

Case Report

Maternal and Neonatal Idiopathic Thrombocytopenic Purpura

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Introduction

Immune thrombocytopenic (ITP) is an autoimmune disease that affects 9.5/100,000 adults in the population.¹ It is characterized by autoimmune destruction of platelets leading to potentially life-threatening thrombocytopenia. About 7% of pregnant mothers are affected by thrombocytopenia with 1 to 10 per 10,000 mothers being affected with ITP. Studies have shown that ITP is generally a benign condition in the birthing mother and rarely has a long-lasting effect on the newborn child.² In the rare event that the newborn is severely affected, treatment is required. We present a case of ITP in both a mother and her newborn.

Case report

The infant is a 40 weeks and 1 days gestational female who was born via spontaneous vaginal delivery to a 35 year old now gravida 3, para 3 Hispanic mother with a history of ITP of pregnancy. Mother was group B streptococcus positive but otherwise negative for all other serologies. Per maternal report and prior medical records, her first pregnancy revealed platelets that had declined to 19,000/ mm3 that recovered to 85,000/mm3 after intravenous methylprednisolone. That delivery was complicated with post-partum bleeding requiring a transfusion of two units of packed red blood cells. Records were unavailable for her first infant, but it reportedly did well and treatment was not necessary. Mother was discharged with steroids and hematology follow-up but did not continue evaluation and monitoring secondary to financial difficulty. No data was available for her second infant as she did not deliver at our institution. Per maternal report, that infant also suffered from ITP but severity was unknown.

The mother returned to our institution to establish care for our patient at 34 weeks gestation. Her prenatal course was complicated only with gestational diabetes that was diet controlled. She was admitted to the hospital at 39 weeks 4 days gestation for induction due to gestational diabetes mellitus and a presumed large for gestational age fetus. Her platelets upon admission were 36,000/mm³ with no signs of bleeding or bruising. The hematology service was consulted prior to induction and recommended high dose prednisone (80 mg) initially. Maternal platelets did not recover after this single steroid therapy, thus intravenous immunoglobulin (IVIg) 1g/ kg was started along with an additional dose of prednisone 80 mg. Again, maternal platelets did not respond but remained stable at 43,000/mm³. After discussion with the hematology service, the obstetrics team administered an additional dose of IVIg and proceeded with induction with the plan to give one unit of platelets during active labor. The infant's delivery was vaginal and uncomplicated. Her initial APGAR scores were 1 at one minute and 6 at 5 minutes and she was transferred to the neonatal intensive care unit (NICU) for continuous positive airway pressure for treatment of apnea. Her apnea quickly resolved and she was observed in the NICU for several hours and then transferred to the newborn nursery service. Following transfer, a complete blood count (CBC) was drawn that revealed a platelet count of 108,000/mm³. She had no signs and symptoms of bleeding or bruising and thus was observed. A repeat CBC was drawn on postnatal day two which resulted in a declining platelet count of 69,000/mm³. The pediatric hematology service was consulted and recommendations included prednisone 2 mg/kg twice daily for 5 days. Her platelets continued to decrease



to 25,000/mm³ by her third day of life. She was subsequently transferred to the NICU for IVIg therapy 1g/kg every 12 hours. The infant tolerated IVIg therapy well with an improvement in her platelets to 64,000/mm³. She was discharged in good condition at 5 days of life.

Discussion

Here we have presented a case of a neonate with passive immune thrombocytopenia secondary to ITP. When managing ITP in pregnancy, one must always remember that there are potentially two patients that can be affected: mother and infant. A complete review of adult immune thrombocytopenia is beyond the scope of this review, but we will summarize the treatment of pregnant mothers and review the available literature regarding management of affected infants in the peripartum period. ITP of pregnancy occurs in 1 to 10 per 10,000 pregnant women.2 It is differentiated from gestational thrombocytopenia based on the timing of presentation and the severity of the thrombocytopenia. Gestational thrombocytopenia does not usually present until the mid-second trimester, is usually less severe (platelets >80,000/mm³) and platelet counts typically normalize once the baby is delivered. It is a diagnosis of exclusion and other causes of thrombocytopenia such as pre-eclamptic syndromes, disseminated intravascular coagulation (DIC) or autoimmune syndromes, must be ruled out. As in non-gestational ITP, testing for anti-platelet antibodies is not recommended.²

Treatment for the mother with ITP is usually undertaken when platelet counts decrease to <30,000/mm³ and only when closer to term. Treatment is usually with IVIg or prednisone with a goal platelet count of 80,000/mm³ at the time of delivery.^{2,3} Although recent studies have suggested that IVIg is more effective and better tolerated 1,3,45, corticosteroids are often given as first line treatment.^{1,4,5} IVIg can be given if steroids are ineffective or intolerable, or if the platelet count needs to be increased acutely.^{2,3} If refractory to monotherapy, combination therapy with corticosteroids and IVIg can be attempted4. There are 2 main concerns in regards to the newly birthed infant: 1) determining if the infant is thrombocytopenic and 2) the management of the infant if they become thrombocytopenic. Earlier studies, before 1990, estimated the incidence of thrombocytopenia in infants born to mothers with ITP at 70%.^{5,6} More recent studies have shown a lower incidence, with only 13 to 29% of infants having a platelet count of less than 50,000/mm^{3.7,8} Complications such as submucosal bleeding, purpura and skull hematomas affect only 6% of patients.9 Cases of intracranial bleeding or death attributable to thrombocytopenia are exceedingly rare, occurring in 0 to 2% of cases.⁶⁻¹⁰

Numerous studies have examined factors predictive of neonatal thrombocytopenia. Low maternal platelet counts during pregnancy have been associated with an increased risk of severe neonatal thrombocytopenia.^{7,10,11} A prospective Japanese study looked at risk factors for thrombocytopenia in newborn infants from mothers with ITP and found that previous history of thrombocytopenia in siblings, history of splenectomy and presence of anti-platelet antibodies in the mother gave increased risk for neonatal thrombocytopenia.¹² This was further confirmed by a national study of 284 women with ITP in Japan.⁷

Most infants who are affected have a normal platelet count at birth, which decrease over the following 1 to 7 days.⁹ Treatment of the newborn infant is generally not taken until the platelets fall below 30,000/mm³ unless there is some evidence of bleeding or bruising.² It generally follows along the same guidelines as treating ITP in children. If platelets are beginning to trend down or are below 50,000/mm³ is with, initial treatment is commonly with IVIg or prednisolone.²⁻⁴ As was illustrated in our case, the neonate responded rapidly to IVIg. They should then be monitored frequently to ensure that platelet counts are maintained.

Here, we present a case of maternal ITP with a resulting neonate that required treatment. Though this occurs rarely, it illustrates a few important aspects in the management of a neonate who is born to a mother with ITP. The first is prompt monitoring. Given the known risk severe thrombocytopenia, a CBC should be drawn at birth and platelet counts should be monitored for at least 1 to 2 weeks. Although there is no set limit for treatment in the absence of bleeding, treatment should probably be undertaken when platelet counts approach 20,000/mm³ Treatment can include both corticosteroids and/or IVIg.^{11,12}

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Conflicts of interest

The authors report no financial interests or potential conflicts of interest.

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