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REVIEW ARTICLE

Childhood Obesity and Type 2 Diabetes Mellitus

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ABSTRACT. Until recently, the majority of cases of diabetes mellitus among children and adolescents were immune-mediated type 1a diabetes. Obesity has led to a dramatic increase in the incidence of type 2 diabetes (T2DM) among children and adolescents over the past 2 decades. Obesity is strongly associated with insulin resistance, which, when coupled with relative insulin deficiency, leads to the development of overt T2DM. Children and adolescents with T2DM may experience the microvascular and macrovascular complications of this disease at younger ages than individuals who develop diabetes in adulthood, including atherosclerotic cardiovascular disease, stroke, myocardial infarction, and sudden death; renal insufficiency and chronic renal failure; limb-threatening neuropathy and vasculopathy; and retinopathy leading to blindness. Health care professionals are advised to perform the appropriate screening in children at risk for T2DM, diagnose the condition as early as possible, and provide rigorous management of the disease. ***Pediatrics*** **2005;116:473–480;** ***obesity, type 2 diabetes mellitus, children, insulin resistance, prediabetes, oral hypoglycemic agents, insulin.***

ABBREVIATIONS. T2DM, type 2 diabetes mellitus; T1DM, type 1 diabetes mellitus; A1C, glycosylated hemoglobin.

The extent of the epidemic of childhood obesity is increasing such that children are suffering chronic complications that were once only seen in adults. The purpose of this review is to (1) explain the relationship between adiposity and glucose metabolism and (2) present strategies for screening, diagnosis, and management of children and adolescents with obesity and type 2 diabetes mellitus (T2DM).

WHAT IS THE PREVALENCE OF OBESITY AND T2DM AMONG CHILDREN?

During the past 30 years, the number of children diagnosed as being overweight has increased by >100%.[1] The 1999–2002 National Health and Nutrition Examination Survey data indicate that 22.6% of 2- to 5-year-olds and 31% of 6- to 19-year-olds in the United States are "at risk for overweight" as defined by a body mass index (BMI) between the 85th and 95th percentiles for age.[2] The prevalence of "overweight," defined as a BMI of ≥95th percentile for age, was 10.3% in 2- to 5-year-olds and 16% in 6- to 19-year-olds. By contrast, between 1988 and 1994, an average of 11.3% of 6- to 11-year-olds and 10.5% of 12- to 19-year-olds were overweight.[3]

Children from racial minority groups suffer disproportionately. The prevalence of overweight among children 2 to 5 years of age was 8.6% in non-Hispanic white children, 8.8% in non-Hispanic black children, and 13.1% in Mexican American children. Among 12- to 19-year-olds, significantly more non-Hispanic black and Mexican American adolescents were overweight (23.6% and 23.4%, respectively) compared with non-Hispanic white adolescents (12.7%). Unfortunately, overweight children and adolescents are likely to become overweight adults.[4] Overweight and obesity are associated with serious medical, psychological, and social problems throughout the lifespan.[5,6]

Overweight or obesity is the most important risk factor for the development of T2DM in youth. Indeed, the increasing prevalence of overweight closely parallels the rise in the number of cases of T2DM.[7–9] T2DM now accounts for a considerable proportion of newly diagnosed cases of diabetes (as many as 50% of cases in some clinics) in the pediatric population.[8,10] A retrospective diabetes clinic–based study from the greater Cincinnati, Ohio, area revealed that the incidence of T2DM among children and adolescents (≤19 years of age) increased 10-fold between 1982 and 1994 (0.7 vs 7.2 per 100 000 per year).[11] All of the newly diagnosed children with T2DM in this study were overweight (mean BMI: 37.7 ± 9.6 kg/m^2) and had significant family histories of T2DM. Similar results have been reported elsewhere.[9]

Increasing rates of T2DM among children and adolescents will have considerable long-term implications for the affected individuals, society, and the public health system as a whole.[11–13] Earlier onset of T2DM leads to earlier onset of complications including progressive neuropathy, retinopathy leading to blindness, nephropathy leading to chronic renal failure, and atherosclerotic cardiovascular disease leading to stroke, myocardial infarction, and (in some cases) sudden death. In addition to their impact on physical well-being, the economic, social, and psychological impact of these conditions is enormous.[5]

WHAT IS THE PATHOPHYSIOLOGY OF T2DM AMONG CHILDREN?

Insulin resistance, which develops as a result of both genetic and environmental factors, is strongly

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associated with obesity. Moreover, insulin resistance is now widely believed to be the first step in the development of T2DM, cardiovascular disease, and other conditions.[14–18] The term "metabolic syndrome," otherwise referred to as syndrome X or the insulin-resistance syndrome, was coined by Reaven[19] in reference to the constellation of metabolic abnormalities including insulin resistance, glucose intolerance and T2DM, hypertension, and dyslipidemia, which promote long-term cardiovascular complications.

Genetic and environmental factors influence the development of insulin resistance and T2DM. Puberty is also associated with a decrease in insulin sensitivity. Puberty is associated with an increase in secretion of growth hormone, which in turn promotes a transient state of physiologic insulin resistance.[20–23] Puberty, therefore, is a vulnerable time at which hormonal changes together with a genetic predisposition and environmental factors can tip the balance from insulin resistance to frank diabetes.

Effects of Obesity on Insulin Sensitivity

Glucose homeostasis is maintained by insulin secretion, insulin action, hepatic glucose production, and cellular glucose uptake.[24] Insulin receptors in the liver, muscle, and adipose tissue are normally exquisitely sensitive to insulin. During the absorptive (fed) state, insulin secreted in response to rising blood glucose concentration inhibits hepatic glucose production and stimulates glucose disposal, primarily in muscle. During the postabsorptive (fasting) state, insulin secretion decreases to basal levels, inhibiting hepatic glucose production to a lesser degree to maintain normal fasting blood glucose concentrations.

In the presence of increased adiposity, the initial metabolic abnormality in the pathway toward glucose intolerance is insulin resistance. Insulin resistance is the diminished ability of insulin-sensitive tissues to respond normally to insulin at a cellular level because of genetic, metabolic, and nutritional perturbations. Visceral adiposity promotes insulin resistance to a higher degree than subcutaneous adiposity.[18] Early in the pathogenesis of glucose intolerance, insulin-producing pancreatic β cells are able to compensate for the cellular insulin resistance by increasing insulin secretion. This compensatory hyperinsulinemia is able to maintain blood glucose levels in the normal range.

Progression From Insulin Resistance to T2DM

Insulin sensitivity and insulin secretion are inversely and proportionately related. The lower the insulin sensitivity (ie, the greater the insulin resistance), the more insulin that is secreted. The product of insulin sensitivity and insulin secretion is a constant referred to as the glucose-disposition index.[25] If insulin sensitivity decreases, pancreatic β-cell insulin secretion must increase to maintain the same glucose-disposition index in an individual. At a certain point, this compensatory β-cell response fails and the glucose-disposition index decreases.[26–28] The failure of the pancreatic β cell, resulting in insufficient insulin secretion, underlies the transition from insulin resistance to clinical diabetes. Consequently, the beginning of the disease process is silent, evading medical efforts to intervene until there is deterioration of pancreatic β-cell function and need for therapy with medications and/or insulin.

As is in patients with type 1 diabetes mellitus (T1DM), the lack of insulin in patients with T2DM can lead to ketoacidosis. Indeed, T2DM first presenting with ketosis has become common among adolescents, particularly black and Hispanic adolescents.[29] Such patients are insulin resistant with acute, severe defects in insulin secretion that are not immune mediated.[30] After the institution of therapy with insulin, some endogenous insulin-secretory capacity may be recovered. It is not known if ketosis-prone T2DM is different in etiology from non–ketosis-prone T2DM. Genetic pancreatic β-cell defects, however, are believed to predispose to the development of insulinopenia and ketosis.[31]

WHAT CONSTITUTES A COMPREHENSIVE APPROACH TO THE EVALUATION OF THE OVERWEIGHT/OBESE CHILD FOR T2DM?

Identification of Overweight, Impaired Glucose Tolerance, and Diabetes in Children

Genetic and environmental risk factors such as maternal obesity, gestational diabetes, and lack of physical activity can and should be identified at an early age.[32–36] BMI should be plotted by health care providers annually on the Centers for Disease Control and Prevention BMI growth charts, specific for age and gender, for all children in their care. Age-, gender-, and ethnicity-specific data for waist circumference can be used as an indicator of visceral distribution of fat.[37] Counseling to promote weight loss through lifestyle modification should be offered to all children identified as being at risk for overweight or being overweight.

Screening of Individuals at Risk for T2DM

T2DM is often asymptomatic. Risk factors for T2DM include overweight and obesity, and signs of insulin resistance including acanthosis nigricans, precocious puberty, hypertension, dyslipidemia, and polycystic ovary syndrome (Table 1).[12] The American Diabetes Association recommends screening for diabetes among children with a BMI of $\geq$85th percentile for age and gender, with 2 additional risk factors for T2DM (Table 1). There is evidence to indicate that complications of diabetes frequently begin before symptoms appear. Findings of microangiopathic damage in newly diagnosed patients indicate that such damage predates the onset of clinical diabetes.[38–40] Indeed, autopsy studies reveal that atherosclerotic vascular change is prevalent among children and the extent of atherosclerosis is correlated with risk factors such as BMI and lipid levels.[41] Aggressive treatment has been shown to retard the development of complications. Early identification of children with T2DM, therefore, holds the promise of preventing serious complications.

TABLE 1. Testing Guidelines for T2DM

Criteria*
Overweight or at risk for overweight
BMI >85th percentile for age and gender; or
Body weight for height >85th percentile; or
Body weight >120% of ideal for height
Plus any 2 of the following:
Risk factors
Family history of T2DM in first- or second-degree relatives
Race/ethnicity (American Indian, black, Hispanic, Asian/Pacific Islander)
Signs of insulin resistance or conditions associated with insulin resistance (acanthosis nigricans, hypertension, dyslipidemia, polycystic ovary syndrome)
Age of screening initiation
10 y or at onset of puberty if puberty occurs at a younger age
Frequency of testing
Every 2 y
Test
Fasting plasma glucose†

* Clinical judgment should be used to test for diabetes in high-risk patients who do not meet these criteria.
† Fasting plasma glucose is the test recommended by the American Diabetes Association. Alternative tests include casual plasma glucose and oral glucose-tolerance test.
Adapted from American Diabetes Association.[12]

Diagnosis of T2DM

Well-known standards for the diagnosis of T2DM are based on values of fasting blood glucose, random blood glucose, and the oral glucose-tolerance test and are identical for adults and children.[14] Normal fasting plasma glucose is <100 mg/dL (6.11 mM). Patients with fasting levels between 100 and 125 mg/dL have impaired fasting glucose. Patients with fasting levels ≥126 mg/dL have diabetes. Two elevated readings on 2 separate days are needed to make a diagnosis. A random or "casual" plasma glucose value ≥200 mg/dL is diagnostic of diabetes if the patient has additional symptoms such as polyuria (Table 2). During an oral glucose-tolerance test, a 2-hour plasma glucose value of <140 mg/dL is considered normal, ≥140 and <200 mg/dL is considered impaired glucose tolerance, and ≥200 mg/dL is diagnostic of diabetes.

Early in the evaluation of a new patient with diabetes mellitus, it is important to distinguish between T1DM and T2DM to optimize therapy. Clinical signs helpful in distinguishing T2DM from T1DM are obesity and signs of insulin resistance (acanthosis nigricans, hypertension, polycystic ovary syndrome). Patients with T2DM frequently have elevated C-peptide levels. The absence of autoantibodies to insulin, the islet cell, and/or glutamic acid decarboxylase is also typical in most (but not all) cases of diabetes that are classified as T2DM.[42,43]

WHAT ARE THE TREATMENT OPTIONS FOR THE CHILD WITH IMPAIRED GLUCOSE TOLERANCE OR T2DM?

Lifestyle Modification

Two recent randomized, controlled clinical trials on the prevention of diabetes among adults have demonstrated the benefits of lifestyle intervention on the prevention of progression from impaired glucose tolerance to T2DM.[44,45] The Diabetes Prevention Program[44] demonstrated that among adult patients with impaired glucose tolerance, over a 3-year period, a low-fat diet in combination with 150 minutes per week of exercise reduced body weight by 5% to 7% and the risk of developing T2DM by 58% compared with no lifestyle intervention. Metformin also reduced the risk of developing T2DM, although less dramatically (31% reduction compared with placebo group). A 58% reduction in progression from impaired glucose tolerance to T2DM was also demonstrated in a Finish study[45] comparing individuals with impaired glucose tolerance who received aggressive lifestyle intervention (individualized behavioral counseling, low-fat diet, and physical activity) with those who did not. Others have shown that lifestyle changes that promote a modest weight loss of ~10% of body weight improve lipid profiles and insulin sensitivity.[46] Unfortunately, the impact of such interventions on children has yet to be studied rigorously.

Weight loss and/or prevention of weight gain is the best way to prevent T2DM among children with risk factors for the disease.[34] The American Academy of Pediatrics recommends supporting breastfeeding, promoting healthy eating habits and physical activity, and discouraging sedentary activities such as watching television.[35] In 1998, The Maternal and Child Health Bureau, Health Resources and Services Administration, and Department of Health and Human Services convened the Expert Committee for Obesity Evaluation and Treatment.[34] Its recommendations include screening for family readiness for change, education regarding the medical complications of obesity, and family involvement with treatment. The committee's long-term goals for physical well-being include achieving and maintaining a more healthy body weight, developing cardiopulmonary fitness via regular physical activity, and avoidance of smoking. Unfortunately, evidence for the long-term effectiveness of obesity-treatment and -prevention programs among children is scarce. The most promising approaches to prevention involve schools and families.[47] Some programs have been effective in improving knowledge of healthy life-

TABLE 2. Plasma Glucose Criteria for the Diagnosis of Impaired Glucose Tolerance and Diabetes

Plasma Glucose	Normal	Impaired	Diabetes
Fasting	<100 mg/dL	100–125 mg/dL (IFG)	≥126 mg/dL
Oral glucose-tolerance test, 2 h PG	<140 mg/dL	140–199 mg/dL (IGT)	≥200 mg/dL
Casual			≥200 mg/dL + symptoms*

IFG indicates impaired fasting glucose; 2 h PG, plasma glucose at 2 hours postingestion of glucose; IGT, impaired glucose.
* Polyuria, polydipsia, weight loss

styles but have failed to have an impact on the prevalence of obesity.[48] Despite the lack of successful obesity-prevention and -treatment programs, aggressive lifestyle modification is widely recommended for all children who are at risk for overweight or are overweight, have risk factors for T2DM, have impaired glucose tolerance, or have already been diagnosed with T2DM.

Most American children consume too many highly processed, high-fat, or sweetened foods and too few fruits and vegetables.[49] Physicians should encourage a healthier diet.[50] Calories from sweetened beverages should be eliminated from the diet entirely. The "stoplight" or "traffic-light" diet approach for children, described by Epstein et al,[51] is a useful paradigm to help children and families understand which foods should be consumed in which amounts. "Red foods" such as potato chips should be avoided except on rare occasions. "Yellow foods" are moderate in calories and include whole-grain carbohydrates and fruits. These should be consumed in moderation. Green foods include most vegetables and should be eaten as much as possible. Obesity and diabetes disproportionately affect children from certain minority groups, including black and Mexican American children. Dietary recommendations based on the traffic-light paradigm, therefore, should take cultural food preferences into account. Encouraging healthy eating habits among the parents of at-risk-for-overweight and overweight children is another effective implementation strategy.[51–53]

Physicians should promote increased physical activity and reduced sedentary activity. Exercise programs should be carefully adjusted to the needs of a growing child. Aerobic exercise (swimming, bicycling, walking) for at least 30 minutes per day should be encouraged, with gradual increases in the frequency, intensity, and duration of exercise according to each individual's fitness level and goals. Limiting the time spent on sedentary behaviors such as television viewing has been shown to be an effective way to both increase physical activity and help maintain or achieve a healthy weight.[54,55] Television promotes obesity by displacing physical activity from a child's routine and also because a large proportion of food advertising on television targets children. Robinson et al[54] introduced a 6-month classroom curriculum to decrease television and videotape viewing and video-game use to 198 3rd- and 4th-grade students at 2 public schools in San Jose, California. Reduction in the amount of time spent watching television and playing video games, together with reduced food consumption during television watching, was significantly correlated with declines in BMI, triceps skinfold thickness, waist circumference, and waist-to-hip ratio. In a study among Mexican children, the risk for becoming overweight increased by 12% for each 1-hour increment in daytime television viewing and decreased by 10% for each daily hour of moderate or intense exercise.[55]

In addition to lifestyle interventions, some physicians have prescribed metformin to promote weight loss among overweight children. At best, the use of metformin has been associated with very modest weight loss in blinded, randomized, controlled clinical trials.[56] Although metformin has been shown to effectively reduce the rate of progression from prediabetes to T2DM,[44] although not as well as lifestyle modification, it is not clear that metformin alone is efficacious in the treatment of obesity. More research is indicated to establish the range of potential uses of metformin in children.

Medical Treatment of Children With T2DM

Currently, the initial medical management of children with confirmed T2DM depends on the severity of the clinical presentation (Fig 1). The effectiveness of lifestyle modification may be limited, but so are its risks. For this reason, lifestyle changes are always indicated in patients with T2DM. Patients presenting with mild hyperglycemia (126–200 mg/dL) and glycosylated hemoglobin (A1C) $< 8.5\%$ or an incidental diagnosis of T2DM can be treated initially with therapeutic lifestyle changes in combination with metformin, the only drug approved by the Food and Drug Administration for pediatric patients with T2DM. Metformin, a biguanide, decreases hepatic glucose production and increases insulin-mediated glucose uptake in peripheral tissues, primarily muscle tissue.[57,58] A child who presents with severe hyperglycemia (>200 mg/dL), A1C $> 8.5\%$, and/or ketosis should be treated initially with insulin to achieve metabolic control. Metformin is prescribed to nonketotic patients at a low dose (500 mg twice a day or 850 mg once a day, given with meals) and increased as tolerated (in increments of 500 or 850 mg every 2 weeks, up to a total of 2000 mg per day) (Fig 1). Metformin is associated with disturbances in the gastrointestinal tract and, on rare occasions, with lactic acidosis.[59] A modest amount of weight loss is a desirable side effect. Metformin should not be given to a child with T2DM and ketosis, because it may precipitate lactic acidosis. It should be started, however, once the child recovers from ketosis after treatment by rehydration and with insulin. Insulin should be added whenever glucose control cannot be achieved after 3 to 6 months of metformin therapy. Growing evidence in adult patients suggests that the early introduction of insulin therapy improves long-term glucose control, possibly reversing to some degree the damage imparted by hyperglycemia on β cells and insulin-sensitive tissues.[60] Fig 1 provides a working algorithm for the management of youth with T2DM based on our current knowledge and available and approved therapies.

Other Oral Agents

Sulfonylureas (glimepiride, glyburide, and glipizide [second-generation agents]) and meglitinides (repaglinide and nateglinide) are insulin secretagogues that exert their effect in the presence of glucose (Table 3).[61,62] Sulfonylureas are associated with hypoglycemia and weight gain,[63,64] which can be particularly troublesome for children and adolescents. The thiazolidinediones (rosiglitazone and pioglitazone) reduce glucose production by the liver and increase glucose uptake by muscle, reducing the availability of glucose precursors for hepatic glucose

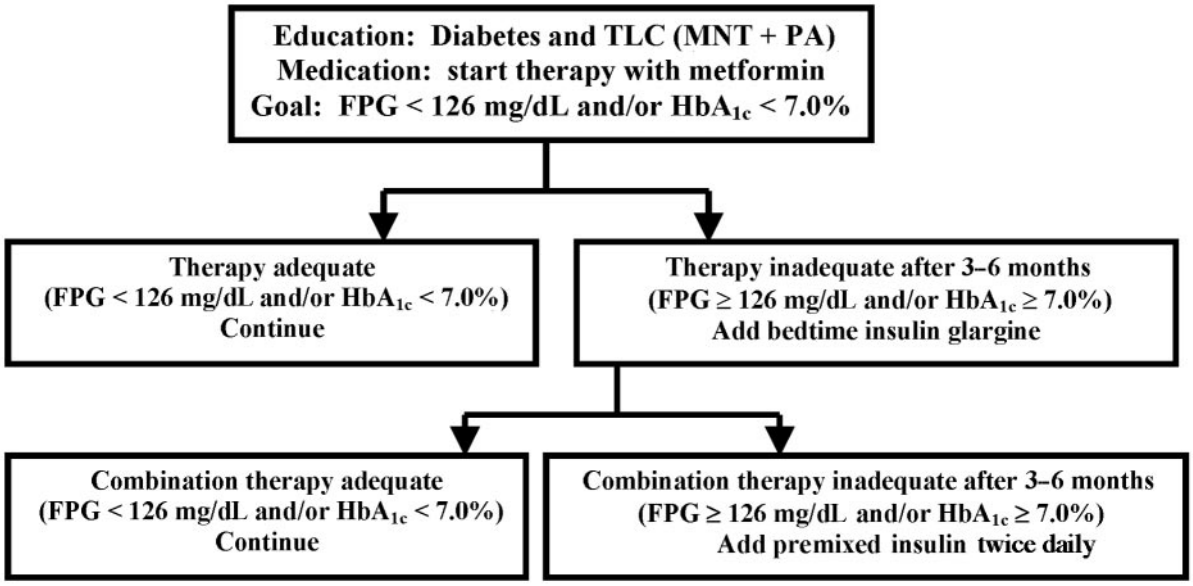

Fig 1. Decision tree for the treatment of T2DM in children. FPG indicates fasting plasma glucose, TLC; therapeutic lifestyle change; MNT, medical nutrition therapy; PA, physical activity. (The figure was modified, with permission, from refs 10 and 58.

production.[65,66] Thiazolidinediones also inhibit lipolysis in adipose tissue. Edema, weight gain, anemia, and possibly liver damage are major adverse effects of the thiazolidinediones.[67] Liver-function tests (transaminases) should be measured before the initiation of therapy and periodically thereafter in patients taking rosiglitazone or pioglitazone. Liver-function tests should be obtained acutely if symptoms suggestive of hepatic dysfunction occur (nausea, vomiting, abdominal pain, etc).

Currently, 3 clinical trials are under way (one with rosiglitazone, another with meglitinide, and the third with Glucovance [metformin and glyburide combination pill]) in pediatric patients with T2DM. Most of the trials in adult patients with T2DM demonstrate that all 4 classes of agents improve A1C similarly (reduction of 1.0–2.0%).[67] Approval of these drugs for use in children will greatly expand the range of treatment options.

Insulin Therapy

Evidence in adult patients suggests that the early introduction of insulin therapy facilitates glucose control in the long-term, possibly reversing to some degree the damage imparted by hyperglycemia on β cells and insulin-sensitive tissues.[60] Furthermore, rapid deterioration in pancreatic β-cell function occurs in some individuals with T2DM, necessitating early the introduction of insulin to achieve metabolic control.[26,68,69] It is possible that the disease is more aggressive in certain populations, including the young, and serious consideration should be given to starting insulin early. Therapeutic studies are needed to address this question.

Insulin glargine, a newly available insulin analog, is systemically absorbed from the subcutaneous injection site at a slower and more consistent rate than are the other insulin preparations used for basal

TABLE 3. Oral Hypoglycemic Agents Being Studied or Approved for Use in Children

Medication Class	Major Mechanisms of Action	Examples of Drugs	Adverse Effects
Biguanides	Decrease hepatic glucose production	Metformin*	Gastrointestinal upset Lactic acidosis in patients with renal failure and conditions predisposing to hypovolemic dehydration and acidosis
α-Glucosidase inhibitors	Reversible inhibition of gastrointestinal sucrase, glucoamylase, dextrinase, maltase and isomaltase enzymes	Acarbose	Gastrointestinal upset
Meglitinides	Insulin secretagogues	Repaglinide Nateglinide	Hypoglycemia Weight gain
Sulfonylureas	Insulin secretagogues	Glimepiride Glyburide Glipizide	Hypoglycemia Weight gain
Thiazolidinediones	Decrease hepatic glucose production Increase glucose uptake in tissues (muscle) Inhibit lipolysis	Rosiglitazone Pioglitazone	Edema Weight gain Anemia Increased liver enzymes (liver damage)

* Approved for use in children.

insulin supplementation. It has a prolonged duration of action (~24 hours) with a relatively smooth blood concentration profile without a pronounced peak, making it useful as a once-a-day basal insulin.[70] Clinical trials in adults with T2DM have shown bedtime insulin glargine to effectively promote more optimal glycemic control.[71] Similar studies of insulin glargine in children and adolescents with T2DM are needed, although it is already being used.[72,73]

Monitoring Glycemic Control

Children with T2DM, regardless of whether they are receiving insulin treatment, should be educated about diabetes management and routine self-monitoring of blood glucose. Glucose should be monitored frequently, especially when medications are being adjusted, when symptoms of diabetes appear, or during acute illness. Patients should also check urinary ketones with a dipstick at such times. Routine glucose self-monitoring should include both fasting and postprandial measurements. Oral and insulin therapy should be titrated to maintain fasting glucose levels between 70 and 100 mg/dL. A1C should be checked every 3 months. The American Diabetes Association recommends a goal A1C of <7%.[12] The American College of Endocrinology recommends a more stringent goal of ≤6.5%, based on evidence showing that there is no minimum level of A1C at which complications of diabetes and mortality do not occur.[74,75]

Hypertension and Dyslipidemia in Children With Diabetes

Diabetes is frequently associated with the comorbidities of hypertension and dyslipidemia. Height- and age-specific population blood pressure percentiles for boys and girls are available.[76] A systolic and diastolic blood pressure <90th percentile, adjusted for age, gender, and height, is normal. If either the systolic or diastolic blood pressure is between the 90th and 95th percentiles, the child has prehypertension. Systolic or diastolic blood pressure ≥95th percentile indicates stage 1 hypertension. If either the systolic or diastolic blood pressure is >99th percentile plus 5 mm Hg, the child has stage 2 hypertension.

As with T2DM, lifestyle modifications in the form of weight loss, dietary changes, and increased physical activity form the foundation of initial therapy for children with hypertension. Angiotensin-converting enzyme inhibitors, calcium channel blockers, β blockers, and diuretics are acceptable medications in children and should be used to treat hypertensive children who do not respond to lifestyle modification and all children with stage 2 hypertension.

Similarly, therapy for dyslipidemia begins with dietary changes and increased physical activity. Lipid-lowering medications can be added if lipids remain elevated after 6 months of lifestyle modification.[77] 3-Hydroxy-3-methylglutaryl coenzyme A reductase inhibitors (statins) are the most commonly used lipid-lowering agents in pediatric patients. Statins (atorvastatin, lovastatin, pravastatin) are currently indicated for use in boys over the age of 10 and in postmenarchal girls with familial hypercholesterolemia.

Monitoring for Microvascular Complications

In addition to counseling, adjustment of medications, treatment of comorbidities, and monitoring of glucose control, encounters with children with T2DM should include monitoring for signs of microvascular complications. Urinary albumin and renal function should be checked annually. A dilated retinal examination to look for retinopathy should be performed annually by a qualified physician.

A Team Approach

Ideally, the care of a child with T2DM is shared among a physician, diabetes nurse educator, nutritionist, physical-activity leader, and behavioral specialist. Such specialty teams are successful in optimizing therapy and promoting behavioral change. More importantly, conscientious involvement by family members is necessary for children to reach therapeutic goals.[51,77,78]

CONCLUSIONS

The epidemic of pediatric obesity is having a huge impact on the physical and social well-being of today's children. Obesity promotes insulin resistance, which in turn is related to a number of problems including T2DM. Reversing obesity through lifestyle changes is an important step in caring for patients at risk for or diagnosed with T2DM. Helping children achieve or maintain a healthy weight requires accurate identification by health care professionals and promotion of lifestyle modifications. It will also require significant societal change to create a healthier environment for children. Childhood obesity remains a challenging problem, and more effective interventions are desperately needed.

In addition to lifestyle modification, patients diagnosed with T2DM can be treated with metformin and/or insulin. Treatment of comorbid hypertension and dyslipidemia with lifestyle modification and/or pharmacotherapy and monitoring of microvascular complications are also necessary. Specialty teams (when available) provide comprehensive management. As in the case of so many problems, families play a crucial role in all aspects of care. Well-designed interventional trials of treatment modalities (lifestyle modification, oral agents, and insulin therapy) are imminently needed to evaluate therapeutic outcomes in children and adolescents with T2DM. For pediatricians, T2DM is an emerging phenomenon that is in its infancy. A tremendous amount of scientific knowledge will be gained during the next years as significant resources are dedicated to providing state-of-the-art care for children with obesity and T2DM.

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