Review Article

Paediatric Diabetes

Dr.Shriraam Mahadevan*, Dr R Bharath**

*Consultant Endocrinologist, Chennai, India, **Consultant Endocrinologist, Chettinad Super Speciality Hospital, Chennai, India

He completed his training in Internal Medicine and Endocrinology from the prestigious Postgraduate Institute of Medical Education and Research (PGIMER), Chandigarh. He is currently working as Consultant Endocrinologist at Sundaram Medical Foundation, Fortis Malar, Apollo Speciality and Apollo Children's hospitals, Chennai. He is actively involved in academic and clinical research activities and has published several papers in peer reviewed national and international journals. He is also a teaching faculty at Sri Ramachandra Medical College, Porur, Chennai. His special areas of interest include metabolic bone diseases, gonadal disorders, obesity and diabetes.

Corresponding author - Dr.Shriraam Mahadevan (mshriraam@gmail.com)

Abstract

Childhood onset diabetes is encountered more commonly than before. Previously, the main form of diabetes seen in young children was type 1 diabetes characterized by life threatening ketoacidosis without insulin treatment. However, with the alarming rise in childhood obesity worldwide, type 2 diabetes is increasingly seen in children. With advancement in molecular and genetic basis of diseases, other subtypes of diabetes in children which are monogenic in etiology like Maturity Onset Diabetes in Young (MODY) and neonatal diabetes due to sulphonyl receptor mutation responding to oral drugs are also added to the list of different varieties of diabetes seen in children. A brief review of diagnosis and approach to diabetes in young with special focus on various aspects of management of type 1 diabetes is discussed in this article.

Key Words : Diabetes in young, Childhood diabetes, Paediatric diabetes, Insulin therapy

Chettinad Health City Medical Journal 2012; 1(4): 169 - 176

Type 1 Diabetes mellitus

Type 1 Diabetes mellitus (T1DM) is a condition characterized by beta cell destruction resulting in absolute insulinopenia predisposing the affected individuals to ketosis even in basal conditions. Though it can occur at any age, the onset is usually in younger children and hence the previous name juvenile onset diabetes. Since insulin is required for survival it is also aptly called insulin dependent diabetes mellitus (IDDM) though this name is not used anymore.

Pathogenesis

The basic pathophysiology is an immune mediated inflammatory destruction of pancreatic beta cells in a genetically susceptible individual.

Genetics

Clinical studies have shown a greater concordance rate of Type 1 DM among monozygotic than dizygotic twins. Also, the risk of developing T1DM is more among first degree relatives of a proband (5%) compared to overall risk of 0.2 -0.3% among general population. Offspring studies show that the risk is ~3% if the mother has the disease versus 6% if father is affected. Several genetic loci have been identified to have possible role in pathogenesis of T1DM, the leading loci are IDDM1 and IDDM2 on chromosome 6 and 11 respectively.[1]

HLA (Human Leucocyte Antigen) system complex

From population studies, there is a clear association between certain HLA alleles and T1DM, especially HLA DR, DQ and DP loci. The different HLA alleles and their strength of association with T1DM are shown in Table 1.

Table 1: Diabetes risk stratified based on HLA haplotypes

Risk	DRB1	DQA1	DQB1
Low (protective)	1101,1501,0701,1401	0501,0102,0201,0101	0301,0602,0303,0503
Moderate	0401,0403,0101,1601	0301,0101,0102	0301,0303,0501,0502
High	0401,0405,0402,0301,0801	0301,0501,0401	0302,0201,0402

Modified from teaching slides www.barbaradaviscenter.org

Environmental triggers

Discordance in the occurrence of T1DM among identical twins has been a favourable point for reasonable role of environmental triggers in the pathogenesis. From population observations in countries with higher incidence of T1DM there has been a suggestion of possible link between early initiation of cow's milk and T1DM. Possible mechanisms include molecular mimicry like effect between bovine serum albumin and beta cell antigens. Vitamin D deficiency has been associated with occurrence of T1DM mainly from epidemiological observations. Few studies of vitamin D supplementation in early infancy have shown little but promising results in reducing the incidence of T1DM.

Viral infections

Few cases of isolation of Coxsakie B virus from the deceased pancreatic islets and T1DM occurring in patients with congenital rubella link viruses in the pathophysiology. However, exact mechanisms have not been completely elucidated.

Chemical toxins

Nitrosourea compounds, alloxan, streptozocin and pentamidine have been linked with T1DM in some studies, however, only in genetically susceptible individuals.

Immunologic factors

Literature is replete with evidence for immunological mediation of beta cell destruction in T1DM. The immune processes involved in isolated T1DM are significantly different from the beta cell damage process that occurs in T1DM as a part of polyendocrinopathy syndrome. It is generally accepted that the immune destruction is "autoimmune" in origin. However, this too occurs mainly in genetically susceptible individual with possible environmental triggers.

Insulitis

Pathologically, mononuclear infiltration of pancreatic cells is observed near the clinical onset of T1DM. This is similar to the lymphocytic infiltration seen in other autoimmune endocrinopathies as in thyroid. Predominant destruction occurs due to activation of cell mediated immune process – NK cells, lymphocytes and macrophages are mainly involved.

Circulating antibodies

Antibodies to varieties of islet antigens are detected at diagnosis or even prior to onset of T1DM. They may serve as markers of immune damage to beta cells. Earliest antibody to appear is usually anti-insulin antibody and it may help predict rapidity in progression to overt diabetes. Glutamic Acid Decarboxylase (GAD 65) antibody and islet cell antibody are usually present in adult/adolescence onset T1DM. Also, more the number of antibodies present in an individual the risk of progression to T1DM is increased. The sensitivity of various clinically relevant autoantibodies are given in Table 2.Clinically these antibodies especially GAD and ICA are used to confirm underlying autoimmune process and may be helpful in predicting T1DM in the first degree relatives of probands. Immunological intervention early in the course of immune destruction may help abort the damage too. Few interventional studies especially with immunosuppressants like azathioprine or anti CD3 monoclonal antibodies have shown only modest albeit reasonable alteration in the course of the onset of disease.[2] T1DM may also occur in patients who have not been demonstrated to have autoantibodies called Type 1B by ADA classification.

Table 2: Autoantigens and sensitivity in predicting development of Type 1 Diabetes mellitus

Autoantigen	Sensitivity
Insulin	49-92%
GAD (Glutamic acid Decarboxylase)	65-75%
ZnT8 (Zinc transporter 8)	65-75%
ICA512/IA-2 (Islet tyrosine phosphatase)	74%
IA-2β/Phogrin	61%
Carboxypeptidase H	10%

Modified from teaching slides www.barbaradaviscenter.org

Presentation and Diagnosis

Usually typical osmotic symptoms like polyuria, polydipsia and weight loss, fatigue and weakness are the major manifestations of severe hyperglycemia. Significant number of children who are not diagnosed in this phase would end up with life threatening ketoacidosis and may land up in the emergency department with altered sensorium or even coma. The differentiation from type 2 diabetes and other varieties of diabetes in children is not always easy but certain pointers may help. Type 2 DM invariably occurs in an obese child with significant family history of adults with Type 2 DM and signs of insulin resistance (as discussed).

Laboratory evaluation

Classic presentation with osmotic symptoms like polyuria, polydipsia and weight loss with a casual

plasma glucose more than 200mg/dl would suffice to diagnose DM and presence of ketoacidosis strongly suggests T1DM. In other early and milder presentations a fasting and 2hr post glucose challenge test may be required. Other tests which are useful include glycosylated haemoglobin, C-peptide levels (in certain ambiguous situations). Circulating antibodies especially anti-islet cell, GAD 65 and anti-insulin antibodies, if significantly elevated, confirm autoimmunity. In addition, screening for other autoimmune disorders especially thyroid, adrenal and parathyroid dysfunction and celiac disease may be required as T1DM may be a part of polyglandular syndrome. Genetic testing is useful in diagnosing monogenic forms of diabetes like MODY (maturity onset diabetes in young) and are also very helpful in rare neonatal forms of diabetes associated with potassium channel or sulphonylurea receptor (SUR) abnormalities where the diabetes can be managed with oral sulphonylureas instead of insulin.[3]

Management

The main goals of management of T1DM are

- Achievement of near normal blood glucose levels as much as possible
- Avoidance of severe hypoglycaemia
- Ensure normal growth and puberty
- Prevent long-term complications of diabetes

A multidisciplinary team is required to manage these children effectively and should usually include a physician or paediatrician with special interest in diabetes, nutritionist and other specialists as the need may arise.[3,4]

Diet

Principles of diet in children with diabetes should be tailored to the patient's lifestyle and where possible should avoid drastic changes. Children should be encouraged to eat regular meals containing complex carbohydrates, to reduce refined sugar intake and to increase dietary fibre content. No particular food should be considered forbidden as this may lead to disturbed attitudes to food. Furthermore, to deprive children of some foods such as sweets which their friends consume may be psychologically damaging. Dietary principles may be improved only if the whole family can make similar modifications. Family should also be educated about the dietary treatment of the child experiencing hypoglycaemia or intercurrent illness and dietary management during parties and holidays. Timing of meals and snacks need to be discussed based on the insulin regime. For example, children receiving twice daily injections of premixed (regular + NPH) usually require a snack in between meals to avoid hypoglycaemia. On the other hand, patients on ultrashort acting analogues based basal bolus regime may not require a snack.[2,3]

Dietary composition

It is recommended that approximately 50% of dietary energy intake should be derived from carbohydrates, 35% from fat (mainly mono- and polyunsaturated fats), and 15% from proteins. Basically the approach is to allow the child to choose from certain number of carbohydrate containing foods ('portions') from a list of such foods, at each meal and snack time based on "healthy eating" principles and to involve the whole family. Carbohydrate exchange principles are also taught to the family. Recently, stress is on carbohydrate counting which may be easily done in the developed world.

Exercise

Children should be encouraged to involve in consistent daily physical activity.Supervisory personnel at school or at a park must be made aware of the presence of a child with diabetes and be provided with a source of quick acting carbohydrate to manage hypoglycaemia should it occur. Short burst activity would usually require extra carbohydrate intake just before or just after the exercise to accommodate for the increased demand for calories. However prolonged aerobic activity may result in delayed hypoglycaemia due to accentuation of insulin effect several hours afterward. In the latter case, it is preferable to reduce the insulin dose prior to the planned prolonged physical activity and to eat a snack after the exercise.

Insulin treatment

Insulin is lifesaving in T1DM and proper knowledge about various aspects of insulin therapy will go a long way in the long term management of these children. Insulin preparations available in India are shown in Table 3. A number of insulin regimes are available and one need to be aware that it should be flexible and family's needs and wishes also be considered. In resource poor setting like ours, financial background may also need to be kept in mind in selecting the right insulin. In general, the insulin analogues are 2-3 times costlier than conventional insulins.

Following diagnosis, most children would require around 0.5 units of insulin/kg body weight. There is some evidence that early aggressive insulin therapy may "rest" the damaged beta cells and help "induce" remission ("honeymoon") period of early T1DM when the child may go off insulin for some period (even upto 2 years occasionally). Pre-school children (<4yrs) may be very sensitive to rapid acting insulins and possible regimens in this age group may include once or twice daily isophane (NPH) insulin or the long acting analogues (glargine or detemir). With technological advancement in the pen devices for insulin delivery even 0.5 units may be given currently and is very useful especially in infants. In many older children, use of twice daily injections with a mixture of rapid/short acting and intermediate/long acting commonly in a ratio of 30:70 or 25:75 with two thirds of the daily requirement being given at breakfast time helps achieve reasonable glycemic control. This is especially

useful in the first few years of diagnosis or following "honeymoon" period.

In older children and adolescents especially for appropriately motivated children and families use of a basal – bolus regime may be the most appropriate. The latter comprises of a regular insulin or rapid acting analogue every time before meals and NPH or long acting analogue prior to bedtime. The long acting analogues like glargine and detemir which do not have significant peak help avoid nocturnal hypoglycemia. This regime is very flexible and can be adjusted based on the type and quantity of food taken and in a younger child who may eat erratically, the rapid acting analogue with very quick onset of action may be administered immediately after food intake. Children and/or their parents need to be educated to alter the dose of rapid acting insulin in line with the planned dietary intake and any anticipated exercise. Children initially treated in the hospital are less active than those treated at home and most of them will experience a fall in their glucose following discharge.[1,2,5,6.]

Sick day management

Any illness like infection can cause alteration in the blood glucose levels in a diabetic child. Rise in blood glucose levels can occur due to high level of stress hormones promoting gluconeogenesis and insulin resistance. Inadequate insulin levels can cause ketone body formation. If the child has poor oral intake, diarrhoea and vomiting, there is a risk of hypoglycemia. To avoid precipitation of DKA and hypoglycemia, certain rules have to be followed during sick day. The sick day rules include:

- Never stop insulin.
- Frequent blood glucose monitoring i.e every 4-6 hours.
- Check for urine ketones or blood ketones if Home Monitoring of Blood Glucose (HMBG) is high.
- Insulin dose to be increased or decreased based on HMBG.
- Maintenance of adequate hydration.
- Treatment of inter-current illness.

Table 3: Insulin preparations currently available in India and their pharmacokinetics

Type	**Insulin**	**Onset of action**	**Peak**	**Duration of action in hours**
Rapid acting (analogues)	Lispro	5-15min	30-90min	3-5
	Aspart	5-15min	30-90min	3-5
	Glulisine	5-15min	30-90min	3-5
Short acting	Regular insulin	30-60min	2-3h	5-8
Intermediate-acting	Isophane insulin (NPH)	2-4h	4-10h	10-16
Long-acting	Glargine	2-4h	No peak	20-24
	Detemir	2-4h	No peak	16-20
	Degludec **(yet to be introduced in Indian market)**	2-4 hrs	No peak	24 – 48
Premixed	70%NPH+30% Regular	30-60min	Dual	10-16
	50% NPH+50% Regular	30-60min	Dual	10-16
	70%NPA+30%Aspart	5-15min	Dual	10-16
	75%NPL+25%Lispro	5-15min	Dual	10-16
	50%NPL+50%Lispro	5-15min	Dual	10-16

- Recognise the "warning signs" and seek specialist advice if necessary- some of the warning signs include 1) recurrent vomiting, poor oral intake & dehydration 2) Persistent hyperglycemia or hypoglycemia despite insulin dose adjustment 3) Heavy ketonuria or ketonemia 4) Child is exhausted, confused, hyperventilating, dehydrated or having severe abdominal pain.[7]

Monitoring

Glycosylated Hemoglobin (HbA1c), which reflects average glycemic control in the previous 2-3 months helps in assessment of intermediate to long-term control of diabetes. However, day to day adjustment of insulin dosage and modification of dietary pattern can be made only with frequent monitoring of blood glucose. This is most often accomplished by self-monitoring of blood glucose (SMBG) by capillary method. Atleast 3 -4 values at different times of the day is required to adjust the insulin doses especially those on basal-bolus regime to achieve stricter control. The targets of blood glucose and HbA1c of different age groups are shown in Table 4. During the last decade Continuous Glucose Monitoring Systems (CGMS) are available which may be very useful in patients with brittle diabetes to fine tune the insulin regime and diet.[8]

Monitoring and long term follow up of a child with T1 DM

- Detailed fundus examination for retinopathy once the child is 10 years of age or has diabetes for 3-5 years or more
- TSH levels for hypothyroidism every 1- 2 years
- Urine for microalbumin for nephropathy: starting once the child is 10 years of age or after 5 years of diabetes and then yearly.
- Blood pressure measurement at least once or twice a year with appropriate cuff and adjust for the age specific centiles.
- Lipid profile on at least yearly basis especially in patients with history of cardiovascular disease in the family.
- Celiac disease screen with transglutaminase antibodies or endoscopy as clinically indicated.

Psychological support

The diagnosis of diabetes is invariably a shock to the child and family. Psychological and adjustment issues may arise at diagnosis or during adolescence. Clinical psychologists or psychiatrists,diabetes nurses, other parents and local support groups play a vital role in the overall care of diabetic children. Parent and patient support groups may complement office visits. They may be informational, supportive and even therapeutic at times.[1,2,10]

Newer insulin delivery systems

Insulin therapy for diabetes is commonly delivered by vial and syringe. Though this method of delivery is cost-effective, many patients especially children with type 1 diabetes feel this as inconvenient. The use of insulin pens has minimized some of the short-comings associated with the use of vials and syringes. The advantages of using insulin pens are:

- More convenient and easy to transport than vial/syringe.
- Accurate dosing.
- Less pain in injection site due to short, fine needles (5-6mm and 30-32 gauge).
- User-friendly for patients with visual impairment (presence of audible click) and decreased fine motor skills.

Insulin pens have definite advantage in the management of diabetes in school-going children as it can be easily carried by them.[6]

Continuous Subcutaneous Insulin Infusion (CSII)

CSII or Insulin pump is the near physiological insulin delivery system currently available. In this method, insulin is delivered into the subcutaneous tissue at a selected rate using a portable electro-mechanical pump. Either regular insulin or the rapid acting analogues can be used in CSII. Using the insulin pump, a patient can set a continuous delivery of insulin throughout the 24 hours which is called the "basal" infusion and this should be superimposed by meal related insulin "boluses". Different rates of basal insulin infusion can be programmed in the pump based on the diurnal variation in blood glucose levels. All these applications are user friendly and have helped to improve the quality of life of a type 1 diabetic patient by enabling them to have a flexible life style and better glycemic control. However, the enormous cost involved in CSII therapy makes this technology beyond reach for the majority of Indian children with type 1 diabetes.

Alternative insulin delivery systems

It is the dream of almost every type 1 diabetic patient to have his or her insulin dose in a painless non-injectable method. The most widely studied alternative routes of insulin administration include:

Table 4: Blood glucose and HbA1c goals in children with type 1 DM

Age in years	Blood glucose goals (mg/dl)		
	Before meals	Bedtime/overnight	HbA_1c
0-6	100-180	110-200	<8.5%
7-12	90-180	100-180	<8.0%
13-19	90-130	90-150	<7.5%

i) Inhaled insulin ii)Oral insulin and iii)Buccal insulin. Inhaled insulin was about to be introduced in the market when it was withdrawn due to pulmonary complications. Other modes of delivery are still in the experimental stage of development.[9]

Artificial Pancreas

The basic requirement of an artificial pancreas or a closed loop system is that it should be able to sense the blood glucose levels and deliver an accurate insulin dosage based on a standard algorithm. Also, it should be able to sense hypoglycemia, have the ability to interrupt insulin infusion and to inject counter-regulatory hormone such as glucagon. The system should be able to identify the problems like sensor malfunction or block in the insulin cannula and alert the patient/ parents. When a continuous glucose monitoring system (CGMS) and CSII are interconnected by wireless transmission, and operated together, they can be made to work as a closed loop system. A fully implantable artificial pancreas system is being investigated and the short term results are providing hope for a better set of type 1 diabetic children with affordability.

Future directions

Pancreatic transplantation, Islet replacement therapy and Stem cell therapy are under different phases of evolution. These therapies, if successful, may provide a hope for complete resolution of type 1 diabetes.[9]

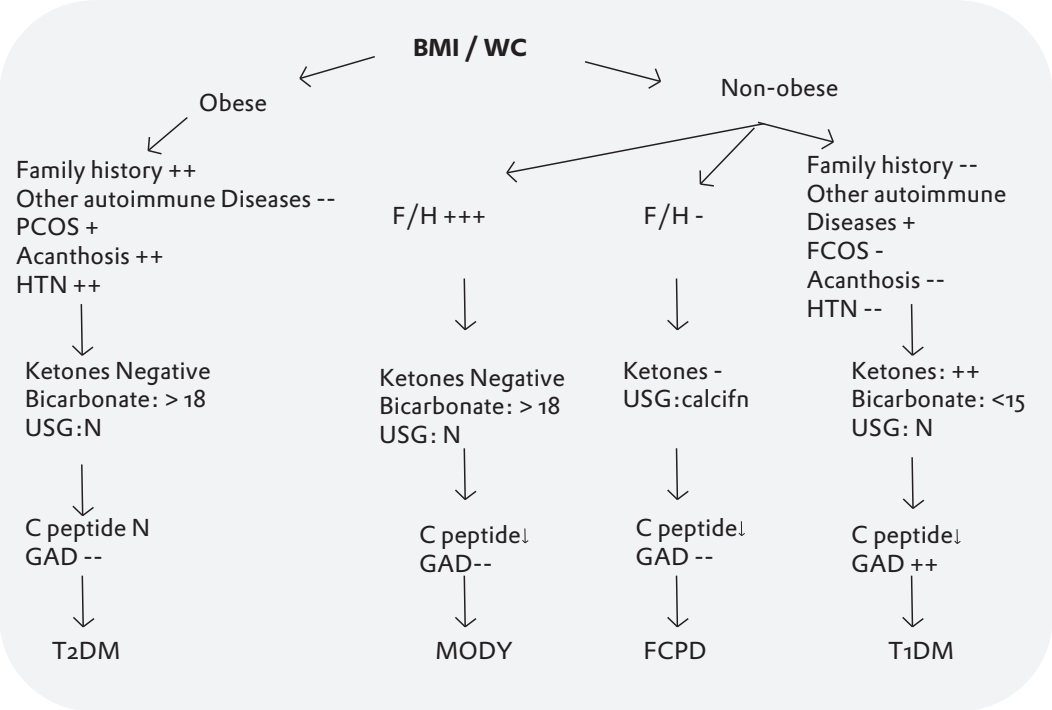
Type 2 DM in children

Cross sectional studies from all the corners of India have shown rise in childhood obesity by several folds consequent to modernization of lifestyle and unhealthy food habits. Disturbingly, in tandem with this rise in childhood obesity an increase in type 2 diabetes in children is also noted. The conditions closely associated with obesity and type 2 DM include hypertension, dyslipidemia, non-alcoholic fatty liver disease and metabolic syndrome, all of which are related to increased risk of cardiovascular disease.[11–13] Pathogenesis of type 2 diabetes is discussed in detail in the relevant section.

Clinical presentation

A small fraction of patients, may be around 10 -20% of children with type 2 DM present with ketoacidosis initially. Contrary to classic type 1 DM the beta cells recover to the extent that they do not require insulin later. This has been called 'flat bush' diabetes in literature. Hyperglycemic hyperosmolar state like in adults with type 2 DM and malignant hyperthermia like syndrome with rhabdomyolysis may also be a very rare presentation. The latter is usually associated with very poor outcome unless diagnosed very early. Typical

Fig1: Approach to Diabetes Mellitus in young



F/H - Family History
GAD - Glutamic acid Decarboxylose
HTN - Hypertension
FCPD - Fibro Calcific Pancreatic Diabetes

BMI - Body mass Index
WC - Waist Circumference
PCOS - Polycystic Ovarian Syndrome
USG - Ultra Sonography

osmotic symptoms may not occur always and one may need to screen all obese children with signs of insulin resistance and family history of diabetes to detect diabetes early. [14,15]

Diagnosis

Usually fasting and 2hr post 75 gm glucose sample is enough to diagnose diabetes. In younger children the glucose load for testing is 1.75 gm/kg (maximum of 75gm). Fasting venous plasma glucose ≥ 126 mg/dl or a 2 hour post glucose challenge value of ≥ 200 mg/dl confirms the diagnosis of diabetes. Clinical features of insulin resistance (central adiposity and acanthosis nigricans), family history of diabetes in close relatives, presence of other features of metabolic syndrome viz. hypertension and dyslipidemia and absence of other autoimmune disorders narrows down the diagnosis to type 2 diabetes mellitus. Absence of ketonuria and acidosis also rules out significant insulinopenia and favours the diagnosis of type 2 DM. Further testing including C-peptide, autoimmune markers like GAD 65 and islet cell antibodies may not be necessary always in all patients. Maturity onset monogenic diabetes (MODY) is suspected when the family history suggests more than 3 generations affected. The latter usually presents with mild hyperglycemia, especially the common MODY type 2 and respond to oral hypoglycemic agents (sulphonylureas). An algorithmic approach to diabetes mellitus in young is given in Figure 1.

When a child should be screened for type 2 diabetes

As in adults, a child who is overweight (BMI more than 90th centile) and features of insulin resistance like acanthosis nigricans and family history of type 2 diabetes should be screened. Obese adolescent girls who present with features of polycystic syndrome like menstrual irregularities, hirsutism and acne also need to be screened.

Management

This is discussed in the relevant section.

Metformin

Of the available oral anti-diabetic agents, not all are approved for Paediatric use except metformin for children with type 2 DM above 12 years of age. The main mechanism of action is decreased hepatic glucose output. Strictly, it is anti-hyperglycemic and also has modest effect on suppressing appetite and promoting weight reduction. Through its significant effect on insulin resistance, it has good effect on improving ovulatory dysfunction in girls with polycystic syndrome. Metformin should be discontinued during administration of radiocontrast agents if a patient has renal dysfunction, hepatic diseases or serious infections. Side effects are usually gastrointestinal disturbances which may improve on continued use and occasionally vitamin B12 deficiency which may need to be supplemented. The usual starting dose of metformin in an adolescent girl is 250 mg oncedaily after dinner for a week or two and then gradually up titrate to a maximum of 2000mg per day in 2 to 3 divided doses.

Insulin

If adequate glycemic control is not attained with good lifestyle measures and metformin then the child should be managed with insulin which is discussed with type 1 DM.

Maturity onset diabetes in young (MODY)

Maturity –onset diabetes of the young is a genetically and clinically heterogenous group of disorders characterized by non-ketotic diabetes. It is inherited in an autosomal dominant manner with onset usually before 25 years of age or in childhood or adolescence. Mutations in atleast[10] different varieties of genes are described under MODY. One of these genes (GCK) encodes the glycolytic enzyme glucokinase; mutations this gene causes MODY 2. The other genes encode transcription factors. Pathophysiologically, the genetic defect results in abnormalities in atleast one of the critical steps involved in insulin secretion resulting in beta cell dysfunction and hyperglycemia. Abnormalities in liver and kidney functions occur in some form of MODY, reflecting expression of the transcription factors in these tissues.

Clinically, MODY usually causes a mild hyperglycemia in a non-obese young patient with significant family history of diabetes in successive generations conforming to autosomal dominant pattern of inheritance. Being a genetic defect the mild hyperglycemia is present from childhood, though it may be picked up only when the patient is evaluated in adulthood. They usually respond to oral hypoglycemic agents and only rarely, a patient may present with rapidly progressing severe hyperglycemia necessitating insulin therapy. Absence of obesity, prominent family history and young age of presentation are few clinical clues which help in differentiating MODY from Type 2 DM.[16]

References

1) Bangstad H-J, Danne T, Deeb LC, Jarosz-Chobot P, et al. Insulin treatment in children and adolescents with diabetes. Paediatric Diabetes 2009;10 (Suppl.12):82–99.

2) Kaufman FR. Intensive management of type 1 diabetes in young children. Lancet. 2005;365:737–8.

3) Donaghue KC, Chiarelli F, Trotta D, Allgrove J, Dahl-Jorgensen K. Microvascular and macrovascular complications associated with diabetes in children and adolescents. Paediatric Diabetes 2009;10 (Suppl. 12):195–203.

4) Rewers M, Pihoker C, Donaghue K, Hanas R, et

al. Assessment and monitoring of glycemic control in children and adolescents with diabetes. Paediatric Diabetes 2009;10 (Suppl.12):71–81.

5) Hirsch IB, Farkas-Hirsch R, Skyler JS. Intensive insulin therapy for treatment of type 1 diabetes. Diabetes Care 1990;13:1265-83.

6) Eesh Bhatia, Ajay Aggarwal. Insulin Therapy for Patients with Type 1 Diabetes. J Assoc Physicians India 2007;55(Suppl):29-40.

7) Brink S, Laffel L, Likitmaskul S, Liu L, Maguire AM, et al. Sick day management in children and adolescents with diabetes. Paediatric Diabetes 2009;10 (Suppl.12):146–53.

8) Hanas R, Adolfsson P. Insulin pumps in Paediatric routine care improve long-term metabolic control without increasing the risk of hypoglycemia. Pediatr Diabetes 2006;7:25–31.
.

9) Shalitin S, Phillip M. The role of new technologies in treating children and adolescents with type 1 diabetes mellitus. Paediatric Diabetes 2007;8 (Suppl.6):72–9.

10) Delamater AM. Psychological care of children and adolescents with diabetes. Paediatric Diabetes 2009;10 (Suppl.12): 175–84.

11) Rosenbloom AL, Silverstein JH, AmemiyaS, Zeitler P, Klingensmith, G. Type 2 diabetes in the child and adolescent. Paediatric Diabetes 2009;10 (Suppl.12):17–32.

12) Amutha A, Datta M, Mohan V. Type 2 diabetes in children – an emerging problem in India. Indian Journal of Practical Paediatrics 2011;13(4):410.

13) A Amutha et al. Clinical profile of diabetes in the young seen between 1992 and 2009 at a specialist diabetes centre in south India. Prim.CareDiab 2011.doi:10.1016/j.pcd.2011.04.003

14) Hamiel O P, Zeitler P.Acute and chronic complications of type 2 diabetes mellitus in children and adolescents. Lancet 2007; 369:1823–31.

15) Amutha A, Datta M, Unnikrishnan R, Anjana R M, Mohan V.Clinical Profile and Complications of Childhood- and Adolescent-Onset Type 2 Diabetes Seen at a Diabetes Center in South India. Diabetes Technology & therapeutics 2012;14(6):497.

16) Fajans SS, Bell GI, Polonsky KS. Molecular mechanisms and clilnical pathophysiology of maturity-onset diabetes of the young. N Engl J Med. 2001;345:971-80.

Answer to : **Diagnose the Condition**

Discussion

ECG Shows sinus rhythm as evidenced by normal P wave axis and negative P wave in AVR lead. PR interval is normal. Occurrence of P wave is regular. But all the P waves are not followed by a QRS complex. Alternate P wave is not conducted. QRS complex shows RBBB pattern with right axis deviation.

Final diagnosis – MOBITZ TYPE 2 AV BLOCK WITH RBBB.

- Dr. M.Chokkalingam, Consultant Cardiologist, CSSH.