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Case Report

Diagnostic Dilemma and Challenges in Management in a Case of Immune Thrombocytopenic Purpura in Pregnancy and Review of Literature

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Abstract

Thrombocytopenia is encountered in as many as 10% pregnancies, and the causes of thrombocytopenia in pregnancy are manifold. Failure to identify the correct cause can lead to unnecessary platelet transfusions in certain conditions while it (thrombocytopenia) could be corrected by appropriate drug therapy. Furthermore, consideration has to be given to the effect of drug therapy on the fetus. Here, we present a case where a pregnant patient of immune thrombocytopenic purpura showed no improvement in platelet count despite having received multiple platelet transfusions and intravenous methylprednisolone but in the end, responded to intravenous immunoglobulin.

Key words: Immune thrombocytopenic purpura, Intravenous immunoglobulin, Methylprednisolone, Pregnancy, Transfusion

INTRODUCTION

Incidence of thrombocytopenia in pregnancy is 8–10%.[1] The causes of thrombocytopenia in pregnancy are manifold; some are seen to be associated only with pregnancy, whereas others occur in non-pregnant patients as well. Management options become fewer during pregnancy as many drugs can prove to be toxic to the fetus. Immune thrombocytopenic purpura (ITP) is an autoimmune disorder in which platelets are destroyed due to the binding of antiplatelet antibodies. It is one of the most common autoimmune disorders seen nowadays. Although it can present at any age, it has a predilection for young women.[2] We report a pregnant patient with ITP who posed a therapeutic challenge.

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CASE DESCRIPTION AND RESULT

A 28-year-old woman, G_3P_{2002}, was admitted to our department at 36 weeks gestation with complaints of pain in abdomen and vaginal bleeding for 1 day. Her medical history was unremarkable, with no previous history of bleeding from any other site. Her blood pressure was 116/78 mmHg, and pulse rate was 88/min. She did not have pallor, icterus, or lymphadenopathy. Abdominal examination revealed that her fundal height was corresponding to the period of gestation, with a longitudinal lie and cephalic presentation and the fetal heart rate was 140 beats/min. Uterus was relaxed. Pelvic examination revealed no bleeding with the closed external os. Investigations revealed hemoglobin of 10 g/dL, white blood cell count of 7800/μL and platelet count of 20,000/μL. Her total bilirubin was 1.3 mg/dL with indirect bilirubin of 0.9 mg/dL. Renal function tests were normal. C-reactive protein level was 6 mg/L. Ultrasonography of the abdomen revealed mild hepatosplenomegaly along with single live fetus with the fundal placenta. Anti-nuclear antibody was negative. Urine examination was unremarkable. Peripheral smear examination showed

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moderate anisocytosis, occasional macrocytes, and normocytic normochromic red cells with severely reduced platelets. Dengue serology was negative and prothrombin time and activated partial thromboplastin time were normal. Human immunodeficiency virus, hepatitis B surface antigen and anti-hepatitis C were negative. Blood culture showed no growth. The patient was transfused 24 units random donor platelets in 3 days, but her platelet count kept on decreasing, reaching a nadir of 10,000/μL. Again, she was transfused 16 units of random donor platelets. Bone marrow aspiration was done which showed the normal maturation of myeloid and erythroid series with megakaryocytes. Diagnosis of immune thrombocytopenia was made. Again, her platelet count dropped down to 12,000/μL. Intravenous methylprednisolone therapy was started. Meanwhile, she went into labor. 12 unit random donor platelets were transfused during the intrapartum phase. She delivered a healthy baby weighing 2.5 kg. She had atonic postpartum hemorrhage which was managed by Bakri balloon intrauterine insertion and transfusion of 14 more random donor platelets along with 2 units of packed red cells. Due to the failure of rise in platelet counts, intravenous immunoglobulin (IVIG) was started, after which her platelet count increased to 44,000/μL. During the course of treatment, she received total 50 units of random platelets. The baby's platelet count was 2.01 lac/μL. Mother and baby were discharged in good condition on day 16 postpartum.

DISCUSSION

The incidence of ITP in pregnancy is estimated at 0.1–1/1000.[3] Primary ITP is a diagnosis of exclusion. It has to be differentiated from both non-immune causes of thrombocytopenia and secondary immune thrombocytopenia. Accurate diagnosis is essential for appropriate treatment. Immune thrombocytopenia can be secondary to an autoimmune condition, a lymphoproliferative disease, a chronic infection or medication.[4] Although rare as compared to gestational thrombocytopenia, ITP is the most common cause of isolated thrombocytopenia in the first and early second trimesters of pregnancy.[5]

The aim of treating ITP in pregnancy is to prevent bleeding. Thus, treatment is, generally, not required in patients who are not bleeding and with platelet counts >20,000–30,000/μL. Epidural anesthesia in a thrombocytopenic patient increases the risk of epidural hematoma formation. Therefore, those patients who wish to receive epidural anesthesia, require higher platelet counts.[6] A platelet count of at least 75×10^9/L is, generally, recommended to allow administration of epidural anesthesia. Some believe that a platelet count of at least 50×10^9/L is adequate to allow for cesarean section.[7]

Corticosteroids are the first-line of therapy for ITP even in pregnancy. However, corticosteroids can lead to diabetes and hypertension in pregnancy. Some reports link use of corticosteroids in the first trimester with congenital anomalies, such as orofacial clefts. For these reasons, the use of corticosteroids should be adjusted to the minimal effective dose in pregnancy. Others have argued that due to the toxicity of corticosteroids, IVIG should be considered the first-line of therapy for ITP in pregnancy. Thus, the therapy should be decided keeping the various factors in mind such as the gestational age when the therapy is required, the expected duration of therapy, and specific characteristics of the patient.[6]

For patients who do not respond to corticosteroids or IVIG as monotherapy, combinations of these therapies should be given.[7] If this fails, laparoscopic splenectomy may be safely performed during the second trimester of pregnancy. The rationale behind this is that surgery earlier in pregnancy may lead to premature labor, and later in pregnancy splenectomy may be technically difficult due to obstruction of the surgical field by the gravid uterus.[6]

For refractory ITP in pregnancy, very few therapeutic agents are available. Most cytotoxic agents such as azathioprine and cyclosporine A are teratogenic but have been used safely in pregnant patients with neoplasia and renal transplants. There is very little data available about the safety of use of thrombopoietic agents such as romiplostim and eltrombopag in pregnancy. Rituximab causes a delay in neonatal B-lymphocyte maturation but does not lead to any significant clinical consequences and is thus used in treating ITP in pregnancy.[8]

Antiplatelet antibodies can cross the placenta and cause destruction of fetal platelets. The development of fetal thrombocytopenia cannot be predicted and has no consistent correlation with the degree of maternal thrombocytopenia or any other parameters. 15% of babies of mothers with ITP develop thrombocytopenia with platelet count <100,000/μL. 10.1% of babies have platelet count below 50,000/μL, and only 4.2% babies develop platelet count <20,000/μL.[9] Neonates with thrombocytopenia may have intracranial hemorrhage during vaginal delivery which can prove to be fatal. Previously, all patients of ITP used to undergo cesarean section due to this concern.[7] However, the results of several studies have demonstrated that the risk of fetal intracranial hemorrhage in the offspring of mothers with ITP is only between 0% and 1.5%[9] and that there is no evidence this risk is increased by vaginal delivery.[10] These studies have led to the establishment of

current guidelines, which suggest that the mode of delivery in pregnant patients with ITP should be dictated only by maternal indication. However, the neonatal platelet count should still be determined for the next 5 days, because the nadir of neonatal platelet count may not develop until several days after delivery. All neonates with platelet counts <50,000/μL should undergo transcranial ultrasound to rule out intracranial hemorrhage.[7]

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