Neonatal diabetes is a condition caused by genetic mutations

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Description

Neonatal diabetes (NDM) is a disease that affects an infant and the infant's body's ability to produce or use insulin. NDM is a monogenic (controlled by a single gene) form of diabetes that occurs within the first 6 months of life. Young children do not produce enough insulin, which increases the accumulation of glucose. It is a rare disease that affects only 1 he in 100,000 to 500,000 people.

The first sign of neonatal diabetes is often slow fetal growth, followed by an abnormally low birth weight. Infants with neonatal diabetes usually show typical symptoms of type 1 diabetes, such as thirst, frequent urination, and dehydration, within the first six months of life. Different types of neonatal diabetes have different onset times. Patients with transient neonatal diabetes tend to develop symptoms in the first few days or weeks of life, with affected children showing signs of weight loss, dehydration, and elevated blood and urinary sugar levels. Some children have elevated blood and urine ketone levels or show signs of metabolic acidosis.

Neonatal diabetes is divided into three subtypes: permanent, transient, and symptomatic. Each has different genetic causes and symptoms. Neonatal diabetes is a genetic condition caused by genetic mutations, either naturally acquired or inherited from parents. At least 30 different genetic mutations can lead to neonatal diabetes. The development and treatment of neonatal diabetes depends on the specific genetic cause. Known genetic alterations have been associated with inhibition of pancreatic or beta-cell development, accelerated beta-cell death due to autoimmunity or endoplasmic reticulum stress, inhibition of glucose recognition or insulin secretion by beta-cells, or the 6q24 region chromosome 6. Most long-term neonatal diabetes is caused by alterations in the ATP-sensitive potassium channel KATP. Disease-associated variants in KCNJ11 and ABCC8, one of the subunits of KATP, cause channels to 'stay open' and prevent β-cells from secreting insulin in response to hyperglycemia.

Diagnosis of neonatal diabetes is complicated by the fact that hyperglycemia is common in new-borns, especially preterm infants, with 25-75% of them having hyperglycemia. Neonatal hyperglycemia usually begins in the first 10 days of life and lasts only a few days. New-borns with diabetes are initially treated with an intravenous infusion of insulin, usually at a dose of 0.05 units/kilogram/hour. Treatment options vary according to the underlying genetic mutations in neonatal diabetic patients. The most common mutations underlying neonatal diabetes, the KCNJ11 and ABCC8 variants, can be treated with sulfonylureas alone and eventually withdraw insulin completely. In many cases, neonatal diabetes can be treated with oral sulfonylureas such as glyburide. A doctor may order genetic testing to determine if switching from insulin to a sulfonylurea is right for a person.

Outcomes in infants or adults with NDM are different in carriers of the disease. Some affected babies have PNDM, some have relapsed diabetes, and some experience permanent remission. Diabetes can recur during the patient's childhood or adulthood. It is estimated that approximately 50% of new-born diabetics will develop TNDM in her. Neonatal prognosis is determined by disease severity (dehydration and acidosis). Associated abnormalities (such as irregular uterine growth and an enlarged tongue) can affect a person's prognosis. It can be confirmed by genetic analysis to find the genetic cause of the disease. With proper treatment, the prognosis for general health and normal brain development is usually good. People with NDM are strongly advised to check their prognosis with their doctor.

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Conflict of Interest

The author has nothing to disclose and also state no conflict of interest in the submission of this manuscript.

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