Gestational diabetes may take toll on babies, mums

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DIABETES mellitus (DM) is classified into four broad categories: type 1, type 2 gestational diabetes and "other specific types". The "other specific types" are a collection of a few dozen individual causes. Diabetes is a more variable disease than once thought and people may have a combination of these forms.

Type 1 diabetes

Type 1 DM is characterised by loss of the insulin-producing beta cells of the pancreatic islets, leading to deficiency. This type can be further classified as immune-mediated or idiopathic. The majority of type 1 diabetes is of the immune-mediated nature, in which a T cell-mediated autoimmune attack leads to the loss of beta cells and, thus, insulin.

Type 1 diabetes can affect children or adults, but was traditionally termed "juvenile diabetes" because a majority of these diabetes were in children. "Brittle" diabetes, also known as unstable diabetes or labile diabetes, as a term that was traditionally used to describe the dramatic and recurrent swing in glucose levels, often occurring for no apparent reason in insulin-dependent diabetes.

This term, however, has no biologic basis and should not be used.

Type 1 diabetes is partly inherited, with multiple genes, including HLA (human leucocyte antigen) genotypes, known to influence the risk of diabetes. In genetically susceptible people, the onset of diabetes can be triggered by one or more environmental factors, such as a viral infection or diet. Several viruses have been implicated but, to date, there is no stringent evidence to support this hypothesis in humans. Among dietary factors data suggest that gliadin (a protein present in gluten) may play a role in the development of type 1 diabetes, but the mechanism is not fully understood.

Type 2 diabetes

Type 2 DM is characterised by insulin, which may be combined with relatively reduced secretion.

The defective responsiveness of body tissues is believed to involve the insulin receptor. However, the specific defects are not known.

DM cases due to a known defect are classified separately. Type 2 DM is the most common type of diabetes. Type 2 DM is primarily due to lifestyle factors and genetics. A number of life style factors are known to be important to the development of type 2 DM, including obesity (BMI > 30), lack of physical activity, poor diet, distress and urbanisation.

Excess body fat is associated with 30 per cent of cases in those of Chinese and Japanese descent, 60-80 per cent of cases in those of Europeans and African descent and 100 per cent of Pima Indian and Pacific Islanders. Often those who are not obese often have a high waist-hip ratio.

Dietary factors also influence the risk of developing type 2 DM. Consumption of sweetened drinks in excess is associated with an increased risk. The type of fat in the diet is also important, with saturated fat and trans fats increasing the risk and polyunsaturated and monosaturated fat decreases the risk. Eating lots of white rice may increase the risk of diabetes. A lack of physical activity is believed to cause 7 per cent of cases.

Diabetes in pregnancy

Pregnancy or gestational diabetes (**pic**) resembles type 2 DM in several respects, involving a combination of relatively inadequate insulin secretion and responsiveness. It occurs in about 2-10 per cent of all pregnancies and may improve or disappear after delivery.

However, after pregnancy approximately 5-10 per cent of women with gestational diabetes are found to have DM, most commonly type 2.

Gestational diabetes is fully treatable but requires careful medical supervision throughout the pregnancy. Management may include dietary changes, blood glucose monitoring, and in some cases insulin may be required.

Gestational diabetes though may be transient, untreated can damage the health of the foetus or mother. Risks to the baby includes macrosomia (high birth weight), congenital heart and central nervous system abnormalities and skeletal muscle malformations; increased levels of insulin in a foetus' blood may inhibit foetal surfactant production and cause respiratory distress syndrome.

A high blood bilirubin level may result from red blood cell destruction. In severe cases, potential death may occur, most commonly as a result of poor placental perfusion due to vascular impairment. Labour induction may be indicated with decreased placental function. A caesarean section may be performed if there is marked foetal distress or an increased risk of injury associated with macrosomia such as shoulder dystocia.

Other types of diabetes

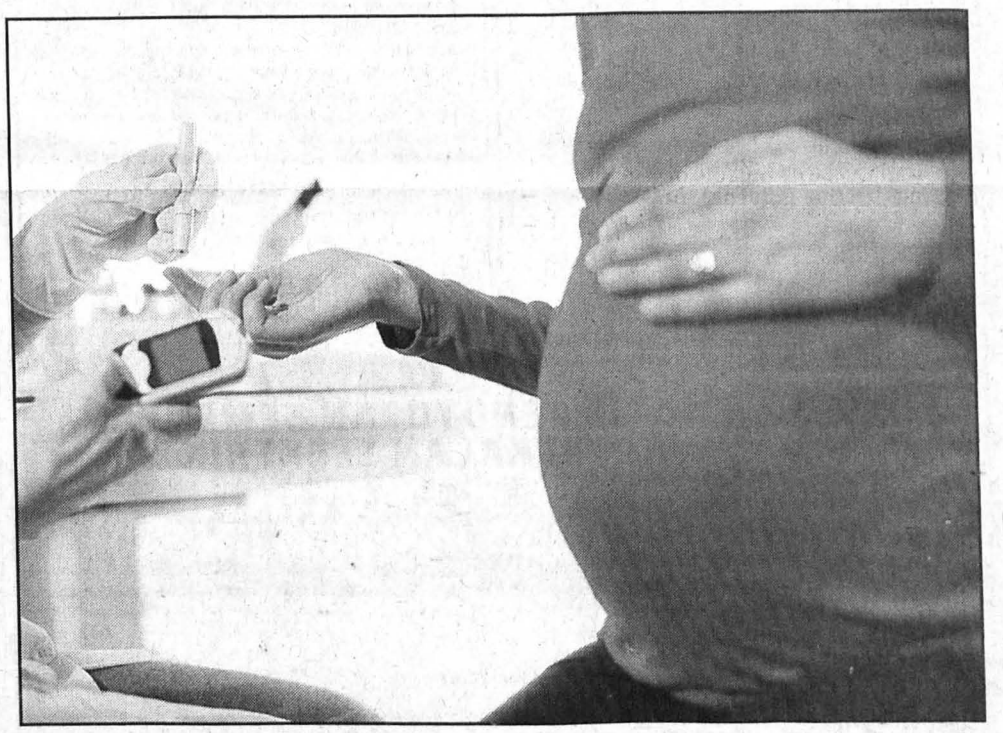
- Maturity onset of diabetes of the young (MODY): MODY is an autosomal dominant inherited form of diabetes, due to one of several single-gene mutations causing defects in insulin production. It is significantly less common than the three main types. The name of this disease refers to early hypotheses as to its nature. Being due to a defective gene, this disease varies in age at presentation and in severity according to the specific gene defect: thus there are at least 13 subtypes of MODY. People with MODY often can control it without using insulin.
- Prediabetic condition: Prediabetic condition indicates a condition that occurs when a person's blood glucose levels are higher than normal but not high enough for a diagnosis of type 2 DM.
- Latent autoimmune diabetes of adults (LADA): LADA is a condition in which type 1 DM develops in adults. Those with LADA are frequently misdiagnosed as having type 2 DM, based on age rather than cause.
- Tissue receptors: Some cases of diabetes are caused by the body's tissue receptors not responding to insulin (even when insulin levels are normal which is what separates it from type 2 diabetes); this form is very uncommon.
- Genetic mutation: Genetic mutations (autosomal or mitochondrial) can lead to defects in beta cell function. Abnormal insulin action may also have been genetically determined in some cases.

Any disease that causes extensive damage to pancreas may lead to diabetes (for example, chronic pancreatitis and cystic fibrosis).

Diseases associated with excessive secretion of insulin-autogonostic hormones can cause diabetes (which typically resolved once hormone excess is removed).

- Drug induced diabetes: Many drugs impair insulin secretion and some toxins damage pancreatic beta cells. The ICD-10 diagnostic entity, malnutrition-related DM (MRDM or MMDM.

ICD 10-cide 12) was deprecated by the World Health Organisation when the current taxonomy was introduced in 1999. Other forms of DM include congenital diabetes, which is due to genetic defects of insulin secretion, cystic fibrosis diabetes, steroid diabetes induced by high doses of glucocorticoid and several forms of monogenic diabetes.



- Type 3 diabetes: Type 3 diabetes has been suggested for Alzheimer's as the underlying process may involve insulin resistance by the brain.

Other causes of diabetes

- Genetic defects of B cell function
- Genetics defects in insulin processing or insulin action
- Exocrine pancreatic defects
- Endocrinopathies
- Infections
- Drugs mainly glucocorticoid, thyroid hormones, beta-adrenergic agnostic
- Statins. Statin therapy is associated with a slightly increased risk of development of diabetes, but the risk is low in absolute terms and when compared with reduction in coronary events. Clinical practice in patients with moderate or high cardiovascular risk or existing cardiovascular disease should not change.

Disease process-pathophysiology

Insulin is the principal hormone that regulates the uptake of glucose from blood into most cells of the body, especially liver, adipose tissue and muscle, except smooth muscle, in which insulin acts via the IGF-1. Therefore, deficiency of insulin or the insensitivity of its receptors plays a central role in all forms of diabetes mellitus.

The body obtains glucose from three main places: the intestinal absorption of food; the breakdown glycogen, the storage form of glucose found in the liver; and gluconeogenesis, the generation of glucose from non-carbohydrate substrates in the body.

Insulin plays a critical role in balancing glucose levels in the body. Insulin can inhibit the breakdown of glycogen or the process of glycogenesis, it can stimulate the transport of glucose into fat and muscle cells, and it can stimulate the storage of glucose in the form of glycogen.

Insulin is released into blood by beta cells (B-cells), found in the islets of Langerhans in the pancreas, in response to rising levels of blood glucose, typically after eating. Insulin is used by about two-thirds of the body's cells to absorb glucose from blood for use as fuel, for conversion to other needed molecules, or for storage.

Lower glucose levels results in decreased insulin release from the beta cells and in the breakdown of glycogen to glucose. This process is mainly controlled by the hormone glucagon, which acts in the opposite manner to insulin.

If the amount insulin available is insufficient, if cells respond poorly to the effect of insulin (insulin insensitivity or insulin resistance) or if insulin itself is defective, then glucose will not be absorbed properly by the body cells that require it, and it will not be stored appropriately in the liver and muscles. The net effect is persistently high levels of blood glucose, poor protein synthesis and other metabolic derangement, such as acidosis.

When the glucose concentration in the blood remains high over time, the kidneys will reach a threshold of reabsorption and glucose will be excreted in the urine (glycosuria).

This increases the osmotic pressure of the urine and inhibits reabsorption of water by the kidney, resulting in increased urine production (polyuria) and increased fluid loss.

Lost blood volume will be replaced osmotically from water held in body cells and other body compartments, causing dehydration and increased thirst (polydipsia).

This is the second in a series on the escalating concerns of Diabetes mellitus. Next week's instalment focuses on the symptoms and treatment of the disease.

About the authors

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