**Editorial**
Volume 11:12, 2021

Journal of Blood & Lymph

ISSN: 2165-7831 Open Access

# Autosomal Disorders of Erythrocytes in New Born and Infants

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## Editorial Note

Red blood cell destruction is a common cause of newborn anaemia, and it usually results from immune-mediated mechanisms that are triggered by RBC antigen mismatches between the infant and the mother. Hemolytic Disease of the Foetus and Newborn (HDFN) is a broad term that refers to any foetus or infant who suffers from alloimmune hemolysis produced by maternal antibodies against RBC antigens in the child's circulation. Because of the highly immunogenic nature of the RhD antigen, HDFN induced by anti-RhD antibodies, which occurs in RhD-positive newborns born to RhD-negative mothers, is the most severe variant.

ABO incompatibility, which is most commonly caused by a mismatch between group O women and their non-group O newborns, affects 15% of pregnancies but is usually less severe than Rh illness, with just 4% of incompatible pregnancies terminating in neonatal hemolytic disorder. ABO incompatibility can arise during a woman's first pregnancy, unlike Rh illness, where sensitization occurs in the first pregnancy and HDFN occurs in later pregnancies, since group O mothers have naturally occurring anti-A and anti-B antibodies. A positive DAT in the blood of the newborn and a positive indirect antiglobulin test in the mother are both indicative of HDFN.

Congenital RBC enzyme and membrane abnormalities can cause hemolytic anaemia and jaundice in the neonatal period, in addition to immune-mediated processes of erythrocyte destruction. The erythrocyte membrane is a complex structure containing various important proteins and lipids that result in a circulating biconcave disc that is resilient, flexible, and circulating.

Instability of the RBC membrane, decreased cellular deformability, and shape alterations result from genetic deficits or abnormalities in RBC membrane proteins (e.g., ankyrin, band 3, -spectrin, -spectrin, protein 4.2); aberrant erythrocytes are entrapped in the spleen and removed by macrophages. The most prevalent RBC membrane abnormality is Hereditary Spherocytosis (HS), an autosomal dominant illness characterised by spherical erythrocytes that affects 1 in 2,500-5,000 people of European heritage. Early in the newborn period, over half of all infants born with HS will develop jaundice.

Another autosomal dominant inherited erythrocyte membranopathy characterised by elliptical-shaped erythrocytes, Hereditary Elliptocytosis (HE), is a less prevalent and less severe RBC membrane condition. Hereditary Pyropoikilocytosis (HPP), is on the other hand, is an autosomal recessive RBC membrane condition that causes morphologic shape changes (poikilocytosis) in the peripheral blood smear, some of which mimic thermally injured erythrocytes. HPP is more common in African-American infants, and it can cause severe anaemia and hemolysis in the newborn period.

Because newborns with HPP frequently have a family history of HE and may develop a milder illness resembling HE later in childhood, there is significant clinical and genetic overlap between HPP and HE. A positive family history of hemolytic anaemia raises clinical suspicion for RBC membranopathy, especially in a newborn who exhibits early jaundice in the first 24 hours of life. The diagnostic evaluation should include a negative DAT, indirect hyperbilirubinemia, and hallmark features noted on the peripheral blood smear. Anemia can vary in severity, and reticulocytosis may also be present. Ligongo Asa.

**How to cite this article:** Christian, Joggeli. "Autosomal Disorders of Erythrocytes in New Born and Infants ." *J Blood Lymph* 11 (2022) : e144.

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**Received**: December 21, 2021; **Accepted**: January 04, 2022; **Published**: January 11, 2022