In addition, genetic interactions could enhance the severity of hemolysis, they are at greater risk of neonatal hyperbilirubinemia even in the environment free from agents that can potentially cause induction of neonatal hyperbilirubinemia in G6PD-deficient neonates [12].

Common genetic disorder in the world. Contact with naphthalene will cause neonatal hyperbilirubinemia with genetic factors, such as glucose-6-phosphate dehydrogenase (G6PD) deficiency [13]. HO and biliverdin reductase might also play roles in the protection of oxidative stress for vulnerable neonates [5,6].

Etiologic verification is essential because the underlying diseases are critical factors of neurological sequelae [7]. There is a wide range of conditions that affect bilirubin levels, including environmental and genetic origins. These events may aggravate the destruction of red blood cells (e.g. cephalohematoma, hemolyisis), delay the metabolism (e.g. prematurity) and increase the absorption of bilirubin (e.g. intestinal obstruction) [8]. Although breastfeeding is a major cause of neonatal hyperbilirubinemia, it does not serve as a risk factor for kernicterus [7,9]. Instead, sepsis carries the greatest risk of poor outcomes.

The most important maternal effect on neonatal hyperbilirubinemia is isoimmune hemolytic disease. The clinical manifestation is early-onset hyperbilirubinemia with anemia [10]. RH incompatibility displays more severe hyperbilirubinemia than ABO incompatibility. With the introduction of Rh immunoglobulin, the incidence of RH isoimmune hemolytic disease has declined. By far ABO incompatibility is the most common cause of isoimmune hemolytic disease.

A large number of evidence has shown a correlation of neonatal hyperbilirubinemia with genetic factors, such as glucose-6-phosphate dehydrogenase (G6PD) deficiency and Gilbert syndrome (a genetic polymorphism of UGT1A1) [11]. G6PD deficiency is the most common genetic disorder in the world. Contact with naphthalene will induce neonatal hyperbilirubinemia in G6PD-deficient neonates [12]. Even in the environment free from agents that can potentially cause hemolysis, they are at greater risk of neonatal hyperbilirubinemia [13]. In addition, genetic interactions could enhance the severity of neonatal hyperbilirubinemia [11,14].

Jaundice persists beyond 14 days of life can be a sign of neonatal diseases [15]. Cholestasis, such as biliary atresia, should be considered. Prompt intervention is essential for identification of biliary atresia that requires early operation. Nevertheless, the vast majority of neonates with prolonged jaundice are associated with breastfeeding. Both environmental and genetic factors are involved in the development of breast milk jaundice [16,17].

Causation of neonatal jaundice carries a geographic difference. Challenge exists in the determination of causation. With an enthusiastic support for breastfeeding, it has become a leading cause of neonatal hyperbilirubinemia. There are complex interrelationships between and within genetic and environmental factors. Clinicians need to develop a systematic approach to identify the possible etiologies in relation to neonatal jaundice.

References


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