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Case Study

AN UNUSUAL CASE OF GESTATIONAL THROMBOCYTOPENIA SUPERIMPOSED BY VIRAL INFECTION- A CASE REPORT

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ABSTRACT

Thrombocytopenia is classically defined as a platelet count of less than 150,000/L. It affect 6-10% of all pregnancy and second only to anemia as the most commonly encountered blood disorder in pregnancy. It may result from diverse etiology- gestational thrombocytopenia, idiopathic thrombocytopenic purpura, von willibrand disease, viral and bacterial infections, preeclampsia complicated by HELLP, hemolytic uremic syndrome, thrombotic thrombocytopenic purpura etc.

KEYWORDS :gestational thrombocytopenia, viral infection

CASE

A 24 yr old primigravida female reported in our department with the complaints of amenorrhoea 8 months. Patient had significant history of chicken pox 25 days back. Review of the documents patient had brought with her showed Haemoglobin 8.3 gm/dl and platelets counts 38,000/ml (18 days back). There was history of transfusion of 1 unit of fresh whole blood and 1 unit of Random Donor Platelet and after that haemoglobin 10.3gm/dl and platelet count was 34,000/ml (14 days ago) so the patient was referred to our hospital. Patient's expected delivery date was 24 august 2014.

General physical examination- patient was active, conscious, well oriented to time, place and person. She had old chicken pox scar marks all over body. There were no bruises, petechiae, ecchymosisvisible on skin. She was afebrile with pulse rate 86/min, respiratory rate 18/ min and blood pressure 120/80 mm of hg. The findings on heart and lungs were normal. There was no oedema, no icterus and no lymphadenopathy.

Per abdominal examination revealed no hepatomegaly, 34-36 week fundal height with longitudinal presentation lie cephalic, fetal heart rate was registered 140/ min regular, uterus was relax and patient was perceiving fetal movements.

Ultrasound scan registered singleton vital pregnancy of 34-36 week with longitudinal lie with eutrophic fetalgrowth , normally implanted placenta and adequate volume of amniotic fluid. Ultrasound whole abdomen upper abdomen normal, liver normal echo texture, normal portal vein diameter, gall bladder normal, spleen normal size, normal splenic vein diameter, bilateral kidney normal

The patient was admitted in the high risk pregnancy ward in order to find out the cause of thrombocytopenia and correct it before labour.

complete laboratory tests :

i. Hb- 10.3 gm/dl,

ii. WBC- 5.43 Ч 10^3/µl,

iii. RBC 2.83 Ч 10^6/µl,

iv. HCT- 31.9%,

v. platelet 34 Ч 10^3/µl,

vi. Bleeding time 135 seconds,

vii. Clotting time 340 seconds,

viii. Prothrombin time INR 0.9%,

ix. D-dimer negative.

x. Biochemical analysis (renal function tests and liver function tests)did not indicate presence of neither HELLP syndrome nor disseminated intravascular coagulation

We started treatment with vitamin C, vitamin B complex , Iron Folic acid and Calcium. The next day patient was examined by consulting haematologist and all blood investigations were repeated including CBC, RFT, LFT, Thyroid profile, complete coagulation profile, PBF, ANA, Anti ds DNA, coombs test, viral markers, serum vitamin B12 and serum folic acid levels.

Investigations revealed

i. Hb - 11.6 gm/ dl,

ii. WBC - 5.08 Ч 10^3/µl,

iii. RBC -3.23 Ч 10^6/µl,

iv. HCT - 35.9%,

v. Platelets 34 Ч 10^3/µl.

vi. PBF - RBC - mainly normocytic normochromic with occasional micro as well as macro-ovalocytes. WBC- counts with in normal range, however majority of cells were lymphocytes. No blast cell seen Platelets – markedly reduced with giant platelets. No haemoparasite seen

vii. Anti HCV, Hbs Ag, HIV I & II - negative

viii. Dengue IgG, IgM - negative

ix. S. TSH -normal

x. S. Vit B12 - normal

xi. S. folate -normal

xii. ANA / ds DNA - negative

xiii. Coombs test - Direct & Indirect negative

xiv. Patient was prescribed prednisone 1 mg/ kg PO and advised repeat CBC after 5-7 days. Repeat CBC after 7 days showed no improvement in platelet counts. Case was discussed with consulting physician and patient was given Inj. Methyl prednisolone 1000 mg in 100 ml NS IV over 45 minutes OD for three days. Repeat investigation after 3 days showed no improvement. So we came to diagnosis as gestational thrombocytopenia superimposed by viral infection.

Till then patient completed 38 weeks so induction of labour was decided as there was no obstetric or fetal contraindication for normal labour. Patient was induced with prostaglandins. Platelets, FFP, and blood was arranged to combat any emergency. She delivered a healthy male child weight 3.4 kg with APGAR 9/10 at one minute and 5 minute. Active management of 3rd stage of labour was done.No episode of PPH occurred. Prophylactically inj. carboprost 250 µg IM given. IV antibiotics were given. On 1st postpartum day patients vitals were stable and condition was good. There was no bleeding. Treatment was continued with antibiotics, analgesics and oral iron preparation with continual monitoring of platelet count. Blood tests for the newborn child were done immediately after birth and all the tests for complete blood count, biochemical analysis, coagulation factors were normal. Ultrasound scan of baby's brain was normal. Both the mother and baby were discharged on request on the fourth postpartum day with mothers platelet count of 48Ч 10^3/µl. On follow up after 2 weeks patients platelet count were 86Ч 10^3/µl and after 4 weeks 109 Ч 10^3/µl and after 8 weeks 152 Ч 10^3/µl.

DISCUSSION

GT is an incidental finding. No diagnostic test exists to accurately distinguish GT from ITP2, 10.

Silver et al.3 said that the degree of thrombocytopenia is usually mild to moderate, remaining greater than 70 000/µL and the lower level has never been established. James et al.11 said that in GT platelet count will not go below 40 000–50 000/µL.

In our case of GT, we had a patient with PLT = 38000/-L. The patient was healthy, and preeclampsia and HELLP syndrome were excluded after the blood and biochemistry tests had been done. Considering the fact that thrombocytopenia started in the third trimester of pregnancy, and that the patient's platelet count was not decreased earlier during pregnancy, nor she had problems with bleeding, ITP was also excluded.

Federiciet al.9 said that thrombocytopenia has many potential causes, but three are responsible for almost all cases: GT 74%, preeclampsia and HELLP syndrome 21% and ITP 4%. Some authors said that no treatment is necessary for GT1,10,12. As it was demonstrated, there are extreme rare cases of GT requiring involvement of the entire team of experts (obstetrician, internal diseases specialist, hematologist and transfusion specialist), extensive treatment and monitoring of the patient.

Kadir and Mc Lintok1 said that the mode of delivery is determined by obstetric/maternal indication. In our case, we insisted on vaginal delivery as there was no obstetric contraindication. There were no complications during labour itself and the postpartum period due to intensive monitoring and active management of labour. In Burrow's8 large 1993 study, 756 out of 1027 (73.6%) women who were thrombocytopenic had GT. Burrows8 concluded that GT is the most frequent type of thrombocytopenia and

poses no apparent risks for either the mother or infant during delivery.

Blood tests for the newborn child were done immediately after birth and all the tests for complete blood count, biochemical tests, coagulation factors, time of bleeding and coagulation were normal. Ultrasound scan of baby's brain was also normal. Samuels et al.7 evaluated 162 pregnant women and their infants with thrombocytopenia, 74 with presumed GT, no infant from a GT gravida had a platelet count less than 50 000/μL or intracranial hemorrhage.

Kamphuis and Oepkes6 said that there is no risk for fetal hemorrhage or bleeding complications in GT. Both the patient and the child were discharged on the fourth day following the delivery. During the following eight weeks the patient had regular checkups, and the platelet count was back to normal. Most authors said that the platelet count returns to normal within 2–12 weeks postpartum3,4,5,10. A rapid return to normal confirms the diagnosis of GT, whereas continued thrombocytopenia after delivery gives diagnosis of ITP11. Thrombocytopenia is an infrequent but occasionally severe manifestation of varicella zoster infection [15].

More commonly thrombocytopenia occurs several days to a week after the eruption of varicella skin lesion, following the acute viremic stage. Several studies suggest that these cases are secondary to platelet destruction, most likely by cross reactive antibodies.[17,18] one early study using electron microscopy demonstrated direct viral infection of megakaryocytes.[16]Pathologic complications of VZV infection include thrombocytopenia, which causes coagulopathy and haemorrhage, particularly when associated with severe hepatitis [13,14]. Thrombocytopenia may be caused by reduced production and survival of platelets; vasculitis, transient hypersplenism, or intravascular coagulopathy may contribute to lower platelet counts. Thrombocytopenia may be related to antibody-mediated destruction of platelets [13, 14]

CONCLUSION

Pregnancy complicated with thrombocytopenia is a relatively common occurrence and challenge to the obstetrician. The myriad of disease processes, either pregnancy-induced disorders or preconception medical conditions, can cloud the correct diagnosis. The great majority of patients with gestational thrombocytopenia have a benign condition, but a minority of patients who have a more serious disease are at risk for serious morbidity and mortality.

With a thorough history, physical examination, focused laboratory evaluation, and multidisciplinary approach including physician and hematologists, these patients uniformly have favorable outcomes and can be safely managed. The aim of this case report is that special attention should be given to patients with thrombocytopenia as awareness of these many causes facilitate proper diagnosis and management of thrombocytopenia in pregnancy setting.

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