

Thrombocytopenia in the Newborn

Thrombocytopenia in the newborn is defined as a drop in platelet count below $150,000/\mu\text{L}$ ($150 \times 10^9/\text{L}$). In healthy full-term newborns, the incidence of thrombocytopenia is around 1%. It occurs much more often in newborns with low birth weight. Thrombocytopenia is one of the most common hematological abnormalities in premature newborns, in the first 72 hours of life is the most common cause of thrombocytopenia the chronic intrauterine hypoxia.^[1]

Platelet production and turnover in newborns is similar to that in older children and adults. The lifespan of platelets is 7-10 days.^[1]

Ethiology of thrombocytopenia

Maternal causes

The most common cause of thrombocytopenia in the first 72 hours of premature newborns is chronic intrauterine hypoxia due to placental insufficiency in diabetes mellitus or gestational hypertension. Another cause might be the use of some drugs such as heparin, thiazide diuretics, quinine and others. Other causes include TORCH infection, disseminated intravascular coagulopathy (DIC), HELLP syndrome. Antibodies against platelets can also be the cause: **autoimmune thrombocytopenia** – antibodies against maternal and fetal platelets: idiopathic thrombocytopenic purpura (ITP), systemic lupus erythematosus (SLE), so-called drug-induced thrombocytopenia and **isoimmune thrombocytopenia** – antibodies against fetal platelets: neonatal alloimmune thrombocytopenia (NAIT; mostly anti-HPA-1a antibodies), so-called isoimmune thrombocytopenia associated with fetal erythroblastosis.

Placental causes

Placental causes include: chorangioma, vascular thrombi or placental abruption.

Neonatal causes

Neonatal causes include:

Reduced production of platelets or congenital absence of megakaryocytes (TAR syndrome, Fanconi anemia, rubella, congenital leukemia, trisomy 13, 18, 21 or Turner syndrome, congenital amegakaryocytic thrombocytopenia, methylmalonic, propionic or isovaleric acidemia, etc.)

Increased destruction of platelet, which is an increased consumption unrelated to a pathological condition, where the lowest number is by the 4th day, normalizes by the 10th day of life; bacterial and candidal sepsis, congenital infection (TORCH) – especially CMV, thrombosis (renal veins, intracardiac, vascular), DIC, IUGR, perinatal asphyxia, necrotizing enterocolitis, extensive hemangiomas – Kasabach-Merritt syndrome).

Clinical picture

Severe thrombocytopenia can be accompanied by petechiae (usually $< 60,000/\mu\text{L}$), bleeding in the digestive tract and mucous membranes ($< 20,000/\mu\text{L}$) or intracranial bleeding. Extensive ecchymoses and bleeding into the muscles tend to be accompanied by coagulopathy.^[1]

Neonatal alloimmune thrombocytopenia (NAIT)

It arises from the active immunization of the mother against fetal platelet antigens. These antibodies (IgG class) pass through the placenta and destroy the fetal platelets. Sensitization can occur in 1% of all pregnant women, but the incidence of manifest thrombocytopenia is 2/1000 live births.

Clinical picture: a severe hemorrhagic diathesis appears with the formation of petechiae and a high incidence of intracranial bleeding. In the blood count of thrombocytopenia appears less than $20 \times 10^9/\text{L}$, which is used to detect antibodies. In terms of **treatment** i.v. immunoglobulins and washed concentrates of PL^{A1}- negative platelets derived from the mother are given.^[2]



Petechiae to purpura

Autoimmune thrombocytopenia of the newborn

It is caused by the passive transplacental transfer of antibodies, e.g. in idiopathic thrombocytopenic purpura of the mother or lupus erythematosus.

Clinical picture: thrombocytopenia appears soon after birth when the bleeding occurs. Thrombocytopenia lasts for 2-3 months. It is treated with i.v. immunoglobulins and platelet concentrates. ^[2]

Laboratory examination

A blood count and blood smear are performed. The blood group is tested, the so-called Coombs test, coagulation (PT, APTT, FBG, D-dimers). According to the anamnesis, the following are further examined: TORCH, blood culture and smears, bone marrow, concentration of thrombopoietin, megakaryocytic progenitors, ratio of reticulated platelets, concentration of glycocalycin, fraction of immature platelets. Emphasis is also placed on maternal blood count testing, HPA-1a phenotyping and anti-HPA-1a antibody testing.^[1]

Ethiology of thrombocytopenia^[1]

	Reduced production	Increased destruction
Platelet size	normal	enlarged (>10,8 fl)
Platelets lifespan	normal	reduced
Megakaryocytes in the bone marrow	reduced	normal or elevated
Platelet count after platelet transfusion	elevated for 4-7 days	the same or slightly increased

Treatment

Primary causes are treated causally. Platelet transfusion (10–20 ml/kg, platelets dissolved in AB0 plasma and Rh compatible with newborn erythrocytes) is given in case of bleeding manifestations or severe thrombocytopenia (< 20,000/μL). IVIG and prednisone are given for immune thrombocytopenia.^[1]

Links

Related articles

- Bleeding conditions (pediatrics)
- Hemorrhagic disease of the newborn

References

1. GOMELLA, TL. *Neonatology : Management, Procedures, On-Call Problems, Diseases, and Drugs*. 6. edition. Lange, 2009. pp. 695-699. ISBN 978-0-07-154431-3.
2. MUNTAU, Ania Carolina. *Pediatric*. 4. edition. Grada, 2009. pp. 25. ISBN 978-80-247-2525-3.