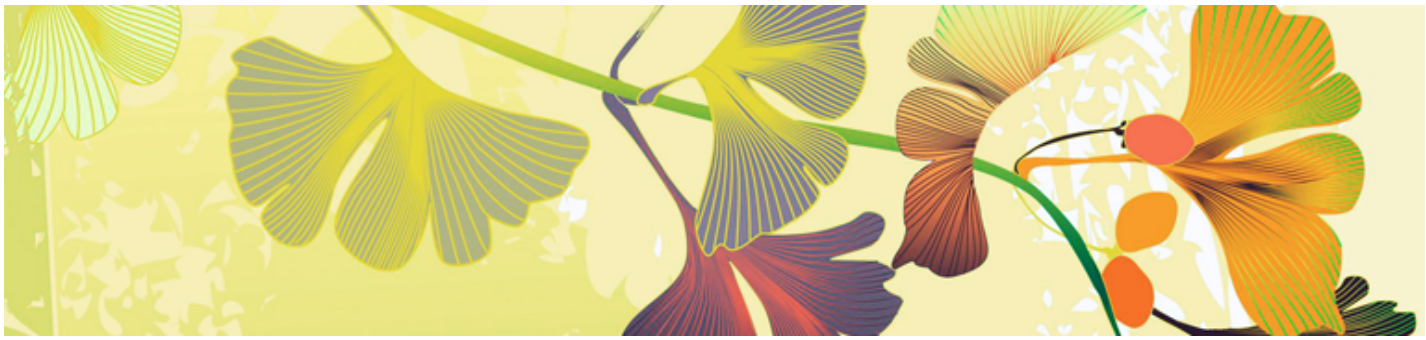

Type 1 Diabetes Mellitus第一型糖尿病(英文)

Preface

Diabetes mellitus refers to the group of diseases that lead to high blood glucose levels due to defects in either insulin secretion or insulin action in the body. Diabetes develops due to a diminished production of insulin (in type 1) or resistance to its effects (in type 2 and gestational).Both lead to hyperglycemia, which largely causes the acute signs of diabetes: excessive urine production, resulting compensatory thirst and increased fluid intake, blurred vision, unexplained weight loss, lethargy, and changes in energy metabolism. The term "type 1 diabetes" has universally replaced several former terms, including childhood-onset diabetes, juvenile diabetes, and insulin-dependent diabetes mellitus (IDDM). Beyond these two types, there is no agreed-upon standard nomenclature. Various sources have defined "type 3 diabetes" as, among others, gestational diabetes, insulin-resistant type 1 diabetes (or "double diabetes"), type 2 diabetes which has progressed to require injected insulin, and latent autoimmune diabetes of adults (or LADA or "type 1.5" diabetes.) There is also maturity onset diabetes of the young (MODY) which is a group of several single gene (monogenic) disorders with strong family histories that present as type 2 diabetes before 30 years of age. Even with the increasing rate of type 2 diabetes, type 1 diabetes accounts for approximately two-thirds of new diagnoses of diabetes in patients ≤19 years of age. The incidence of childhood type 1 diabetes varies based upon geography, age, gender, and family history. In addition, there appear to be environmental factors that increase the risk of developing type 1 diabetes.

Etiology

The cause of type 1 diabetes is still not fully understood. Some theorize that type 1 diabetes is generally a virally triggered autoimmune response in which the immune system's attack on virus infected cells is also directed against the beta cells in the pancreas. The autoimmune attack may be triggered by reaction to an infection, for example by one of the viruses of the Coxsackie

virus family or German measles, although the evidence is inconclusive. In type 1, pancreatic beta cells in the Islets of Langerhans are destroyed or damaged sufficiently to effectively abolish endogenous insulin production. This etiology distinguishes type 1's origin from type 2.

Diagnosis

The diagnosis of type 1 diabetes is usually prompted by recent-onset symptoms of excessive urination (polyuria) and excessive thirst (polydipsia), often accompanied by weight loss. These symptoms typically worsen over days to weeks; about a quarter of people with new type 1 diabetes have developed some degree of diabetic ketoacidosis by the time the diabetes is recognized. Diabetes mellitus is characterized by recurrent or persistent hyperglycemia, and is diagnosed by demonstrating any one of the following:

- Fasting plasma glucose level at or above 126 mg/dL (7.0 mmol/l).
- Plasma glucose at or above 200 mg/dL (11.1 mmol/l) two hours after a 1.75 g/kg (maxima 75 g) oral glucose load as in a glucose tolerance test.
- Symptoms of hyperglycemia and casual plasma glucose at or above 200 mg/dL (11.1 mmol/l).

Clinical manifestation

The classical triad of diabetes symptoms is polyuria, polydipsia and polyphagia, which are, respectively, frequent urination, increased thirst and consequent increased fluid intake, and increased appetite. Symptoms may develop quite rapidly (weeks or months) in type 1 diabetes, particularly in children. Prolonged high blood glucose causes glucose absorption, which leads to changes in the shape of the lenses of the eyes, resulting in vision changes; sustained sensible glucose control usually returns the lens to its original shape. Blurred vision is a common complaint leading to a diabetes diagnosis. Patients may also initially present with diabetic ketoacidosis (DKA), an extreme state of metabolic dysregulation characterized by the smell of acetone on the patient's breath; a rapid, deep breathing known as Kussmaul breathing; polyuria; nausea; vomiting and abdominal pain; and any of many altered states of consciousness or arousal (such as hostility and mania or, equally, confusion and lethargy).

Treatment

Exogenous administration of insulin is designed to replace the deficient hormone of type 1 diabetes mellitus patients and to attain normoglycemia. However, this goal remains elusive because of the difficulty in replicating the minute-to-minute variations of physiologic insulin secretion and the

difference in delivery of exogenous insulin action either subcutaneously or intravenously compared to normal secretion of endogenous insulin directly into the portal vein. The failure of exogenous insulin to completely mimic physiologic insulin secretion results in acute and chronic complications of diabetes.

There are many different insulin preparations and delivery systems available. The selected regimen is individualized for the child and family to fit their lifestyle and optimize compliance while providing glycemic control that meets age-specific goals. Input from the patient, if age appropriate, and the family (e.g., timing of meals and snacks, school/daycare, physical activity) is important to ensure optimal glycemic control and minimize episodes of hypoglycemia. As a result, the types of insulin and regimens used will vary among children and can change for an individual child over time.

Overview

1. Basic understanding — The diabetes team teaches the patient and family the cause and treatment of type 1 diabetes, how to maintain a daily schedule and record of blood glucose test results, insulin administration, and the timing and carbohydrate content of meals and snacks.

2. Insulin administration — Acknowledge the different types of prescribed insulin, how to measure and inject insulin, and how to rotate injection sites. Family members and caretakers must learn about the duration and action of the various types of insulin prescribed for their child. They must also understand how to adjust the insulin dose based upon blood glucose concentrations and carbohydrate intake.

3. Blood glucose testing — Families must master blood glucose testing using a meter that is easy to use. The frequency and timing of monitoring depending upon the needs of their child (usually four times a day: before three meals and before bedtime; at midnight every 2 weeks).

4. Ketonuria — Check urine for ketones at times of illness and/or if the blood glucose concentration is greater than 250 mg/dL (13.9 mmol/L) for two consecutive readings.

5. Hypoglycemia — Families need to recognize the signs and symptoms of hypoglycemia. This is particularly difficult in the non-verbal young child and infant in whom the signs of hypoglycemia are nonspecific. Parents need to check blood glucose level for non-verbal young child or consciousness disturbed patients and, if this is too low, to intervene with dietary measures and/or glucagon.

6. Long-term complications — The relationship between glycemic control and significant long-term sequelae has been firmly established in

randomized controlled trials in adolescents and adults. The Diabetes Control and Complications Trial (DCCT) demonstrated that strict glycemic control delayed the onset of microvascular disease (retinopathy, nephropathy, and neuropathy), slowed progression of already-present microvascular disease, and decreased the incidence of cardiovascular disease. As A1C decreased, so did the risk of long-term sequelae, such as diabetic

7. retinopathy.

若有任何疑問，請不吝與我們聯絡
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