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Case Report

Severe autoimmune thrombocytopenia in a neonate secondary to maternal immune thrombocytopenia: a case report

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ABSTRACT

Neonatal thrombocytopenia is one of the common haematological problems encountered in neonatal intensive care unit. Severe neonatal thrombocytopenia is defined as a platelet count $<50\times10^3$/µl and is relatively uncommon. Based on the time-of-onset, neonatal thrombocytopenia can be categorized into early-onset (<72 h after birth) and late-onset (>72 h after birth) thrombocytopenia. Neonatal autoimmune thrombocytopenia should be considered in any neonate who has early-onset thrombocytopenia and a maternal history of either immune thrombocytopenia (ITP) or an autoimmune disease (with or without thrombocytopenia). A term male baby, born to a 23-year-old primi-gravida with ITP was found to be thrombocytopenic at birth (platelets-85×10^3/µl) without any sign of neonatal sepsis. On serial monitoring, platelet counts kept falling and on day 3, the child developed severe thrombocytopenia (platelets-6.5×10^3/µl). No obvious signs of bleeding were present and the child was clinically well. Given the history of maternal thrombocytopenia (likely ITP), a possibility of neonatal autoimmune thrombocytopenia was considered. Owing to the risk of massive bleed, the baby was transfused random donor platelets and intravenous immunoglobulin (IVIg) was started on day 3. Thereafter, the platelets showed an increasing trend and child was discharged on day 7 with a platelet count of 170×10^3/µl. However, on follow-up platelet count was again found to be low (84×10^3/µl). It normalised subsequently, without any further requirement of IVIg. High index of suspicion, immediate work-up and diagnosis, with close monitoring and prompt management is required to prevent hemorrhagic complications in such children. Counselling for risk of thrombocytopenia in future pregnancies should be provided to parents.

Keywords: Neonatal thrombocytopenia, Autoimmune, IVIg

INTRODUCTION

Neonatal thrombocytopenia is one of the common haematological problems encountered in neonatal intensive care unit (NICU). Its severity is classified as mild ($100\text{-}150\times10^3$/µl), moderate ($50\text{-}99\times10^3$/µl) or severe ($<50\times10^3$/µl).[1] Neonatal thrombocytopenia is the result of impaired platelet production, increased platelet destruction and sequestration or the combination of both. The overall incidence of neonatal thrombocytopenia is relatively low (0.7-0.9%), however, the incidence among neonates admitted to the NICU ranges from 22-35%. The first step while evaluating a thrombocytopenic neonate is to classify the thrombocytopenia as either early onset (within the first 72 h of life) or late onset (after the initial 72 h of life) and to assess whether the neonate is clinically well or ill.[1]

Foetal and neonatal immune thrombocytopenia is an important cause of early onset thrombocytopenia, caused by maternal immunoglobulin G crossing the placenta and leading to destruction of foetal platelets. Neonatal immune thrombocytopenia can be categorized in two forms. The autoimmune condition is related to maternal immune thrombocytopenia, while the alloimmune form, commonly named neonatal

alloimmune thrombo-cytopenia (NAIT), is due to trans placental passage of specific antibodies against foetal platelets exhibiting antigens inherited from the father.[2]

Neonatal autoimmune thrombocytopenia should be considered in any neonate who has early-onset thrombocytopenia and a maternal history of either idiopathic thrombocytopenic purpura (ITP) or an autoimmune disease (with or without thrombocytopenia). The incidence of severe neonatal thrombocytopenia in such children has been reported between 8.9% to 14.7%, with intracranial bleed occurring in 0% to 1.5% of affected neonates.[1] Hereby, we present a case of severe early onset neonatal thrombocytopenia associated with maternal immune thrombocytopenia.

CASE REPORT

A male baby was delivered at 39 weeks of gestation to a 23-year-old primi-gravida mother by caesarean section (CS) with a birth weight of 3000 gm. The mother was in latent phase of labour and had severe thrombocytopenia on presentation (lowest platelet count-35×10^3/µl) in the emergency department. She required one unit of single donor platelet to reach a value of 55×10^3/µl on the day of emergency CS, done in view of meconium-stained liquor. Based on history and available records, the mother received six units of random donor platelets (RDP) for thrombocytopenia eight days prior to presentation at our centre. She was normotensive and rest of the antenatal course of pregnancy was uneventful. Considering the possibility of ITP, she was started on corticosteroids.

The child did not cry immediately after birth and required bag and mask ventilation for 10 seconds. The APGAR score at 1, 5 and 10 minutes were 5, 7 and 9, respectively. Baby was admitted in NICU in view of respiratory distress. On examination baby had no signs of active bleeding such as bruising or petechial haemorrhage, conjunctival haemorrhages, bleeding from oral mucosa or any other site. Vitals were stable and was maintaining saturation at 1 litre of oxygen via nasal prongs. Other systemic examinations were normal. Over next few hours, his respiratory distress subsided and was weaned off oxygen.

On initial work up, the platelet count was found to be 85×10^3/µl with no evidence of platelet clumps on blood smear. All other haematological and biochemical lab parameters were within normal range (Table 1). Given the history of maternal thrombocytopenia (likely ITP), a possibility of neonatal autoimmune thrombocytopenia was considered. Serial platelet counts on day 2 and 3 of life were 52×10^3/µl and 6.5×10^3/µl, respectively (Figure 1). Owing to the risk of massive bleed, the baby was transfused random donor platelets (RDP), at a dose of 15 ml/kg on day 3 and treatment with intravenous immunoglobulin (IVIg) was started shortly (dose-0.5 g/kg). There was no evidence of bleeding from any site and the child was clinically well. On day 4, the platelets increased to 70×10^3/µl and on further monitoring showed an increasing trend thereafter. Ultrasound cranium was normal. The child was discharged on day 7 of life with a platelet count of 170×10^3/µl. After discharge, the child did not come for follow-up until after a month when the platelet count was again found to be low (84×10^3/µl). However, it normalised subsequently, without any further requirement of IVIg.

Table 1: Overview of the initial laboratory work up of the neonate.

Lab parameters	Patient value	Reference values[3]
Haemoglobin (g/dl)	18.6	15-24
Total leukocyte count (cells/mm$^{3)}$	9.6×10^3	$9.1\text{-}34\times10^3$
Platelets (/µl)	85×10^3	$1.5\text{-}4\times10^3$
Urea nitrogen (mg/dl)	8.6	3-12
Creatinine (mg/dl)	0.36	0.03-0.5
CRP (mg/dl)	0.173	<0.6
ALT (IU/l)	22.9	6-40
AST (IU/l)	19.7	30-100
Blood culture	Sterile	-
Platelet clumps	Not seen	-

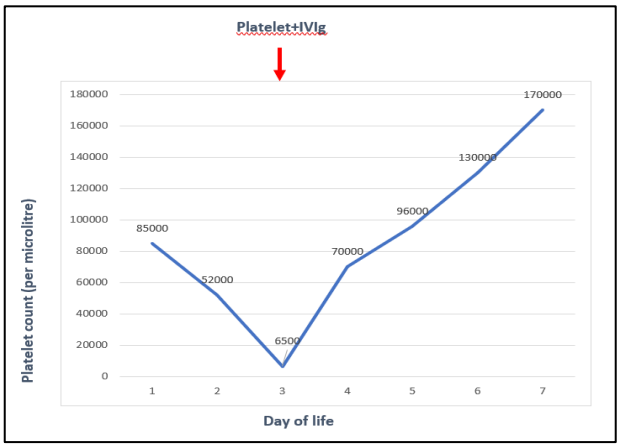

Figure 1: Platelet count and timing of interventions during NICU stay.

DISCUSSION

The incidence of ITP is estimated to be 0.83 per 10000 pregnant women, with a variable course, usually worsening with progression of pregnancy.[4] Careful monitoring of platelet count is required. A stable platelet count of 20 to 30×10^3/µl is safe during most of pregnancy, whereas, during delivery a platelet count $>50\times10^3$/µl is advised.[5] While corticosteroids are considered as the first-line treatment for ITP in pregnancy because of their efficacy and low cost, IVIg may be preferred during delivery when rapid increase in platelet count is required.[5] In our case, the mother presented in labor and was taken up for emergency CS, therefore, only platelet transfusion could be done to bring the platelets

above 50×10^3/µl. IVIg infusion could not be started due to financial constraints.

All neonates born to mothers who have autoimmune diseases or idiopathic thrombocytopenia should undergo a screening platelet count (cord blood) is at or shortly after birth.[5] Neonatal thrombocytopenia is seen in 10-15% of infants born to mothers with ITP; was usually mild to moderate, resolves spontaneously and requires no specific therapy.[6] Around 1-5% will have platelet count $<20\times10^3$/µl and nearly 1% will develop severe bleeding complications.[7] Intracranial haemorrhage leading to death or neurological impairment is the most feared complication. Maternal platelet counts do not correlate with that of neonate and therapy of the mother is not known to affect neonatal outcome. Currently, the only reliable predictor of neonatal thrombocytopenia was its presence in previous sibling. The severity and pattern also correlates with the severity of illness in the previous child.[8] In our case, the baby was first born, so development of severe thrombocytopenia is not expected. The parents were provided counselling regarding the risk of severe thrombocytopenia in future pregnancies.

The principal aim in managing affected infants is to prevent the deleterious consequences of severe thrombocytopenia. If the platelet count is normal, no further evaluation is necessary. If it was $<100\times10^3$/µl, as seen in our case, daily platelet counts are recommended until stable.[5] The absence of platelet clumps on peripheral smear is ruled out pseudo-thrombocytopenia, which was seen frequently in neonates due to difficulty in sampling. The platelet count reached its lowest value on day 3, correlating with the maturation of neonatal spleen. In cases, where the platelet count falls below 30×10^3/µl, IVIg is recommended as the first line of therapy. Random-donor platelets, in addition to IVIG, should be provided only if the infant has evidence of active bleeding.[5] Our patient received both IVIg and RDP on third day, owing to risk of life-threatening bleed due to severe thrombocytopenia. It had been observed that anti-platelet antibody in breast milk may contribute to persistence of neonatal thrombocytopenia.[8] We did not find any effect of breast feeding as the child recovered on exclusive breast feeding.

Cranial imaging should be obtained in all infants with platelet counts 50×10^3/µl during delivery to evaluate for intracranial hemorrhage. Importantly, neonatal thrombocytopenia secondary to maternal ITP may last for months and requires long-term monitoring and sometimes a second dose of IVIG at 4 to 6 weeks of life.[5] Our patient did not come for initial follow-up, however, at 4 weeks of age his platelet counts again dropped though he did not require any transfusion or IVIg and recovered spontaneously over time. In cases of maternal ITP, it might be prudent to discuss the transfer of the mother to a tertiary obstetric center to allow urgent transfer of the newborn to a neonatal ward if complications occur.

CONCLUSION

Although neonatal thrombocytopenia associated with maternal ITP is usually mild to moderate, it is important to consider that some infants may have severe thrombocytopenia for several days following delivery. Our case highlights the importance of post-natal monitoring of platelets in new-borns with maternal history of thrombocytopenia, so that appropriate management can be done in a timely fashion and bleeding complications can be avoided.

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REFERENCES

1. Eichenwald EC, Hansen AR, Martin CR, Stark AR. Cloherty and Stark's Manual of Neonatal Care. Eighth edition. Philadelphia: Wolters Kluwer. 2017;631-3.
2. Becocci A, Civitillo CF, Laurent M, Boehlen F, Luca RD, Fluss J. Intracranial Hemorrhage and Autoimmune Thrombocytopenia in a Neonate: A Rare "Unpredictable" Event**.** Child Neurol Open. 2018;5:1-5.
3. Kliegman, Robert. Nelson Textbook of Pediatrics. Edition 21. Philadelphia, PA: Elsevier. 2020.
4. Care A, Pavord S, Knight M, Alfirevic Z. Severe primary autoimmune thrombocytopenia in pregnancy: a national cohort study. BJOG. 2018;125:604-12.
5. Provan D, Arnold DM, Bussel JB, Chong BH, Cooper N, Gernsheimer T et al. Updated international consensus report on the investigation and management of primary immune thrombocytopenia. Blood Adv. 2019;3:3780-817.
6. Koyama S, Tomimatsu T, Kanagawa T, Kumasawa K, Tsutsui T, Kimura T. Reliable predictors of neonatal immune thrombocytopenia in pregnant women with idiopathic thrombocytopenic purpura. Am J Hematol. 2012;87:15-21.
7. Kelton JG. Idiopathic thrombocytopenic purpura complicating pregnancy. Blood Rev. 2002;16:43-6.
8. Payne SD, Resnik R, Moore TR, Hedriana HL, Kelly TF. Maternal characteristics and risk of severe neonatal thrombocytopenia and intracranial hemorrhage in pregnancies complicated by autoimmune thrombocytopenia. Am J Obstet Gynecol. 1997;177:149-55.

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