Immune Thrombocytopenia in Pregnancy

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ABSTRACT

Introduction: Immune thrombocytopenia (ITP) is a common haematological disorder in pregnancy. It complicates upto 10% of pregnancies. It can be benign to potentially hazardous for maternal and fetal well being. It can raise anxieties during antenatal period, around the time of delivery because of possible bleeding associated with vaginal delivery. ITP occurs in one or two of every 1000 pregnancies. It is an acquired autoimmune disorder haracterized by low platelet count secondary to accelerated destruction and impaired thrombopoiesis due to circulating anti platelet antibodies. These antibody coated platelets are then removed following binding to macrophage Fc receptors primarily in the spleen.

Case report: Hereby we are presenting a case of post partum thrombocytopenia with ecchymotic rash. She posed a diagnostic dilemma and therapeutic challenge as multiple factors were involved. After Bone marrow study we could reach the diagnosis.

Conclusion: The incidence of ITP in pregnancy is 1-2 per 1000 deliveries. It is a common disorder among women of child bearing age. There is always a risk of fetal or neonatal thrombocytopenia. Management of ITP in pregnancy can be a complex and a challenging task.

Keywords: Immune Thrombocytopenia, Low Platelet Count, Pregnancy

INTRODUCTION

ITP is a bleeding disorder in which the immune system destroys the platelets. In this disorder, the immune system produces antibodies against the platelets which attach to the platelets and the body destroys the platelets that carry the antibodies. The American society defines ITP as Isolated thrombocytopenia with no clinically apparent associated conditions or other causes of thrombocytopenia, such as infections, medications, haematological malignancies, DIC or other Autoimmune conditions. The symptoms maybe easy bruising (purpura), superficial bleeding into the skin (petechiae), bleeding gums or from nose, blood in the urine or stools. unusually heavy menstrual flow. Women are three times more likely to develop ITP than men. In children it follows a viral infection like Measles, rubella, mumps. several studies have examined pregnancy outcomes for women with ITP and found that most pregnancies were uneventful, with successful outcomes for both mothers and babies.[1-3]

CASE REPORT

A 25 year old married female presented in the hospital as an emergency case with ecchymotic rash all over the body after caesarean section. The caesarean section was done at a nursing home under regional anaesthesia at 5.30 am on 15th oct 2015. She was shifted to my hospital at 9pm. Her platelet count was 30, 000 at the time of admission.

She had no history of fever, flu like symptoms, pruritus or history of similar complaints in the past. There was no family history of similar complaints or bleeding disorder. There was no history of hypertension in pregnancy.

On examination she was conscious, oriented and responding to questions. Her general condition was stable. Pulse was 90/minute and blood pressure was 100/60 mm of Hg. There were ecchymotic patches on he arms, legs and abdomen. She had average bleeding per vaginum. There was no history of bleeding gums. The uterine height was above the umbilicus. Systemic examination was normal. There was no evidence of splenomegaly and peripheral lymphadenopathy. Her platelet count done at 22 weeks was normal.

On admission the repeat platelet count was 60, 000. It was checked again to avoid confusion due to spurious results.

Investigations

Total leucocyte count was 18000/cmm. The peripheral smear showed reduced platelets but no evidence of red cell fragments (schistocytes). Malarial parasites were not seen

Haemoglobin was 11g/ dl

INR was 1.4

LFT and RFT were normal.

Blood glucose was 92 GM /DL

Urine showed proteins as 4+ by dip stix method and negative for glucose and nitrites

HIV was non reactive

ANA was negative

Screening for APLA and SLE was negative

Management

As she had thrombocytopenia with albuminuria with derangement of coagulation profile, a provisional diagnosis of DIC was made. She was given fluids, antibiotics, vitamin k injection and infused with fresh frozen plasma to revert back her coagulation defect. Her platelet count was repeated the next day which had lowered to 30, 000.

It was thought that there might be chances of consumptive coagulopathy and clots present inside the uterine cavity. The uterus was bigger too. She was give 10 units of oxytocin infusion in an attempt to empty the uterus. Ultrasound study showed no intrauterine or intraperitoneal collection. She was transfused with 3 units of platelet rich plasma.

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Her blood investigations like serum LDH, IDCT was done. Bone marrow study was performed which was suggestive of immune thrombocytopenic purpura.[4] The bone marrow was megakaryocytic. Antiplatelet antibody was advisable but the test was not available at our setup.

She was managed with methylprednisolone 1 gram for 3 days.[3] Her platelets showed a dramatic rise and the count was 1.4 lac/cumm after 3 doses. She was observed in the hospital for 3 days. The neonatal platelet count was checked and it was within normal limits. She was put on oral prednisolone tablet 10 mg twice a day. Her platelet count was 1.6lac/cu mm after 15 days.

She was advised monthly review with platelet count.

DISCUSSION

ITP presents as a primary form as a thrombocytopenia in the absence of other causes that may cause thrombocytopenia. ITP may affect individuals at all ages.[2]

The patient had developed thrombocytopenia in the postpartum period, may be in the late third trimester, as her platelet count was normal in the second trimester. So it was unlikely to be gestational thrombocytopenia.

She was not hypertensive and liver enzymes were normal so HELLP syndrome was excluded.

We had a doubt regarding if she had DIC as she had deranged INR, low platelet count and raised FDPs but in spite of giving fresh frozen plasma she continued to have a fall in platelet though her INR came back to normal value.

TTP and HUS are also seen in postnatal period. Both are a continuum and are manifestations of a similar mechanism of microvascular aggregation of platelets.

If there is systemic with neurologic involvement the disorder is TTP. If platelet aggregation is less extensive, with predominant renal involvement, the disorder is HUS.

As she had no fever or neurological symptoms TTP was ruled out. Her renal function test was normal ruling out HUS. The symptoms of TTP/HUS may be confused with pre eclampsia and HELLP syndrome. However hypertension is not common in TTP/HUS and there is no coagulopathy. These findings lead me to the diagnosis of ITP.

The diagnosis of ITP is one by the exclusion and should be made once other causes are ruled out. In ITP, the bone marrow is normal or megakaryocytic.

Pregnancy does not affect the course of ITP.

Antiplatelet antibodies can cross the placenta and cause fetal thrombocytopenia. It is difficult to predict which fetus or neonate will be affected. Corticosteroids are the first line therapy. It is common to use lower dose like 20-30mg/day. It is safe and effective. Following this the dose is weaned off to the lowest that will maintain a satisfactory maternal platelet count.

IVIg is given in resistant cases or in women who are likely to need prolonged treatment.

Anti-D immunoglobulin is given as an intravenous bolus which may help to raise the platelet count in non splenectomised rhesus positive patient. Splenectomy may be needed in extreme cases. Other options for women who do not respond to these methods are azathioprine or cycloserine. Platelet transfusions are given as last resort though they do not result in a sustained increase in platelet counts. Eltrombopag olamine is a thrombopoietin receptor agonist indicated for the treatment of thrombocytopenia in patients who had an insufficient response to corticosteroids or immunoglobulin or splenectomy. It is given orally with 50 mg OD dose. The dose is adjusted in patients with hepatic impairment.

CONCLUSION

This patient responded very well to corticosteroids and did not need any other medication or splenectomy. She was followed up for 12 months with the haematologist and myself. She was given oral prednisolone which was tapered gradually over the year with monthly platelet count. She was off the steroids in a year and maintaining a normal platelet count.

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