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Neonatal Thrombocytopenia

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Abstract

Thrombocytopenia is one of the most common hematologic problems in the neonate. It affects up to 30% of all patients admitted to the neonatal intensive care unit (NICU). The causes of thrombocytopenia in neonates are diverse and include immune, inherited, and acquired disorders. The evaluation of the neonate with thrombocytopenia and acquired disorders may be challenging. Developing a diagnostic strategy to evaluate the neonate with thrombocytopenia is key for the practicing clinician. Here, we provide a practical approach to the evaluation of the neonate with thrombocytopenia and an overview of its most common etiologies.

Keywords: Thrombocytopenia; Newborn; Treatment

Introduction

Thrombocytopenia is one of the most common haematological problems encountered in the neonatal period presenting in 1-5% of newborns at birth. It is particularly common in newborns admitted to the neonatal intensive care units (NICU) presenting in 22-35% of these neonates. Neonatal thrombocytopenia is defined as a platelet count of less than 150 × 109 per L. This definition is the same for older children and adults as studies have shown that the fetal platelet count is above 150 × 109 per L by the second trimester of pregnancy. The hallmark of platelet disorders is mucocutaneous bleeding however newborns may present more severely with, petechiae, purpura, and intra-cranial hemorrhages [1].

This is the major mechanism underlying neonatal thrombocytopenia. Thrombocytopenia is either present at birth or develops in the first 72 hours of life in 75% of the neonates. Only a small number of these infants have immunological disorders or coagulopathy; the majority of newborns with thrombocytopenia are born prematurely after pregnancies complicated by placental insufficiency and/or fetal hypoxia (i.e., maternal pre-eclampsia and intra-uterine growth retardation of the fetus). These pre-term infants with early-onset thrombocytopenia have impaired megakaryocytopoiesis and platelet production. The megakaryocytes (platelet precursors) and their progenitors are reduced at birth [2].

An increase in platelet consumption and/or sequestration to the spleen and other organs is the mechanism in 25-35% of cases of neonatal thrombocytopenia. Transplacental passage of maternal platelet alloantibodies and autoanitbodies account for 15-20% of thrombocytopenia present at birth. Disseminated intravascular coagulopathy (DIC) associated with perinatal asphyxia and sepsis is responsible for 10-15%, almost always in neonates who are extremely can be identified during assessment of fetuses with ultrasound demonstrating hydrops as a result of congenital infections or aneuploidy. Thrombocytopenia of the fetus may also be discovered following primary diagnostic investigations for either inherited or alloimmune thrombocytopenia [3].

These neonates usually present with hemorrhage and purpura. It is uncommon and usually caused by alloimmune or autoimmune thrombocytopenia but needs to be treated as an emergency due to the risk of hemorrhage.

Sometimes known as isoimmune thrombocytopenia, this is the result of sensitization of the mother to antigens present on fetal platelets during gestation. These antigens are inherited from the father and are thus absent on maternal platelets. The antibodies created then cross the placenta and attack the fetal platelet. The incidence of NAIT is approximately 1 in 1500 pregnancies and is the platelet equivalent of haemolytic disease of the newborn [4].

Suspect NAIT in a thrombocytopenic newborn that is otherwise well, normal maternal platelets, no history of maternal autoimmune disease or ITP. The neonate with NAIT is at risk for intracranial hemorrhage in utero and during delivery [5].

NAIT varies in severity from mild/moderate which typically resolves in the first week of life without sequelae, to severe with extensive intracranial hemorrhage (up to 20% of cases) leading to either death or serious neurological sequelae.

The most common presentations in severe NAIT are petechiae, purpura, and cephalohematoma at birth. The diagnosis depends on demonstrating platelet antigen incompatibility between mom and neonate or mom and father. The most commonly detected antibodies are those directed again human platelet antigen (HPA)-1a (80%) and HPA-5b (10-15%) – this permits prenatal diagnosis in at risk fetuses. This form of neonatal thrombocytopenia occurs in neonates whose mothers have idiopathic thrombocytopenia purpura (ITP) or systemic lupus erythematous (SLE). These mothers carry antibodies directed against platelets. The platelet associated IgG antibody can passively cross the placenta and cause thrombocytopenia in the fetus and the newborn in 10% of cases [6].

The clinical manifestations are less severe than in NAIT; the risk of intracranial hemorrhage is less than 1%, greatest during passage through the birth canal. Most cases usually resolve by 4-6 weeks. All neonates of moms with an autoimmune disease should have a cord blood platelet count determined at birth. Fetal scalp sampling can also be used to measure to fetal platelet count. Fetal scalp sampling can also

be used to measure to fetal platelet count. The platelet count should be repeated for 3-4 days [7]. The maternal platelet count is sometimes a useful indicator of the probability that the infant will be affected [8]. There are no widely accepted guidelines for platelet transfusion in newborns with non-immunologically mediated thrombocytopenia. Recent guidelines are more conservative considering the lack of evidence supporting improved outcomes with platelet transfusion. In general, thrombocytopenic neonates should receive platelets when the degree of thrombocytopenia is such that there is an unacceptable risk of hemorrhage. The following Table 1 gives a summary of when it is appropriate to administer platelets to bleeding and non-bleeding neonates [9].

Platelet count (x 109/L)	Non-Bleeding Neonate	Bleeding Neonate	NAITP (proven or suspected)
<30	Consider transfusion in all patients	Transfuse	Transfuse (with HPA compatible platelets)
30-49	Do no transfuse if clinically stable; Consider transfusion if: • <1000g and <1 week of age • clinically unstable (i.e., fluctuating blood pressure) • previous major bleeding • current minor bleeding (i.e., petechiae, puncture site oozing) • concurrent coagulopathy • requires surgery or exchange transfusion	Transfuse	Transfuse (with HPA compatible platelets if any bleeding)
50-99	Do not transfuse	Transfuse	Transfuse (with HPA compatible platelets if major bleeding) present
>99	Do not transfuse	Do not transfuse	Do not transfuse

Table 1: Neonatal thrombocytopenia: causes and management.

Conclusion

Additionally, it has been shown that the administration of intravenous immunoglobulin (IVIG) before delivery results in increasing fetal platelet counts and may help to reduce thrombocytopenia in cases of NAIT and ITP. Cesarean section delivery is recommended in these neonates to prevent intracranial hemorrhage. There is a high rate of recurrence of NAIT in successive pregnancies and thus antenatal therapy should be offered.

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