Case Report

Recurrent severe gestational thrombocytopenia in pregnancy: a case report

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ABSTRACT

Most of the newly diagnosed cases of thrombocytopenia in pregnancy are mild, asymptomatic and accidentally discovered on routine antenatal screening. Common causes for the same include gestational thrombocytopenia, Preeclampsia, HELLP syndrome, and less commonly immune causes like ITP. As a matter of fact, HELLP and Preeclampsia have specific diagnostic signs and symptomatology, others are rather difficult to distinguish, as they are usually asymptomatic. We present a case of 21 years second gravida at 35 weeks and six days period of gestation, referred from a local practitioner for severe thrombocytopenia (Platelet count-20000/mm³). She had history of previous still birth due to cord prolapse, and severe thrombocytopenia (Platelet count-6000/mm³) in previous pregnancy. She recovered rapidly and spontaneously in postpartum period. The newborn platelet count was also normal. She was considered to be a case of rare but severe recurrent gestational thrombocytopenia, after ruling out other causes of severe thrombocytopenia in pregnancy. Management includes adequate preparations for ensuring optimal fetomaternal outcome. Treatment initiation and modification should be done, with preparation and anticipation for regional anesthesia, blood loss and appropriate styptic measures.

Keywords: Gestational thrombocytopenia

INTRODUCTION

Thrombocytopenia in pregnancy is the second most common hematologic disorder after anemia. It is encountered in around 7-12% of the pregnancies. It can be a result of a wide variety of causes, and some of them can be pregnancy related. Diagnosis is usually made after a thorough laboratory evaluation. Signs and symptoms are largely associated with the etiology of the thrombocytopenia, gestational thrombocytopenia and ITP being an exception. They both are diagnosis of exclusion. Except in severe degrees of ITP, severe bleeding manifestations are rarely encountered.

Management of thrombocytopenia in pregnancy poses a different challenge. Fetal considerations, optimal timing of delivery, issues regarding regional anesthesia, peripartum complications and presence of other comorbid conditions, pose a management dilemma to the obstetricians. In the present case report, we present a case of recurrent, severe gestational thrombocytopenia, in the light of brief review of literature.

CASE REPORT

A 20 years G2 P1 L0 at 35 weeks 6 days of gestation, with documented severe thrombocytopenia (PLT-20000), presented to our labor unit after being referred from a local practitioner. Her past medical and family history was negative for any bleeding disorders. She had positive obstetric history. One year back, she presented to our hospital in second stage of labor with ante partum...
hemorrhage and fetal distress and cord prolapse. She delivered a still born female and had mild PPH, which was managed medically. Her work up revealed anemia (Hb-7.8 gm %), and severe thrombocytopenia (PLT-6000), other laboratory parameters, including SLE profile was normal. She was shifted to ICU and was transfused with 6 units of platelet and one unit of packed cell transfusion was done. She had good postnatal recovery and was discharged on day 6, with a platelet count of 1 lakh 12 thousand.

Table 1: Selected common causes of thrombocytopenia during pregnancy.

<table>
<thead>
<tr>
<th>Disease</th>
<th>Incidence during pregnancy (%)</th>
<th>Diagnostic features</th>
<th>Lab finding</th>
<th>Clinical features and symptoms</th>
<th>Pathophysiology</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gestational TCP</td>
<td>75%</td>
<td>Onset at late second or third trimester</td>
<td>PLT&gt;70 lakhs</td>
<td>Typically normal</td>
<td>unclear</td>
<td>Largely, a diagnosis of exclusion. Resolution of thrombocytopenia postpartum. No fetal thrombocytopenia.</td>
</tr>
<tr>
<td>ITP</td>
<td>&lt;1</td>
<td>Onset any trimester</td>
<td>PLT &lt; 1 lakh+/-large PLT on PBS</td>
<td>May have signs of bleeding, bruising, petechiae</td>
<td>Antibody induced peripheral PLT destruction. Decreased thrombopoiesis.</td>
<td>Diagnosis of exclusion. May be associated with fetal thrombocytopenia.</td>
</tr>
<tr>
<td>HELLP</td>
<td>&lt;1</td>
<td>70 % onset in third trimester 30 % onset postpartum</td>
<td>MAHA Elevated LFT Elevated LDH</td>
<td>Any or all signs of PE. 15-20% cases no HTN or proteinuria</td>
<td>Systemic endothelial dysfunction.</td>
<td>Variant of PE.</td>
</tr>
<tr>
<td>TTP/HUS</td>
<td>&lt;0.01</td>
<td>Any trimester, commonly third or postpartum</td>
<td>MAHA CRP increased Schistocytes on PBS</td>
<td>Fever, abdominal pain, visual changes, altered mentation</td>
<td>ADAMTS13 deficiency. Complement dysregulation(HUS).</td>
<td>ADAMTS13 activity &lt;5% in TTP. LFT, BP normal.</td>
</tr>
</tbody>
</table>

In the present pregnancy, her platelet count was 20 thousand and manual platelet count was 30 thousand. Hb was 7 gm%. General blood picture showed dimorphic microcytic hypochromic anemia with no evidence of megakaryocytes. Serum levels of Vitamin B12 and folate were normal. Liver and kidney functions, viral markers and other laboratory evaluation was also normal. Coagulation profile revealed no abnormality. There was no history of fever or drug exposure. She had normal blood pressure on examination. Tourniquet test was negative, there was no splenomegaly. However obstetric examination revealed small for gestational age fetus and decreased amount of liquor. Ultrasound showed corresponding fetal biometry and AFI of 1-2 with estimated fetal weight of 2 kilograms.

Antenatal corticosteroids were given to her and she was put on strict feto-maternal surveillance. Meanwhile, six unit’s platelet and one unit of packed red cells were transfused to her. On fourth day of her admission, she was taken up for elective cesarean delivery in view of small for date fetus with severe oligohydramnios, and delivered a female weighing 2 Kg. There was minimal surgical bleed. Neonate had normal platelet counts on day 1 and day 4 of delivery. Repeat serial platelet counts in mother revealed a spontaneous recovery pattern and on day 6 of delivery her platelet count was 1 lakh 3 thousand.

She was discharged in a healthy condition, and after six weeks her platelet count was 1 lakh 46 thousand.  

**DISCUSSION**

Physiologic thrombocytopenia in pregnancy is believed to occur because of hemodilution and accelerated platelet destruction, but rarely do counts fall below 1 lakh. It is observed to affect around 7-12% obstetric population at term. Gestational thrombocytopenia is found in 5-11% of all pregnancies and occurs due to increased platelet activation and consumption. There are no confirmatory laboratory tests available and diagnosis is largely based on history and postpartum surveillance. Serious morbidity to mother or fetus has not been known. Some associated comorbidities can increase the incidence of thrombocytopenia. At term, thrombocytopenia can be due to gestational thrombocytopenia (75%), hypertensive disorders of pregnancy (20%), autoimmune etiology as...
ITP and SLE (3%) and some rare causes, accounting for <2% of the total share. ITP affects 3% of thrombocytopenic mothers antenatally, and occurs due to increased platelet destruction. Antiplatelet antibodies are known to cross placenta and can result in serious fetal thrombocytopenia in around 12% cases. Diagnosis of this entity also depends on clinical manifestations. Antiplatelet antibody test is not a good test to establish diagnosis and it should not be done to differentiate between GT and ITP.

In the present case there was severe thrombocytopenia, so a work up plan, to exclude causes of severe thrombocytopenia, was initiated. Although her pre-conception and early trimester platelet counts were not known, however she had history of severe thrombocytopenia in previous pregnancy. A detailed history of hematuria and GI bleed and a medical examination for petechiae, ecchymosis was done, and it was negative. Peripheral blood examination ruled out pseudo thrombocytopenia, megalakaryocytes, schistocytosis and other cell lineage abnormalities for aplastic anemia. Minor bleed in thrombocytopenia is seen in dependent parts of lower limbs. Moderate bleeds manifest as epistaxis and excessive bleeding following minor trauma, whereas GI bleed, hematuria and intracranial hemorrhage are considered as severe forms of thrombocytopenia. All bleeding patterns were negative in this patient and absence of splenomegaly suggested an autoimmune variety.

Gestational thrombocytopenia is a transient condition occurring during pregnancy and is cured spontaneously as is seen in the present case. She was investigated for other causes and was not found to have any other etiological factor. Along with thrombocytopenia she had microcytic hypochromic anaemia also, for which she was given packed red cells. Platelet transfusions were given with an aim to build platelet counts to a safe level of 50,000/cmm. Obstetrical management was carried by cesarean delivery in view of small for gestational age fetus with AFI < 2 cm. Interestingly, in postpartum period there was spontaneous and complete remission, the platelet count increased to 1 lakh 3 thousand, which would have been unlikely in the case of ITP. Her previous pregnancy records also revealed a spontaneous recovery of platelets. The contradictory aspect of gestational thrombocytopenia in this case is very low platelet count which usually occurs in ITP, but similar low platelet counts have been reported by Ramadan KM et al.

Although we did not give any other treatment for thrombocytopenia, except blood and platelet transfusion, but suggested treatment includes steroids and intravenous immunoglobulins, anti-D immunoglobulin and splenectomy, if ITP is strongly suspected. ITP cases need treatment and success rates ranges from 30-50%. The platelet count has to be kept more than 50,000/cmm and route of delivery has to be decided as per the obstetric indications, but NSAIDs, salicylates, ventouse and forceps are avoided.

The ideal fetal and neonatal outcomes depend on avoidance of immunosuppression and corticosteroids, especially in first trimester. These are associated with cleft palate and other malformations. Cord blood should be screened in suspected cases of ITP and newborns should be monitored for thrombocytopenia. In our case there was no evidence of neonatal thrombocytopenia on serial monitoring. It is wiser to monitor neonates until two weeks of age.

CONCLUSION

We managed a case of severe, recurrent gestational thrombocytopenia. To distinguish between ITP and Gestational Thrombocytopenia in case of newly diagnosed severe thrombocytopenia is difficult. In our case, not only the clinical picture was contrasting, history also was of no valuable help. Final conclusion could only be reached after postpartum surveillance. As far as management is concerned, both the entities are to be treated in the same manner, especially in cases of severe grades, so as to maintain the safe level of PLT for styptic measures and other serious causes should be ruled out simultaneously. Neonatal screening is recommended in such cases.

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REFERENCES


