Childhood and Adolescent Onset Type 1 Diabetes in India

ABSTRACT
According to International Diabetes Federation, there are 382 million people with diabetes globally. While the majority of this is constituted by type 2 diabetes, numbers of type 1 diabetes are also increasing. This paper reviews the clinical and epidemiological features and management issues in children and adolescents with type 1 diabetes.

Keywords: Type 1 diabetes, Children and adolescents, Management, Complications, Epidemiology, India.

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INTRODUCTION
Recently, physicians and pediatricians are facing fresh challenges due to new epidemics affecting children’s physical and mental health. Earlier, infectious diseases like viral infections, mumps, chicken pox, pneumonia, diarrhea, nutritional deficiencies dominated childhood diseases. Today these are being replaced by noncommunicable diseases like overweight/obesity and diabetes.1-3

Diabetes mellitus is one of the commonest endocrine and metabolic diseases of childhood. Till recently, diabetes in children (defined as onset below 12 years) and adolescents (defined as onset between 12 and 19 years) was almost exclusively type 1 diabetes (T1DM) and this has changed, as there is increased recognition of a number of different forms of ‘nontype 1 diabetes’ in the young. This includes type 2 diabetes (T2DM), maturity onset diabetes of young (MODY), fibrocalculous pancreatic diabetes (FCPD) and diabetes due to genetic disorders. This review will focus exclusively on the epidemiology, clinical profile, management and complications of childhood onset type 1 diabetes with special reference to published studies from India.

Epidemiology
T1DM is a disorder that arises following the autoimmune destruction of insulin-producing pancreatic β-cells.4 The disease is most often diagnosed in children and adolescents, usually presenting with a classic triad of symptoms, (i.e. polydypsia, polyphagia, polyuria) along with severe hyperglycemia, necessitating the need for exogenous insulin replacement on a lifelong basis.

T1DM probably accounts for 5 to 10% of all diagnosed diabetes. About 40 to 60% of persons with T1DM are younger than 20 years of age at onset, thus making diabetes one of the most common severe chronic diseases of childhood affecting 0.3% of the general population by the age of 20 years and 0.5 to 1% during the lifespan.5 The worldwide prevalence of T1DM is 0.1 to 0.3%, with 78,000 new cases every year, especially among young individuals (<5 years). Some 79,100 children under 15 years are estimated to develop T1DM annually worldwide.6

According to the reports of SEARCH group7 the incidence of T1DM peaks around the age of 10 years and is highest among non-Hispanic Whites followed by non-Hispanic Blacks, Hispanics, Asia and Pacific Islanders and American Indian/Alaskan Natives. The South-East Asia Region (SEAR) has a high prevalence of T1DM in children, with an estimated 77,900 children affected. In 2013, alone an estimated 12,600 children under the age of 15 in SEAR developed T1DM.6

India due to its sheer size (1.2 billion people) accounts for most of the children with T1DM in the SEAR. The incidence rate for T1DM in India was frequently used in extrapolation for other countries in the region and therefore the estimates in India are of great significance. In the 1990’s, Menon et al8 had done an overview of childhood onset diabetes mellitus in India. Prevalence of juvenile diabetes (onset below 15 years) among all diabetes hospital/clinic based data was presented and the prevalence ranged from 0.8 to 3.61% during the period from 1964 to 1989.

After a long gap of two decades, this review tries to fulfill the lacunae by presenting the incidence and prevalence/percentage of T1DM reported in India so far in Table 1. In a population based study conducted in South India for a period of 1991 to 1994, the incidence for the 4 years period was 10.5/100,000/year (CI 5.0) for children up to 15 years of age.9 Similarly, a study from Karnal district in 2008, showed a prevalence of 18.3/100,000 in the 0 to 14
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Etiology and Pathogenesis of T1DM

The precise cause of T1DM is unknown but there are a number of possible contributory factors and some of them were discussed below.

Genetic Factors

T1DM tends to run in families. Epidemiologic studies have shown that brothers and sisters of children with T1DM have a higher chance of developing the disease (6% in siblings vs 0.4% in the general population) among the relatives of T1DM patients, underlying the role of genetic factors as a cause of T1DM.35 Twin studies of T1DM from a large Finnish cohort of 22,650 twin pairs, 228 of which had at least one twin with T1DM (44 monozygotic (MZ), 183 dizygotic (DZ), and 1 of unknown zygosity), demonstrated a 27.3% MZ pair-wise concordance, and a 3.8% DZ concordance.36

Since, the 1970s it has been acknowledged that genes belonging to the human leukocyte antigen (HLA) system on chromosome 6 constitute the most important genetic risk factor.37–39 The chromosomal locations of these ‘diabetes genes’ are called inherited susceptibility loci. There are now at least 18 insulin-dependent diabetes mellitus (IDDM) susceptibility loci (IDDM 1 to IDDM 18).40 HLA make up the human major histocompatibility complex (MHC), which presents antigens to the immune system, but now more than 40 additional loci are known to significantly affect T1D risk.

Recently, genome-wide association studies (GWAS) have been used to identify genetic loci-associated T1DM. In contrast to the traditional methods of using a candidate gene approach, GWAS scans the whole genome for single nucleotide polymorphisms (SNPs) that occur more frequently

<table>
<thead>
<tr>
<th>Author name</th>
<th>Place</th>
<th>Year or period of study</th>
<th>Number of T1DM in children reported and given as % where applicable</th>
<th>Total sample studied</th>
<th>Age at diagnosis</th>
<th>Ref. no.</th>
</tr>
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<tr>
<td>Bai et al</td>
<td>Chennai</td>
<td>1991</td>
<td>10513</td>
<td>School children</td>
<td>&lt;15 years</td>
<td>11</td>
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<tr>
<td>Ramachandran et al</td>
<td>1991-1994</td>
<td>10.5/100,000 person-years</td>
<td>IDDM registry</td>
<td>&lt;6 years</td>
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<tr>
<td>Kalra et al</td>
<td>Karnal</td>
<td>2008</td>
<td>3.82/100,000</td>
<td>Endocrine center registry</td>
<td>0-6 years</td>
<td>10</td>
</tr>
</tbody>
</table>

Prevalence/percentage of T1DM (clinic based)

<table>
<thead>
<tr>
<th>Author name</th>
<th>Place</th>
<th>Year or period of study</th>
<th>Number of T1DM in children reported and given as % where applicable</th>
<th>Total sample studied</th>
<th>Age at diagnosis</th>
<th>Ref. no.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Verma IC</td>
<td>New Delhi</td>
<td>1980-84</td>
<td>44 (80.0)</td>
<td>55</td>
<td>5-12 years</td>
<td>12</td>
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<tr>
<td>Venkataraman et al</td>
<td>Chennai</td>
<td>1979-89</td>
<td>126 (7.88%)</td>
<td>160</td>
<td>&lt;20 years</td>
<td>13</td>
</tr>
<tr>
<td>Mohan et al</td>
<td>1990</td>
<td>165 (63.9)</td>
<td>258</td>
<td>&lt;20 years</td>
<td>14</td>
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<tr>
<td>Ramachandran et al</td>
<td>1991</td>
<td>0.26/100 (30 children)</td>
<td>116, 486</td>
<td>&lt;15 years</td>
<td>15</td>
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<tr>
<td>Ramachandran et al</td>
<td>2000</td>
<td>617</td>
<td>-</td>
<td>&lt;20 years</td>
<td>16</td>
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<tr>
<td>Kumar et al</td>
<td>1991-2001</td>
<td>8 (0.01%)</td>
<td>70000</td>
<td>≤1 year</td>
<td>17</td>
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</tr>
<tr>
<td>Mohan et al</td>
<td>2007</td>
<td>286 (65.9%)</td>
<td>434</td>
<td>&lt;16 years</td>
<td>18</td>
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<td>Ganesh et al</td>
<td>2003-2007</td>
<td>4 (0.05)</td>
<td>83</td>
<td>≤1 years</td>
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<tr>
<td>Varadarajan et al</td>
<td>1999-2010</td>
<td>350 (81%)</td>
<td>432</td>
<td>&lt;12 years</td>
<td>20</td>
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<tr>
<td>Amutha et al</td>
<td>1992-2009</td>
<td>940 (68.5)</td>
<td>1372</td>
<td>≤19 years</td>
<td>21</td>
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<tr>
<td>Varadarajan et al</td>
<td>1999-2012</td>
<td>40 (7.9)</td>
<td>506</td>
<td>≤1 year</td>
<td>22</td>
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<tr>
<td>Kota et al</td>
<td>Hyderabad</td>
<td>1997-2011</td>
<td>260</td>
<td>-</td>
<td>10.5 ± 7.2 years</td>
<td>23</td>
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<tr>
<td>Sahay et al</td>
<td>1999-2002</td>
<td>28 (59.6)</td>
<td>47</td>
<td>&lt;20 years</td>
<td>24</td>
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<tr>
<td>Abraham et al</td>
<td>Central Kerala</td>
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<td>39 (67%)</td>
<td>58</td>
<td>&lt;20 years</td>
<td>25</td>
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<tr>
<td>Bhadada SK et al</td>
<td>Chandigarh</td>
<td>2002-2008</td>
<td>189</td>
<td>-</td>
<td>10.8 ± 7.3</td>
<td>26</td>
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<tr>
<td>Unnikrishnan et al</td>
<td>Multicentric study</td>
<td>2006-2008</td>
<td>535 (89%)</td>
<td>603</td>
<td>&lt;20 years</td>
<td>27</td>
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<tr>
<td>Bhatia et al</td>
<td>Lucknow</td>
<td>2004</td>
<td>130 (81%)</td>
<td>160</td>
<td>&lt;18 years</td>
<td>28</td>
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<tr>
<td>Balasubramanian et al</td>
<td>10 years period</td>
<td>55 children</td>
<td>-</td>
<td>&lt;20 years</td>
<td>29</td>
<td></td>
</tr>
<tr>
<td>Singh et al</td>
<td>1992-1997</td>
<td>83 (57.2)</td>
<td>145</td>
<td>13.8 ± 7.3</td>
<td>30</td>
<td></td>
</tr>
<tr>
<td>Samal et al</td>
<td>Cuttack</td>
<td>1983-1988</td>
<td>54 (60%)</td>
<td>90</td>
<td>&lt;15 years</td>
<td>31</td>
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<tr>
<td>Mazumder et al</td>
<td>Kolkata</td>
<td>2004-2006</td>
<td>41 (70.7)</td>
<td>58</td>
<td>&lt;18 years</td>
<td>32</td>
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<tr>
<td>Kumar P et al</td>
<td>Karnataka</td>
<td>1995-2008</td>
<td>134 (43%)</td>
<td>311</td>
<td>9-14 years</td>
<td>33</td>
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<tr>
<td>Zargar et al</td>
<td>Srinagar</td>
<td>1990-1999</td>
<td>84 (90.3)</td>
<td>93</td>
<td>&lt;20 years</td>
<td>34</td>
</tr>
</tbody>
</table>
in people suffering from T1DM. The associated SNPs then are used to mark the susceptibility loci. By using the SNP typing technology, a number of additional susceptibility loci were discovered for T1DM, namely: CLEC16A, C1QTNF6, UBASH3A, CD226, PTPN2, CTSH, SH2B3, ERBB3, PRKCQ, TAGAP, IL-2RA, TNFAIP3, BACH2, IL-7R, IL-2, CCR5, IFIH1, IL-18RAP, RGS1, IL-10, IL-19, IL-20, GLIS3, CD69 and IL-27.41-43

**Viral Infections and Toxins**

Epidemics of enteroviral infections in the autumn and winter months are associated with an increase in the incidence of T1DM. Several viruses (e.g. coxsackie B, enteroviruses, rubella, mumps and cytomegalovirus) have been implicated in the etiology of T1DM.44 Also, T cells target the envelope proteins (VP1, VP2 and VP3) of coxsackie virus B4, but the T cell proliferative response was reduced markedly in T1DM patients compared with control subjects, which eventually results in destruction of beta cells.45 Possible mechanisms for their effect include molecular mimicry in which the immune response to the infection cross reacts with islet antigens. Alternatively viral infections including those occurring antenatal may have more direct effects on β-cells. Ingestion of the rodenticide vacor is also known to be associated with development of T1DM.

**Environmental Factors**

Among the various environmental factors, exposure to antigenic substances early in life is thought to contribute to T1DM.46 Undissolved gluten causes subclinical inflammation of intestinal mucosa, which raises the proportion of aggressive T cells. The functional state of beta cells also plays a role in the pathogenesis of T1DM, and food intake with a high glycemic index increases the insulin demand and forces the beta cell to produce more insulin, which accelerates its destruction. This observation has inspired the ‘Accelerator hypothesis’, which states that increased weight gain in youngsters might accelerate T1DM development.46

**Nutrition and Dietary Factors**

Breastfeeding appears to provide protection against the risk of developing T1DM.47 Available evidence to date shows that lack of breastfeeding is a possible modifiable risk factor for the manifestation of both T1DM and T2DM. The benefits of breastfeeding have been attributed to bioactive substances, which promote the maturation of the immune system, reduce insulin resistance, and prevent excessive weight gain during childhood.48

Early introduction of Cow’s milk appears to be a risk factor for the development of T1DM.49 Many new patients with T1DM have IgG antibodies to bovine serum albumin, a protein in Cow’s milk with similarities to the islet cell antigen. This protein may stimulate autoantibody production leading to islet cell destruction as a result of molecular mimicry.

Breastfeeding may be viewed as a surrogate for the delay in the introduction of diabetogenic substances present in formula or early childhood diet. Circumstantial evidence suggests a connection between T1DM and consumption of foods and water containing nitrates, nitrites or nitrosamines.50-52

**Pancreatic β-cell Reserve—C-peptide Assay**

It is known that at the time of T1DM diagnosis, 80 to 90% of the pancreatic islet β-cells are destroyed. C-peptide determination is used to better understand the course of T1DM. It was shown that young children with classical ketosis prone insulin dependent diabetes also had residual insulin secretion.54 Very young children especially those with onset after infections tend to have less C-peptide.

In recent years, C-peptide determinations have gained lot of interest. Efforts to preserve residual insulin secretion have increased dramatically in the last few years.55 The heterogeneity of diabetes at clinical onset along with the increasing incidence in children and adolescents makes it of interest to test if C-peptide may improve the classification of newly diagnosed children. Katz et al57 reported fasting C-peptide levels of 0.38 ± 0.37 ng/ml can distinguish T1DM from T2DM with 83% sensitivity. In our study,21 we found that a combination of clinical criteria and C-peptide criteria is largely helpful for classification of our children with diabetes and this also has been reported by Ludvigsson et al.58

**Antibodies**

Several autoantibodies have been identified in newly diagnosed cases of T1DM. Currently, four major antibodies namely (the 65 kDa form of glutamic acid decarboxylase (GAD65), insulinoma antigen 2 (IA-2), insulin auto, antibodies (IAA) and zinc transporter 8 (ZnT8) have been shown to be found in T1DM and approximately 94% of all T1DM patients have at least one of these antibodies at clinical onset.59 Monitoring these autoantibodies is currently the most reliable biomarker in the prodromal phase of T1D, since their appearance typically precedes overt T1D onset for years or even decades.60,61 This provides a window for therapeutic intervention, and as a measure of treatment efficacy.62-64 Most important, the prevalence of multiple types of autoantibodies identifies individuals with the highest risk of progression to clinical disease in those in the prediabetes stage of T1DM.
Clinical Features of T1DM

1. Abrupt onset of severe symptoms (polyuria, polydipsia and/or weight loss).
2. Presence of ketosis or ketoacidosis.
3. Severe diabetes with markedly elevated glycated hemoglobin levels (HbA1c).
4. Usually patients are nonobese or even lean.
5. Family history of diabetes in parents is usually absent.
6. C-peptide test shows absence or very low pancreatic β-cell reserve.
7. GAD, IA2, zinc transporter or other islet cell antibodies may be present.
8. Patients require lifelong insulin from time of onset for survival and to maintain good health and for control of hyperglycemia.

Management of T1DM

The basic elements of T1DM management are insulin administration, nutrition management, physical activity, self, monitoring of blood glucose (SMBG), and the avoidance of hypoglycemia. In T1DM, since the pancreas can no longer produce insulin, patients are required to take insulin daily, either by injection or via an insulin pump. Other routes of delivering insulin are currently being investigated.

Children are now being treated with basal bolus regime or an insulin pump. Basal bolus regime—this is the most physiological way of matching the insulin secretion in our body by insulin injections. In this, children receive a basal insulin dose with long acting insulin analog and premeal boluses are given by rapid acting insulin analogs just before meals. The total insulin dosage in children is 0.5 units/kg/day to 1.0 units/kg/day with up to 2.0 units/kg/day during puberty period.65

Premeal boluses are calculated more accurately by carbohydrate (CHO) counting, insulin to carbohydrate ratio (ICR) and correction bolus.

Carbohydrate counting is a meal planning wherein the patient identifies the CHO in the meal, estimates the total CHO amount in the meal and calculates the insulin to balance the CHO using ICR (insulin to CHO ratio) thereby controlling postprandial glucose levels more accurately. It is done by two methods, CHO exchange (1CHO serving = 15 gm carb portions) and CHO gram counting (food is weighed and CHO is calculated).

Insulin to carbohydrate ratio is the amount of insulin required to cover a specified number of CHO grams. This is calculated by 500/300 rule [500/total daily dose of insulin (TDD) for children aged 5 years and above and 300/TDD for preschool children].

Correction bolus is the amount of extra, fast acting insulin added to or subtracted from a bolus to correct a blood glucose that is above or below target (90-140 mg). This is calculated by [(Current blood glucose – Target blood glucose)/insulin sensitivity factor (ISF)]. ISF is how much 1 unit of insulin will lower the blood glucose by. This is calculated by 1500 or 1800 rule (1500/TDD for short acting insulin and 1800/TDD for rapid acting insulin).66

Further adjustment of insulin or food intake may be made based on anticipation of special circumstances, such as increased exercise and intercurrent illness. Children on these regimens are expected to check their blood glucose levels (self monitoring of blood glucose) routinely before meals and at bedtime.67

Insulin pumps are being more commonly used because of their unique ability to continuously infuse insulin, closely mimicking that of physiological secretion from a normal pancreas. The insulin pump separates the insulin used as background, or basal insulin, from the insulin needed for meal and corrections boluses and therefore insulin can be more exactly matched to the metabolic need achieving better glycemic control. Scientific evidence from published studies have proven added benefit of insulin pumps in improving quality of life and normalizing sugars. The success of insulin pump therapy depends on selection of the right candidate, extensive education, motivation, and implementing the sophisticated programs with skill.68

Advantages of Insulin Pump

• Improvement in HbA1c
• Reduction in blood sugar fluctuations
• Reduction in major and minor hypoglycemic episodes
• Reduction in total daily dose of insulin
• Improvement in quality of life.

Disadvantages of Insulin Pump

• Cost of pumps and consumables is beyond the reach of the common individual
• There is a risk of infection if the cannula is not changed once in every 3 days
• Overeating and frequent bolusing could result in weight gain and misuse of an insulin pump
• Improper use of insulin pump boluses can lead to insulin stacking and low sugar.68

Complications of Diabetes

Acute Complications

Acute complications of T1DM include diabetes ketoacidosis (DKA), hypoglycemia and infections. An estimated 26% of the patients have at least one episode of severe hypoglycemia
within the initial 4 years of diagnosis, with little relation to
demographic or socioeconomic factors. The incidence of
severe hypoglycemic episodes varies between 6 and 20/100
patient-years depending on age, geographic location and
intensity of insulin treatment. A study by Amutha et al.70
Diabetic ketoacidosis in children continues to be an
important cause of morbidity and mortality. Malnutrition also
increases the risk of diabetic ketoacidosis-related compli-
cations.71 Boys and girls were equally affected. Newly diag-
nosed diabetics constituted more than 50% of total DKA
admission.72 Management requires careful replacement of fluid
and electrolyte deficits, intravenous administration of insulin,
and close monitoring of clinical and biochemical parameters
directed toward timely detection of complications, including
hypokalemia, hypoglycemia and cerebral edema.72,73

Chronic Complications
Long-term complications may be microvascular (retinopathy,
nephropathy and neuropathy) or macrovascular (ischemic
heart disease, peripheral vascular disease). Microvascular
complications may develop in puberty or early adult hood
whereas macrovascular complications affect in later years.
The longer the duration of diabetes, the greater the risk of
complications which increases significantly following puberty.
The risk of developing complications may also be increased
by poor glycemic control, hypertension, dyslipidemia and
behavior, such as smoking in addition to genetic factors.

Background diabetic retinopathy in childhood may
rarely progress to proliferative retinopathy later in life.
This can be successfully treated in its early stages with
laser photocoagulation therapy. Cataracts may also occur in
T1DM patients but is very rare under the age of 20 years.
The prevalence of retinopathy in adolescents varies from
18 to 47%. More than 90% of patients with T1DM will
eventually develop some degree of retinopathy. A pilot
study done by SEARCH group estimated, the prevalence
of diabetic retinopathy among those with T1DM was 17%
which was similar to that reported from Australia74 with
similar duration. The SEARCH for diabetes in Youth study75
reported a high prevalence of elevated albumin creatinine ratio (22.2%)
in youth with T2DM, well over twice the percentage for participants with T1DM (9.2%).

A cohort of 354 patients with T2DM, age of onset between
15 and 30 years (T2DM15-30), were compared with 470
patients with T1DM with a similar age of onset (T1DM15-30)
to study the clinical and mortality outcomes. No significant
differences were found between T1DM and T2DM with
regard to prevalence of retinopathy or renal function assessed
by eGFR, but a marked excess of macrovascular disease was
found in the T2DM15-30 cohort, with a higher prevalence
of ischemic heart disease (12.6 vs 2.5%, p, 0.0001), stroke
(4.3 vs 0.7%, p = 0.002), and the composite end point of any
macrovascular disease despite having shorter duration of
diabetes and remarkably similar glycemic exposure as T1DM
15 to 30 cohort.76

Prevalence of diabetes complications in T1DM reported
from India is outlined in Table 2.

Most of the available data are from clinic based studies
and the prevalence or percentages represents against their
total patients registered from those particular clinics in India.
The age ranges studied are usually inconsistent. There is
also a lack of uniformity in classification of diabetes types.
Some of the adolescents who were originally diagnosed
as having T1DM were later found to have T2DM and vice
versa. Some of the children and adolescents were not able to
be classified due to lack of unavailability of GAD antibody
and C-peptide assays. Another problem is migration of
patients to other centers.

Table 2: Diabetes complications in T1DM reported from India

<table>
<thead>
<tr>
<th>S. no.</th>
<th>Author name</th>
<th>Year or period of study</th>
<th>Age at onset in years/duration of diabetes in years</th>
<th>Retinopathy n (%)</th>
<th>Nephropathy n (%)</th>
<th>Neuropathy n (%)</th>
<th>Ref. no.</th>
</tr>
</thead>
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<tr>
<td>1</td>
<td>Venkataraman et al</td>
<td>1979-1989</td>
<td>&lt;20 years/ 6 to 9 years</td>
<td>7/126</td>
<td>5/126</td>
<td>19/126</td>
<td>13</td>
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<tr>
<td>2</td>
<td>Sharma et al</td>
<td>1991</td>
<td>–</td>
<td>15/35</td>
<td>–</td>
<td>–</td>
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<tr>
<td>3</td>
<td>Ramachandran et al</td>
<td>2000</td>
<td>≤20 years/ 11-15 years</td>
<td>23/74</td>
<td>16/74</td>
<td>19/617</td>
<td>16</td>
</tr>
<tr>
<td>4</td>
<td>Bhatia et al</td>
<td>2004</td>
<td>&lt;18 years/ &gt;5 years</td>
<td>22%</td>
<td>18%</td>
<td>–</td>
<td>28</td>
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<tr>
<td>5</td>
<td>Unnikrishnan et al</td>
<td>2006-2008</td>
<td>&lt;20 years/ &lt;7 years</td>
<td>27/535</td>
<td>29/535</td>
<td>32/535</td>
<td>27</td>
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<tr>
<td>6</td>
<td>Kumar et al</td>
<td>1995-2008</td>
<td>&lt;25 years</td>
<td>14/166</td>
<td>20/230</td>
<td>12/230</td>
<td>33</td>
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<td>7</td>
<td>Amutha et al</td>
<td>1992-2009</td>
<td>&lt;20 years/ 5-14 years</td>
<td>38/171</td>
<td>9/183</td>
<td>5/139</td>
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</tr>
</tbody>
</table>
The Indian Council of Medical Research recently set up a national registry of diabetes in the young (onset <25 years of age). This shows that private diabetes centers have more cases of young-onset T2DM, whereas government hospitals mostly deal with T1DM. This is probably a socio-economic issue as the free supply of insulin would attract type 1 patients to government hospitals, whereas the more affluent with obesity-related T2DM would visit private diabetes centers.

In spite of a number of studies describing the prevalence, distribution and possible causes of diabetes, many government and public health planners still remain largely unaware of the current magnitude and in particular the increases in young diabetes and its serious complications. Special efforts must be made to collect data, especially in those countries where diagnosis may be missed.

REFERENCES


