Neonatal Thrombocytopenia

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Infant Thrombocytopenia is defined as a decrease of platelet count under 150 000/μL (150×10^9/L). Among healthy mature newborns the incidence of thrombocytopenia is about 1%, it is much more common in newborns with low birth weight. Thrombocytopenia is one of the most common hematologic irregularities among premature newborns, the most common cause in the first 72 hours of life being chronic intrauterine hypoxia.[1]

The production and turnover of thrombocytes in newborns is similar to that in older children and adults. Lifespan of thrombocytes is 7-10 days.[1]

Etiology of Thrombocytopenia

Maternal Causes

The most common cause of thromboctyopenia in the first 72 hours of the prematurely born infants' lives is chronic intrauterine hypoxia based on the insufficiency of placenta during diabetes mellitus or maternal hypertension. Another cause may be the usage of medication such as heparin, thiazide diuretics, quinine, etc. Others include the TORCH infections, disseminated intravascular coagulation (DIC) or the HELLP syndrome. Furthermore, it can be influenced by antibodies against thrombocytes: **autoimmune thrombocytopenia** – antibodies against maternal and fetal platelets, idiopathic thrombocytopenic purpura (ITP), systemic lupus erythomatosus (SLE), the so-called drug-induced thrombocytopenia and **isoimmune thrombocytopenia** – antibodies against fetal platelets: neonatal alloimmune thrombocytopenia (NAIT; usually anti-HPA-1a antibodies), so-called isoimmune thrombocytopenia associated with fetal erythroblastosis.

Placental Causes

Placental causes include chorioangioma, vascular thrombi and the abruption of placenta.

Neonatal Causes

Neonatal causes include:

Lowered platelet production or congenital absence of megacaryocytes (TAR syndrome, Fanconi anemia, rubella, congenital leukemia, trisomy 13, 18, 21 or Turner syndrome, congenital amegacaryocytic thrombocytopenia, methylmalonic, propionic and isovaleric acidemia, etc.)

Increased platelet destruction, which is an increased consumption without an association with a pathological state, where the lowest count is usually lowest before the 4^{th} day, it normalizes before the 10^{th} day of life; bacterial and candida sepsis, congenital infection (TORCH) – especially CMV, thrombosis (renal veins, intracardial, vascular), DIC, IUGR, perinatal asphycia, necrotizing enterocolitis, widespread hemangiomes – Kasabach-Merritt syndrome).

Clinical Manifestation

Severe thrombocytopenia can be accompanied by petechiae (usually < 60 000/μL), gastrointestinal or mucosal bleeding (< 20 000/μL), intracranial bleeding. Widespread ecchymoses and intramuscular bleeding are more likely to accompany coagulopathies instead.[1]

Neonatal Alloimmune Thrombocytopenia (NAIT)

It is caused by active immunization of the mother against the antigens of fetal platelets. These antibodies (IgG class) come through the placenta and destroy the fetal thrombocytes. Sensibilisation can happen in 1% of all pregnant women, but the incidence of symptomatic thrombocytopenia is 2 out of 1.000 living newborns.[2]

Clinical manifestation: severe hemorrhagic diathesis appears with the appearance of petechiae and high incidence of intracranial bleeding. Less than 20×10^9/l appears in the blood count of thrombocytopenia, which leads to an antibody assay. Concerning **treatment**, intravenous immunoglobulins and washed platelet

concentrates of PL^{A1}-negative maternal thrombocytes are being administered.[2]

Autoimmune Neonatal Thrombocytopenia

It is caused by passive transplacental transfer of antibodies, e.g. during idiopathic thrombocytopenic purpura or lupus erythematosus of the mother. **Clinical manifestation**: the thrombocytopenia appears soon after the birth, bleeding ensues. Thrombocytopenia lasts for 2-3 months. It is treated by administering intravenous immunoglobulins and thrombocyte concentrates.[2]

Laboratory Tests

Blood count and blood smear are being done. Blood type, Coombs test, coagulation (PT, APTT, FBG, D-dimer) are being tested. Furthermore, depending on anamnesis, TORCH, hemoculture and swabs, bone marrow, thrombopoetion concentration, megacaryocyte progenitors, proportion of reticulocytes, glycocalicin concentration or immature thrombocyte fraction are being investigated. Maternal blood count test, HPA-1a phenotypisation and anti-HPA-1a antibody assay are being emphasized as well.[1]

Etiology of thrombocytopenia[1]

	Lowered production	**Increased destruction**
Size of thrombocytes	normal	increased (>10,8 fl)
Lifespan of thrombocytes	normal	shortened
Megacaryocytes in bone marrow	less	normal or more
Thrombocyte count after platelet transfusion	increased for 4-7 days	same or slightly increased

Treatment

Primary causes are treated causally. If hemorrhagic symptoms or severe thrombocytopenia (< 20 000/µL) is present, platelet transfusion is administered (10–20 ml/kg, platelets are dissolved in plasma that is AB0- and Rh-compatible with the newborn's erythrocytes). In immune thrombocytopenia, IVIG and prednisone are administered.Template:HVLP.[1]

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