recognized as the most frequent cause of neonatal diabetes in children with consanguineous parents, and the mutations in the EIF2AK3 gene should be studied[38]. For patients presenting between 6 and 12 months of life, antibodies for T1DM should be tested[32,33].

## Fibrocalculous Pancreatic Diabetes (FCPD)

Fibrocalculous pancreatic diabetes is due to idiopathic non-alcoholic chronic pancreatitis characterized by recurrent bouts of abdominal pain, steatorrhoea, and pancreatic calcification[40]. The pancreatic calculi develop late in the disease course. It is reported predominantly from tropical countries in a lean adolescent or young adult of either sex. The diagnostic criteria for FCPD are listed in Table III.

**Table III: Diagnostic criteria for Fibrocalculous Pancreatic Diabetes[39]**

1. The patient should be from a tropical country.
2. Diabetes should be present.
3. Evidence of chronic pancreatitis must be present—pancreatic calculi on abdominal X-ray or at least three of the following
   - Abnormal pancreatic morphology on sonography/CT scan
   - Recurrent abdominal pain since childhood
   - Steatorrhoea
   - Abnormal pancreatic function test
4. Absence of other causes of chronic pancreatitis.

Abbreviation: CT scan-Computerized tomography scan

The salient features which may help in differentiating it from youth onset T1DM are presented in Table IV.

Kerala and Tamil Nadu have reported most of the cases in India. Malnutrition and low socioeconomic status are thought to be major predisposing factors, and with improvements in socioeconomic status and nutrition, the prevalence of FPCD has decreased from 1.6% (1991-95) to 0.2% during the years 2006-2010[40].

The patients require multiple doses of insulin for control of glycemia, and management can still be challenging. Despite hyperglycemia, ketosis rarely develops. The reasons for this could be: a) enough endogenous insulin to prevent ketogenesis, but not hyperglycemia, b) decreased glucagon reserve, and c) reduced availability of non-esterified fatty acids (substrate for ketogenesis), due to lack of subcutaneous fat.

**Table IV: Differential diagnosis between Youth onset T1DM and Fibrocalculous pancreatic diabetes (FCPD)**

| Key features | FCPD | Type 1 diabetes |
|---|---|---|
| Age at diagnosis | Most patients are diagnosed between age of 10 to 30 years. | Peak in presentation occur between 10-14 years of age and at or near puberty. |
| Clinical presentation | While some patients present with the classical symptoms of polyuria, polydipsia and polyphagia, most are asymptomatic and are detected incidentally. | Presentation is usually acute with the classical symptoms of polyuria, polydipsia and polyphagia or even with DKA. |
| Geographical variation | Reported predominantly from Kerala and Tamil Nadu. | Reported through India |
| Causes and genetic factors | Exact etiology remains elusive. Malnutrition, chronic consumption of cassava, deficiency of trace elements/ antioxidants, certain genetic factors have been implicated. | Autoimmune; genetic predisposition {HLA and other genes} |
| Body mass index | Most patients are lean, some are of normal weight and few may be obese. | Lean or weight loss at diagnosis. |
| Associated features | Abdominal pain, Steatorrhoea Physical examination may reveal bilateral parotid enlargement and abdominal distension. | Thyroid autoimmunity; coeliac disease |
| Diabetic ketoacidosis at presentation | They are usually ketosis resistant. | Yes; about 25% |
| Family history | FCPD occasionally occurs in different members of the same family, which may reflect shared environmental risk factors . | An individual presenting without a first degree relative is generally suggestive of T1DM. |
| Insulin requirement | Require insulin for control of glycaemia right from time of diagnosis. May require insulin for prevention of DKA in latter stage. | Require insulin for control of glycaemia and prevention of DKA. |
| Insulin resistance | Moderate to severe | None or mild |

Abbreviations: T1DM-Type 1 diabetes mellitus, FCPD-Fibrocalculous pancreatic diabetes, DKA-Diabetic ketoacidosis, HLA-Human leukocyte antigen

## Mitochondrial Diabetes

Mitochondrial diabetes is also known as maternally inherited diabetes and deafness. It is a monogenic form of diabetes, as are MODY and NDM[41]. The average age of onset is between 35 and 40 years, in contrast to T1DM or other monogenic forms of diabetes that usually present much earlier. It can present like T1DM or T2DM. Initially, patients with phenotypes like T2DM can be treated by diet or sulfonylurea[41]. Metformin should not be prescribed due to the risk of lactic acidosis. Most patients will require insulin treatment due to progressive insulinopenia.

It should be suspected where there is robust familial clustering of diabetes, the characteristic also seen with MODY. However, in mitochondrial diabetes, there is a maternal transmission

and a bilateral hearing impairment in most of the carriers. The precise diagnosis can be established by genetic analysis. A range of mutations in mitochondrial DNA (mtDNA) has been implicated. However, in many patients, it is associated with A3243G mutation in mtDNA[41].

## Summary

Type 1 Diabetes mellitus (T1DM) is an autoimmune disease characterized by insulin deficiency and hyperglycemia in people with underlying genetic susceptibility. The incidence of T1DM in India is 4.9 cases/100000/year. The peak incidence for T1DM is seen between 10-14 years of age, though it can affect an individual at any age. Genetic factors play a significant role in the etiology of T1DM. The risk of T1DM is 3%, 5%, and 8%, respectively, when mother, father, and sibling have T1DM[9]. The HLA haplotypes with a strong association with T1DM are DR3-DQ2 and DR4-DQ8, present in 30-40% patients with T1DM than 2.4% in the general population. There are more than 50 non-HLA loci, which increases the susceptibility for T1DM, though the association is weak than the HLA region. T1DM is associated with autoantibodies in 70-90% of the individuals with the disease. T1DM can have variable presentations[17-21]. Children often present acutely, and adults usually present with a more gradual onset. World Health Organization proposed a recent classification system of diabetes in 2019. The WHO 2019 classification system has removed the previous subtypes for T1DM and T2DM. A new entity called 'hybrid forms of diabetes' is introduced, including a) slowly evolving immune-mediated diabetes of adults and b) ketosis-prone type 2 diabetes. T1DM needs differentiation from other forms of diabetes seen in a younger age group. The age group of 20-40 years has the highest heterogeneity and is at risk of misclassification. WHO has also introduced a category called 'unclassified diabetes' which is a temporary label, when it is difficult to make a precise diagnosis regarding the type of diabetes, especially close to the time of diagnosis.

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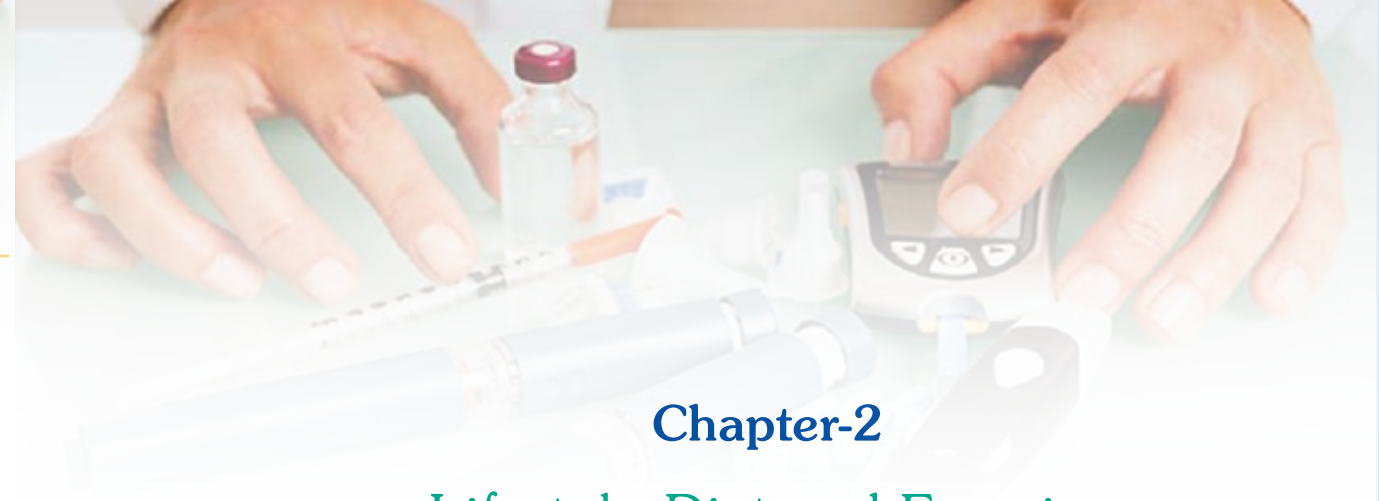

# Chapter-2

# Lifestyle-Diet and Exercise

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## Introduction

Lifestyle management (LSM) plays an essential role in managing type 1 diabetes mellitus (T1DM). Understanding the effect of diet and physical activity on glycemia is essential for optimal management of T1DM.

## Aims of Nutritional Management

1) Maintain glycemia in the normal to the near-normal range with minimal/no hypoglycemia.
2) Maintain optimal blood pressure, weight, and lipid levels.
3) Ensure adequate nutrition to facilitate healthy growth and development in children and adolescents.
4) To prevent the development or progression of diabetes-related microvascular and macrovascular complications.
5) Address individual nutrition needs, incorporating personal, social, and cultural preferences.
6) Improve overall health through appropriate food choices.

## Concepts of energy and proximate principles of diet

Individualize the diet plan for each patient with diabetes, considering his/her socio-cultural environment, food habits, preferences, and work schedule to facilitate proper compliance. The energy requirement for children and adults with T1DM (Table I) is similar to the general population. Consider reduction in the caloric intake for obese and overweight individuals.

**Table I: Nutritional recommendations for individuals with T1DM**

- Carbohydrate: 50–55%
  *Sucrose intake < 10%*
- Fat 25–35%
  *Saturated fat + trans fatty acids: < 10% Trans fatty acids < 1%*
  *Polyunsaturated fat: < 10% Monounsaturated fat: 10-20%*
- Protein 15–20%

Abbreviations: TIDM-Type 1 diabetes mellitus

### *Carbohydrates*

The recommended carbohydrate intake is 50-55% of total calories. Too much carbohydrate restriction may hamper growth in children and adolescents and hence, should be

discouraged1. Even in adults, ensure a minimum of 130 g of carbohydrate per day to provide sufficient glucose as fuel for the brain. The effects of carbohydrate restriction below 130 g/day are not clear. In pregnancy, the minimum recommended carbohydrate intake is 175 g/day[2]. However, in the Indian Subcontinent, approximately two-third of calories are obtained from carbohydrates in individuals with diabetes, which is higher than the recommended amount[3]. Hence, it is essential in Indians with diabetes to monitor carbohydrate intake and reduce if possible.

Moreover, Indian (especially South-Indian and East-Indian) diets are rich in simple carbohydrates. The intake of complex carbohydrates should be encouraged to constitute at least 70% of the total carbohydrates. The use of sucrose should be limited to less than 10%, and preferably less than 5% of total calories. Although sugar is not much different from isocaloric amounts of starch for glycemic excursions, consuming high amounts of sugar in sugary drinks may not be adequately covered with insulin, and hence, should be avoided. Healthy sources of carbohydrate foods include wholegrain bread and cereals/ millets, legumes (peas, beans, and lentils), fruits, vegetables, and low-fat dairy products. With a reduction in the intake of carbohydrate components in the diet, the intake of fat increases. In this situation, execute caution to ensure the consumption of good quality fat.

### *Fiber*

Dietary fiber is a non-digestible complex carbohydrate derived from foods of plant origin. The recommended dietary fiber intake for children ≥ one year of age is 14 g per 1000 kcal. Another way of calculating the daily dietary fiber requirement in children over two years is 'Age in years + 5 g'. Encourage consumption of foods such as legumes, fruits, vegetables, and wholegrain cereals to ensure adequate fiber intake. Higher fiber foods may help to improve satiety and replace more energy-dense foods. Soluble fiber helps moderate postprandial blood glucose levels and lower serum cholesterol levels to prevent cardiovascular disease. Good sources of soluble fiber include oats and oatmeal, legumes (peas, beans, lentils), barley, fruits, and vegetables (especially oranges, apples and carrots). Insoluble fiber also offers several benefits to intestinal health, including a reduction in the risk of hemorrhoids and constipation. Most of the insoluble fibers come from the bran layers of cereal grains. It is good to increase fibers gradually to avoid bloating and discomfort.

### *Fat*

Recommended daily intake for fats is up to 30% of total calories. Infants and children younger than two years of age may have a higher daily fat intake of up to 35%. Dietary fats are of two kinds: visible fats, like that in cooking medium or invisible fat, which is inherently present in the foods. Take into consideration the calorie consumption of both these fats. Excess consumption of saturated fats and cholesterol increases serum LDL cholesterol associated with increased cardiovascular disease risk. Hence, the daily intake of saturated fats should be limited to less than 10% of the total calories and dietary cholesterol to less than 300 mg/day. In patients with raised LDL cholesterol, restrict the daily saturated fat intake to below 7% of total calories and cholesterol intake to less than 200 mg/ day. The common foods rich in saturated fats and cholesterol include egg yolk, ham, bacon, red meats, whole milk, cheese, butter, ghee, cream, and cream-based desserts, vanaspati, coconut oil, palm oil and it is best to limit their consumption. The right options for low cholesterol and low saturated fat foods are fish, lean chicken, low-fat milk and curd, low-fat cheese, buttermilk, and cooking oils rich in healthy fatty acids.

**Unsaturated fatty acids** are essential components of lipid membranes and comprise polyunsaturated and monounsaturated fatty acids. They are derived mainly from plant and vegetable sources. These fats have beneficial effects on LDL cholesterol and, in the case of monounsaturated fatty acids, also on HDL cholesterol and glycaemic control. These help to reduce the risk of cardiovascular disease.

**Monounsaturated fatty acids (MUFA)** are there in olive, canola, groundnut, peanut, sesame, rice bran, mustard oils, almonds, and avocados. This form of fat (particularly MUFA with *cis*-configuration) is most lipid-friendly, and 10-20% of the total daily calories should be from their intake.

**Polyunsaturated fatty acids (PUFA)** should be less than 10% of total daily calories. Significant components of PUFA are ω-6 PUFA and ω-3 PUFA. Omega-3 PUFAs lower serum triglycerides. Encourage regular ω-3 intake from natural food sources rather than supplements. Coldwater fatty fishes (mackerel, salmon, sardine, herring, and tuna) are good non-vegetarian sources of ω-3 PUFA whereas flaxseeds, walnuts, chia seeds, soybean oil, canola oil, kidney beans, tofu, broccoli, spinach, cauliflower, and Chinese cabbage are good vegetarian sources of ω-3 PUFA. Omega-6 PUFA helps to reduce serum LDL cholesterol and is found in various vegetable cooking oils (safflower, sunflower, soy, cottonseed, corn, canola, peanut, and sesame), pulses, vegetables, cereals, walnuts, seeds, eggs, and poultry. However, do not recommend consumption of ω-6 PUFA > 10% of the total daily fat. A higher ω-6/ω-3 PUFA ratio of > 5-10 may promote several diseases, including cardiovascular disease. ω-6/ω-3 PUFA ratio in Indian diet is much higher than optimal; hence, a mix or alternate oils rich in ω-6 PUFA and ω-3 PUFA to attain the lower ratios is preferred. Similarly, mixing and alternating oils should also focus on allowing the recommended MUFA in the diet.

Plant **sterol and stanol esters**, typically found in enriched foods, may modestly reduce total and LDL cholesterol and may be considered for children ≥ five years with high serum total or LDL cholesterol.

**Trans-fatty acids,** though unsaturated fatty acids, are structurally different and have adverse health effects. They not only increase LDL cholesterol but also reduce HDL cholesterol. Hence, the use of trans-fats should be limited as much as possible, ideally to less than 1% of total caloric intake. They form when vegetable oils are processed to make them solid (partial hydrogenation). They are scarce in nature but are commonly found in packaged baked goods (cakes, cookies), snack foods (potato chips), fried food (French fries, doughnuts, fried chicken) and margarine (stick margarine, vanaspati as a cooking medium).

### *Proteins*

The protein requirement in children and adolescents varies from 2 g/kg at one year, 1 g/kg at ten years to 0.8-0.9 g/kg in adolescence. Recommended protein intake is 15-20% of the caloric requirement. Protein promotes growth only when calorie intake is sufficient, and high protein intake may compromise growth by reducing calorie intake and may also reduce vitamin and mineral intake.

Proteins from animal sources (fish, milk, egg white, poultry, and meats) are of better quality as they provide all essential amino acids, but their use is associated with higher salt and saturated fat content. Remove the skin and visible fat while consuming the animal sources of proteins. On the other hand, proteins from vegetarian sources (soy, beans, and lentils) contain less saturated fat and are rich in fiber and complex carbohydrates. Hence,

the consumption of proteins from both vegetarian and low-fat non-vegetarian sources, preferably in equal amounts, is recommended.

In patients with diabetic nephropathy, daily protein intake should not be restricted to less than 0.8 g/kg body weight to avoid the risk of malnutrition. There is no benefit of protein intake less than 0.8 g/kg body weight on glycemia, cardiovascular risk, or decline in glomerular filtration rate.

### *Vitamins and Minerals*

Vitamin and mineral requirements in children with diabetes are the same as in other healthy children. There is no clear evidence to suggest that routine vitamin or mineral supplementation in children with diabetes is beneficial. Ensure the RDA of all vitamins and minerals in the diet by appropriate choice of macronutrients.

### *Salt*

Recommendations for salt intake in children with diabetes are similar to that of healthy children. Daily salt intake should be limited to 1000 mg (2.5 g salt) in 1–3 years old children, 1200 mg (3 g salt) in 4–8 years old children, 1500 mg (3.8 g salt) for children and adolescents aged ≥ nine years and 2300 mg (6.0 g salt) in adults. Processed foods are rich in salt, and hence their intake should be limited.

### *Sweeteners*

The commonly used non-nutritive and hypocaloric sweeteners include saccharin, neotame, aspartame, acesulfame K, stevia, alitame, and sucralose. They are commonly used in low sugar, 'light' or 'diet' products to improve sweetness and palatability and are also used to replace table sugar in cooking at home. Intake of sweeteners not exceeding acceptable daily intakes (ADI) is considered safe[4]. They have the potential to replace caloric or carbohydrate intake if substituted for caloric sweeteners. However, the benefits of their use for better glycemia, weight reduction, or reduction of cardiometabolic risk factors are limited.

## Carbohydrate assessment, Glycaemic Index (GI), Glycaemic Load (GL) and carbohydrate counting

The GI is an indicator of the quality of carbohydrates in food and measures the rapidity of increment in plasma glucose after ingestion of a food item. GI is evaluated by calculating the area under the curve for blood glucose rise for 2 hours after consuming 50 g of a test-food compared to that of 50 g of oral glucose (preferably) or white bread (reference food). A value of 100 represents, rise in blood glucose similar to pure glucose. The GI of foods varies from 0 to 100. Foods with a low GI produce a lower rise in blood glucose during the first 2-3 hours after their ingestion. Table II presents the classification of common Indian foods as low (≤ 55), medium (56-69), and high (≥ 70) glycaemic index. Remember, GI is not a sole criterion for deciding diet composition as there are certain limitations to its use. Some food products (such as ice cream) may have a large amount of fat, delaying the absorption of carbohydrate and rendering a relatively low GI value but may have adverse health effects in the long term.

GL is a measure of both the quality (GI value) and quantity (g per serve) of a carbohydrate in a meal. Determine foods GL by multiplying its GI by the amount of carbohydrate the food contains in each serve and dividing the product by 100. Therefore, the GL provides a summary measure of the relative glycaemic impact of a "typical" serving of the food. Classify foods with a GL < 10 as low GL and those with ≥ 20 as high GL value.