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Commentary

# Commentary on Neonatal Diabetes

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## COMMENTARY

Neonatal diabetes mellitus (DM) is characterized by the onset of persistent hyperglycemia within the first six months of life due to impaired insulin function and is frequently caused by a mutation in a single gene affecting pancreatic beta cell function. Neonatal diabetes mellitus (also termed congenital diabetes or diabetes of infancy) is highly likely to be due to an underlying monogenic defect when it occurs under 6 months of age. Early recognition and urgent genetic testing are important for predicting the clinical course and raising awareness of possible additional features, and in many cases these are essential for guiding appropriate and cost-effective treatment. Additionally, early treatment of sulfonylurea-responsive types of neonatal diabetes may improve neurological outcomes. It is important to distinguish neonatal diabetes mellitus from other causes of hyperglycemia in the newborn. Other causes include infection, stress, inadequate pancreatic insulin production in the preterm infant, among others. Insulin-dependent hyperglycemia that persists longer than a week should raise suspicion for neonatal diabetes mellitus and prompt genetic testing.

Diabetes mellitus most commonly occurs after the neonatal period and results from complex interactions between both environmental and incompletely-penetrant genetic factors. Advances in molecular genetics over the past decade hastened the realization that diabetes that occurs very early life is most often due to underlying monogenic defects — disorders caused by mutation(s) in a single gene. There are over 20 known genetic causes for neonatal diabetes mellitus.

NDM may be categorized by phenotypic characteristics into transient, permanent and syndromic forms. While neonatal diabetes may be recognized within the first few days of life, there are alternative causes of hyperglycemia in neonates, which can make the diagnosis of diabetes difficult. This is especially true in the preterm or low birth weight infant. The prevalence of high glucose levels in preterm infants is 25-75 percent. Neonatal hyperglycemia is more common in the first three to five days after birth, but can be found in infants up to 10 days of life; it usually resolves within two to three days of onset.

Typical causes for hyperglycemia in this group include increased parenteral glucose administration, sepsis, increased counter-regulatory hormones due to stress, and medications such as steroids. There is some evidence of insufficient pancreatic insulin secretion and relative insulin resistance in hyperglycemic and non-hyperglycemic critically ill preterm neonates.

Term infants and premature infants born at > 32 weeks gestational age (GA) are more likely to have a monogenic cause for their diabetes than are very premature infants born at < 32 weeks GA. 5 However, according to the same study, 31 percent of all preterm infants with diabetes born at < 32 weeks GA were diagnosed with a monogenic cause, strongly suggesting that such infants should have genetic testing. These preterm infants also tend to present earlier with diabetes (around 1 week of age) compared to full term infants (around 6 weeks of age). Data gathered from the Monogenic Diabetes Registry at the University of Chicago and others show that patients with transient forms of neonatal diabetes present earlier on average (most often within days of birth) as compared to those with permanent forms.

Neonatal diabetes is often mistaken as type 1 diabetes, which is much more common. But type 1 diabetes usually occurs in children older than 6 months

- Half of babies diagnosed with neonatal diabetes have a lifelong condition. This is called permanent neonatal diabetes mellitus. It occurs in 1 in 260,000 babies in some areas of the world.
- For the other half, the condition disappears within the first twelve weeks of life: but it can reoccur later. This is called transient neonatal diabetes mellitus.

Insulin is acutely required in most infants to establish metabolic control in NDM. Early initiation of sulfonylurea treatment is also recommended as a treatment option in selected cases of NDM caused by mutations, and, in responsive cases, sulfonylurea therapy provides better long-term metabolic control and could even improve neurodevelopmental outcomes.

Specific genes that can cause NDM have been identified. The onset of NDM can be caused by abnormal pancreatic development, beta cell dysfunction or accelerated beta cell dysfunction. Individuals with monogenic diabetes can pass it on to their children or future generations. Each gene associated with NDM has a different inheritance pattern.

In some cases, people with permanent neonatal diabetes mellitus also have certain neurological problems, including developmental

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delay and recurrent seizures (epilepsy). This combination of developmental delay, epilepsy, and neonatal diabetes is called DEND syndrome. Intermediate DEND syndrome is a similar combination but with milder developmental delay and without epilepsy.

A small number of individuals with permanent neonatal diabetes mellitus have an underdeveloped pancreas. Because the pancreas produces digestive enzymes as well as secreting insulin and other hormones, affected individuals experience digestive problems such as fatty stools and an inability to absorb fat-soluble vitamins.

Neonatal diabetes may not always present in the immediate neonatal period. More recent studies show that monogenic forms of NDM may still occur up to 12 months of age, albeit at a reduced frequency. The likelihood of monogenic diabetes causing hyperglycemia in children older than 12 months of age is much lower. Patients may present insidiously (with polyuria, polydipsia, or failure to thrive), acutely (with ketoacidosis or altered mental status), or incidentally without symptoms.

Prognosis and treatment options for monogenic forms of NDM depend heavily on which gene is affected. Advances in genetic testing have allowed for more efficient and comprehensive testing to be readily available.