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Editor Note on Neonatal Diabetes Mellitus

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Description

Neonatal diabetes mellitus is a disease that affects the infant's body's ability to produce or use insulin. It is a monogenic form of diabetes usually diagnosed in the 1st 6 months of life. Not producing enough or required insulin may results in glucose accumulation. It is caused by a change in a gene that effects insulin production. It can be treated by a drug called glibenclamide which causes pancreas to release more insulin. Insulin dependent hyperglycemia that continues longer than a week should take genetic test for neonatal diabetes mellitus.

There are two types of neonatal diabetes:

Transient: this disappears within a year but can come back. The genetic change that causes 6q24-related transient neonatal diabetes mellitus can be inherited in some cases. In most cases, diabetes in childhood is unrelated to type 1 autoimmune diabetes. However, it is possible that it is caused by precocious diffuse autoimmunity, as in the IPEX syndrome (related to mutation in the FOXP3 gene). More than half of transient NDM cases are related to chromosome 6q24 imprinted area abnormalities. Mutations in the glucokinase (GCK) gene cause a small number of permanent cases (leading to complete insensitivity of the -cell to glucose).

Permanent: this stays for life. The affected gene and the underlying pathogenesis determine clinical symptoms (transient versus permanent diabetes mellitus, extra pancreatic features), prognosis, and treatment. Permanent neonatal diabetes mellitus can cause a variety of neurological issues, including developmental delays and recurrent seizures (epilepsy). DEND syndrome is a combination of developmental delay, epilepsy, and neonatal diabetes. Intermediate DEND syndrome is a variant of DEND syndrome that has a milder developmental delay and no epilepsy. A small percentage of people with permanent neonatal diabetes mellitus have a pancreas that is underdeveloped. Affected people experience digestive problems such as fatty stools and an inability to absorb fat-soluble vitamins because the pancreas produces digestive enzymes in addition to secreting insulin and other hormones. When mutations in the KCNJ11 or INS genes cause this condition, it is inherited in an autosomal dominant pattern, which means that one copy of the mutated gene in each cell is enough to cause the disorder. The condition is caused by new mutations in the gene in about 90% of these cases, and it affects individuals who have no family history of the disorder. In the remaining cases, the mutation is inherited from one of the affected parents. When mutations in the ABCC8 gene cause permanent neonatal diabetes mellitus, it can be inherited in an autosomal dominant or autosomal recessive pattern. Both copies of the gene in each cell have mutations in autosomal recessive inheritance. The parents of a person with an autosomal recessive disorder each have one copy of the mutated gene, but they usually don't show any symptoms. K-ATP channels do not close as a result of mutations in the KCNJ11 or ABCC8 genes that cause permanent neonatal diabetes mellitus, resulting in decreased insulin secretion from beta cells and poor blood sugar control.

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