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ITP with Severe Thrombocytopenia in Pregnancy

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Abstract

Thrombocytopenia is second common hematological disorder in pregnancy with an incidence of 6-15%. Most common cause of low platelets in pregnancy is gestational thrombocytopenia followed by infections, hypertensive disorders of pregnancy and idiopathic thrombocytopenic purpura. We are presenting a case of ITP with severe thrombocytopenia resulted in adverse perinatal outcome.

Keywords: ITP, thrombocytopenia, intracranial hemorrhage, prednisolone, IVIg

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Introduction

Thrombocytopenia occurs in 6-15 % of pregnant women and is a common diagnostic and management issue. It has several causes including gestational thrombocytopenia, viral and bacterial infections, preeclampsia complicated by HELLP syndrome and idiopathic thrombocytopenic purpura (ITP) [1]. Maternal thrombocytopenia is associated with risk of fetal thrombocytopenia which is usually mild. However in few patients there can be fetal intracranial hemorrhage and sinister consequence in form of intrauterine fetal demise. Invariably the cause of still birth remains unexplained. Here we present a case report of a lady who presented with extremely severe thrombocytopenia, was managed with steroids and intravenous immunoglobulin but unfortunately had a poor perinatal outcome.

Case Report

A 26 year old G5P1L1A3 lady with Post LSCS pregnancy and Rh negative non allo-immunised status was admitted to our hospital at 27W3D POG with severe thrombocytopenia (Platelets - 25,000/µl). Her third pregnancy was complicated by severe thrombocytopenia in the postpartum period following which she was transfused four platelet concentrates. She had no history of menorrhagia, bleeding diathesis or family history of bleeding disorder. On investigation, ACLA, LAC, ANA, dengue serology and thyroid hormones were normal. HIV, HBV, HCV serology was negative. No hepatosplenomegaly was detected on ultrasound. Bone marrow biopsy showed normal M:E ratio and megakaryocytic thrombocytopenia.

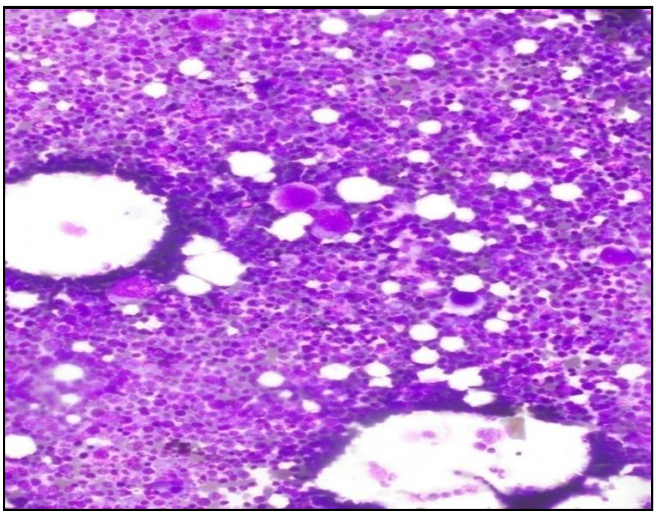

Figure 1. Bone marrow aspirate picture

After evaluation she was diagnosed as Idiopathic thrombocytopenic purpura (ITP) and started on tab Prednisolone 40 mg daily. Surfactant induction was carried out with 4 doses of Inj Dexamethasone at 28 weeks. Patient was discharged at 29 weeks with platelet count of 1,29,000/µl. Patient lost to follow up thereafter and reported at 35W5D POG with antepartum hemorrhage. She was normotensive, there was no active bleeding per vaginum and non-stress test (NST) was reactive. There were no petechiae, purpura or bleeding from other orifices. USG pelvis revealed no retroplacental clot and placenta was anterior, not previa. An urgent hemogram showed significantly reduced platelets (6,000/µl). Coagulation parameters were normal. She was immediately started on intravenous immunoglobulin (IVIg) 2 gm/kg in divided doses under close supervision. But after 6 hours of initiation of therapy, patient had an intrauterine fetal demise.

Improvement in platelet count was noticed after 3 days of IVIg therapy, following which she was given tab Mifepristone 600 mg orally. She went into spontaneous labor within 48 hrs and delivered a macerated still born (male) weighing 2.32 kg. The still born was sent for autopsy and placenta for histopathology. However the cause of fetal demise was unexplained. Postnatally patient was started on tab Dapsone 100 mg daily in addition to Prednisolone 30 mg. She was discharged on 10th postnatal day with platelet count of 86,000/µl. Patient is on regular follow up.

Discussion

Thrombocytopenia is second to anemia as the most common hematological abnormality in pregnancy. Defined as platelet count less than 1.5 lakh/µl, it occurs in 6-15 % of pregnant women [3]. 75 % cases stem from gestational thrombocytopenia. ITP accounts for 1-4 % of cases and is caused by a cluster of IgG antibodies directed against one or more platelet glycoproteins. Antibody coated platelets are destroyed prematurely in the reticuloendothelial system, especially the spleen [4].

The diagnosis of ITP is not difficult when patients have prior history of thrombocytopenia, or in the setting of moderate to severe thrombocytopenia (<50,000/µL) early in gestation with decreasing platelet count as gestation progresses [1]. This was exactly the scenario in our patient who had past history of platelet transfusion in view of severe thrombocytopenia and had progressively declining platelet count with increase in gestational period in present

pregnancy. Pregnant women with ITP can be asymptomatic or may present with a history of easy bruisability, bleeding into the mucous membranes (gum bleed, epistaxis), petechiae or purpura [1]. Our case did not have any of these except for presenting with antepartum haemorrhage in third trimester. ITP is a diagnosis of exclusion with peripheral thrombocytopenia and normal or increased megakaryocytes in the bone marrow, red and white cell count is normal [2]. Our patient had megakaryocytic thrombocytopenia on bone marrow aspiration.

Therapy is considered if platelet count is less than 30,000 - 50,000/µL [4]. Due to their efficacy and low cost, many consider corticosteroids as the first choice of treatment. Prednisolone 1 mg/kg daily is given to suppress the phagocytic activity of the splenic monocyte macrophage system. However, since steroids may increase the incidence of pregnancy induced hypertension, and exacerbate gestational diabetes, an alternative is IV immunoglobulin (IVIg) (2 gm/kg during 2-5 days) which some experts consider as first line therapy for pregnancy associated thrombocytopenia due to its low toxicity profile [2]. Our patient was initially started on tab Prednisolone and later given IVIg. Intravenous anti-D, in a dose of 50 - 75 microgram/kg intravenously can be used in Rh D positive women. Splenectomy can be done in mid pregnancy for refractory cases. Platelets to be transfused in cases of acute bleeding irrespective of Platelet count, patient in labour or being taken for LSCS and Platelets <50,000/µL in conjunction with IVIg.

ITP causes mild fetal thrombocytopenia. There is no relationship between maternal and fetal platelet count. Adverse outcome includes intracranial hemorrhage in fetus which is unusual (<1.5%). Mortality is occasional (<1%) because of haemorrhage. However most fetal deaths are unexplained as in our case.

Conclusion

Thrombocytopenia in pregnancy requires detailed evaluation. Diagnosed cases of ITP need to be followed up regularly. Maternal thrombocytopenia can cause fetal thrombocytopenia and at times lead to adverse perinatal outcome in the form of intrauterine fetal demise. A very miniscule of these still births occurs because of fetal intracranial hemorrhage but the majority have no specific cause.

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