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Case Report

Thrombocytopenia in early pregnancy predicting partial haemolysis, elevated liver enzyme and low platelet count syndrome: a case report and review of literature

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ABSTRACT

The incidence of thrombocytopenia in pregnancy is 6-10% and is classically defined as a platelet count of less than 150,000/ L. Counts less than 100,000 to 150,000/L are considered mild, 50,000 to 100,000/L as moderate, and less than 50,000/L are considered as severe thrombocytopenia. It is the second most common hematological condition in pregnancy with anaemia being the leading cause. Thrombocytopenia may be related to disorders that are intrinsic to pregnancy such as gestational thrombocytopenia that is seen in three-fourths of all cases. The second common cause is hypertensive disorders in pregnancy more commonly seen in severe pre-eclampsia in 21% and in HELLP (haemolysis, elevated liver enzyme and low platelet count) that accounts for 12% of thrombocytopenia cases in pregnancy. This case report revisits the diagnosis of partial HELLP under the background of preeclampsia that warrants aggressive treatment like complete HELLP syndrome to optimize the maternal and fetal outcome.

Keywords: Pregnancy, Thrombocytopenia, HELLP syndrome

INTRODUCTION

The incidence of thrombocytopenia in pregnancy is 6-10% and is classically defined as a platelet count of less than 150,000/L. Counts less than 100,000 to 150,000/L are considered mild, 50,000 to 100,000/L as moderate, and less than 50,000/L are considered as severe thrombocytopenia. It is the second most common haematological condition in pregnancy with anaemia being the leading cause.[1] Thrombocytopenia may be related to disorders that are intrinsic to pregnancy such as gestational thrombocytopenia that is seen in three-fourths of all cases. The second common cause is hypertensive disorders in pregnancy more commonly seen in severe pre-eclampsia in 21% and in HELLP (haemolysis, elevated liver enzyme and low platelet count) that accounts for 12% of thrombocytopenia cases in pregnancy. The thrombocytopenia in preeclampsia is usually moderate, and haemorrhage is an uncommon feature, unless the patient develops disseminated intravascular coagulation related to HELLP syndrome.[2-4] However, isolated thrombocytopenia may still be the only biochemical abnormality as referred to partial HELLP that suggest worsening hypertensive disease. There is paucity of data on clinical characteristics and maternal and perinatal outcomes of partial HELLP syndrome.[5] We report a clinical case of pregnant patient who develops partial HELLP syndrome with severe thrombocytopenia unusually related to mild preeclampsia. The absence of definitive clinical criteria and overlapping clinical characteristics with immune thrombocytopenic purpura has resulted in significant diagnostic and therapeutic challenge.

CASE REPORT

A 43-year-old woman at 40 + 2 weeks pregnant (gravid 7, para 4) presented to obstetric patient assessment centre (PAC) with new onset high blood pressure. The mother's blood pressure was found to be 140-147/95 mmHg on two separate occasions. Fetal heart monitoring trace showed reactive fetal tracing. Laboratory evaluation revealed urine protein 2+, moderate thrombocytopenia (65x10 9/L), normal haemoglobin and hematocrit, normal liver function, normal renal function tests, and coagulation profile. Her previous antenatal follow up at the peripheral health clinic revealed decreasing trends of platelets from second trimester (Table 1). However, there were no signs and symptoms of gestational hypertension during the follow up. The full blood picture was suggestive of large platelets and normal blood morphology. The patient was admitted with the diagnosis of severe preeclampsia for further monitoring and serial laboratory evaluations. A repeat full blood count in 24 hours revealed severe thrombocytopenia (24,000 cells/mm^3). The further plan at this point was to optimize her platelet count with platelet transfusion. With 8 units of platelets transfusion the count improved to 44,000cell/mm^3. She was induced with Foley's catheter and after 4.5 hours in active labour, she successfully delivered a 3.2 kg male baby with Apgar score of 9/10. Following two hours post-delivery, she had an episode of major postpartum haemorrhage with estimated blood loss of 1.1 liters that was managed conservatively. The platelet count was 33,000cells and haemoglobin of 8.2g/dl following postpartum haemorrhage and 4 units of platelets transfused. Her further clinical course was uneventful and platelet count on second day postpartum improved to 69,000 cells/mm^3. There was no neonatal thrombocytopenia. At postnatal follow up two weeks later, thrombocytopenia resolved and the platelet count was 330,000 cells/mm^3.

Table 1: Platelet trend during pregnancy.

Parameter	24 weeks	28 weeks	34 weeks	40 weeks
Platelet count 150-400x10^9/L	132x10^9/L	126x10^9/L	112x10^9/L	65x10^9/L

DISCUSSION

In our case, we had the patient presenting with new onset hypertension in third trimester. She was asymptomatic and blood pressure was well controlled with mild proteinuria. Besides isolated severe thrombocytopenia, the blood and biochemistry results were normal. Gestational thrombocytopenia although the onset is common in the third trimester, is unlikely to result in severe thrombocytopenia. A transient mild thrombocytopenia is seen physiologically in pregnancy due to increased platelet consumption and hemodilution.[6] The causes of thrombocytopenia from pregnancy-induced hypertension and HELLP syndrome is thought to be related to abnormal vascular tone with resultant accelerated platelet destruction, activation, and consequent coagulation defects.[7] Some authors are of the opinion that thrombocytopenia may precede the various other manifestations and thus preeclampsia should be considered in the event of isolated thrombocytopenia seen in the late second or third trimester.[8]

Considering the fact that there was no history of previous bleeding or low platelet count, immune thrombocytopenia could have been excluded. The presence of large platelets in the peripheral blood picture was suggestive of ITP or microangiopathies or DIC secondary to complication of severe preeclampsia related to HELLP syndrome. It is of note that 15-20% of patients presenting with HELLP syndrome do not have severe eclampsia or antecedent hypertension.[9] We are of the opinion that our patient is case of partial HELLP syndrome with feature of isolated thrombocytopenia that was probably a marker of disease progression from partial HELLP to complete HELLP over a period of time.

The incidence of HELLP syndrome is 1:1000 pregnancies and is seen in 4-12% of pregnancies complicated with severe preeclampsia or eclampsia. The term HELLP was coined by Weinstein to describe this severe complication of pregnancy that is characterized by haemolysis, elevated liver enzymes and low platelet count.[10] Two main classifications for HELLP syndrome that are commonly used include: The Tennessee System classification which is based on the assessment of the following parameters: AST>70 UI/L, LDH >600 UI/L, thrombocytes <100,000/ mm. In the presence of one or two of the characteristics of this syndrome (ELLP, HL, and LP) it is termed as Partial HELLP Syndrome (PHS) similar to our case presented with isolated thrombocytopenia (LP).[11] The Mississippi classification relies on the thrombocyte counts: class I (<50,000/mm), class II (50,000-100,000/mm) and class III (100,000-150,000/mm). The incidence of partial HELLP although is unclear is probably estimated around 21 to 24%.[12] It is considered that HELLP syndrome may begin as partial HELLP, and becomes a progressive disease with the alterations in the laboratory tests takes place after different elapsed times.

In the clinical practice of HELLP syndrome, there is less evidence among the Asian women about the difference in maternal and perinatal outcomes based on the disease distinction of HELLP syndrome. Few studies focused on the specific prognosis of this condition. There is no information about incidence of partial HELLP syndrome in Malaysia. Earlier observations by Audibert et al suggest that there are fewer incidences of DIC and maternal and perinatal complications observed among women with partial HELLP over complete HELLP syndrome.[13] This emphasizes the fact of recognizing the condition distinctly from HELLP and the option of more conservative management. This is further supported by retrospective observations by Abbade et al, that there was

a high rate of caesarean section rates and preterm delivery due to interruption of pregnancy with diagnosis of partial HELLP syndrome and such decisions needs review.[14] These findings were contradictory to later observations indicating that complete HELLP is associated with slightly high risk of maternal complications such as acute renal failure and DIC in comparison to partial HELLP with no difference in neonatal, long term and subsequent pregnancy outcomes.[15] Further support to these findings in an Asian study by Rakshit et al concluding partial HELLP has similar grave prognosis as complete HELLP. The evidence is being conceivably obvious that both partial HELLP and complete HELLP are the continuum in the natural evolution of the same disease.[17] The flow chart (Figure 1) illustrates the initial approach and management of thrombocytopenia in pregnancy.

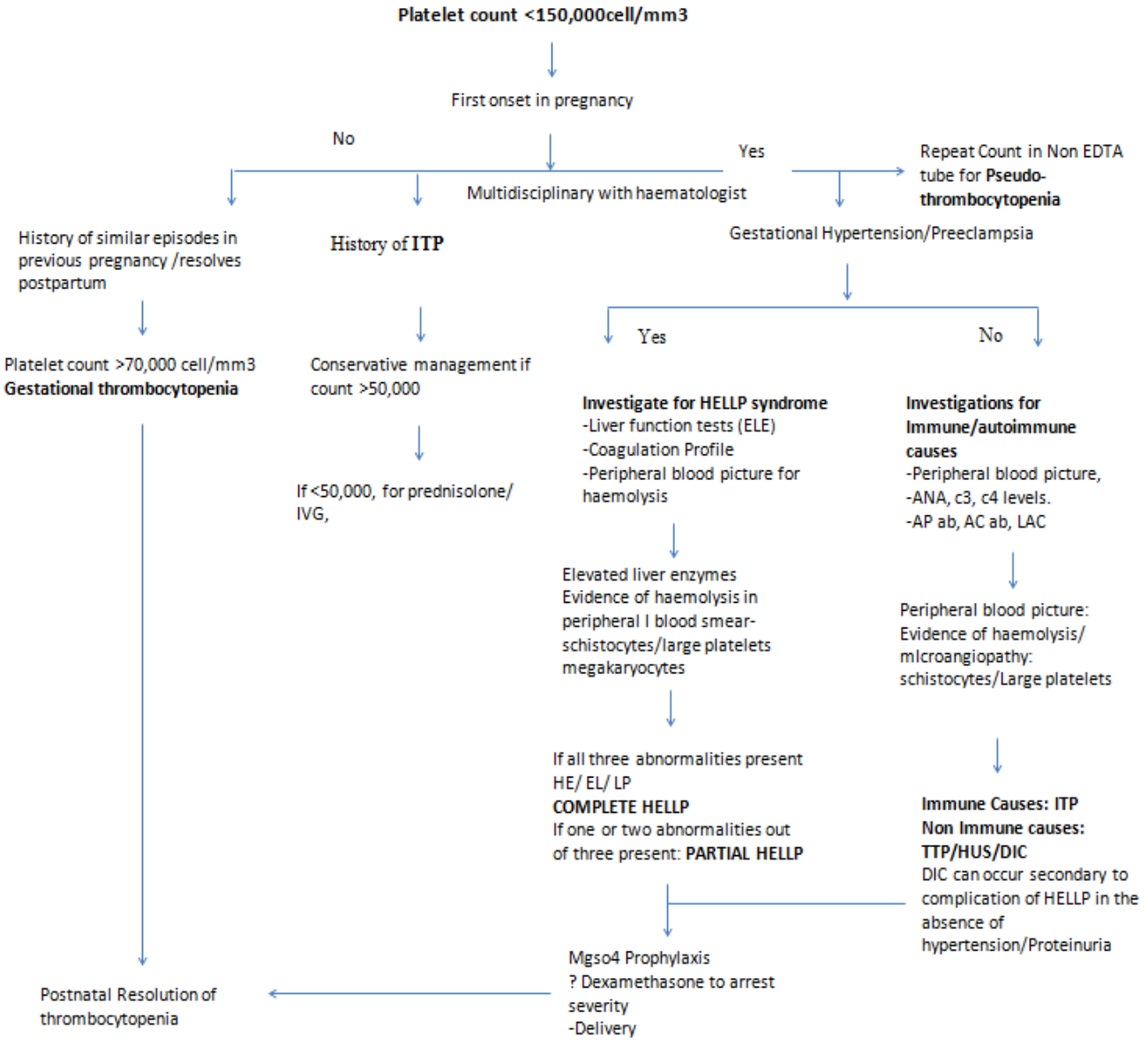

Figure 1: Flow chart in the initial assessment of thrombocytopenia.

The treatment goals include arrest, reverse, and shorten the disease process and prevent major maternal and neonatal morbidity. Besides antihypertensive and seizure prophylaxis, corticosteroids are advocated to resolve the clinical and biochemical parameters of the disease. However, the recent evidence suggests insufficient data for benefits of overall clinical improvements, but justified in situations that mandates increased rate of recovery of platelets.[16,17] This include any patient with class 1 or class 2 HELLP syndrome regardless of gestational age or any patient with class 3/partial-incomplete HELLP syndrome regardless of gestational age who has eclampsia, severe epigastric pain, severe hypertension, or any major organ system morbidity.[18]

Future research

The future research is to study the repercussions of partial HELLP syndrome on maternal and fetal outcomes in comparison to complete HELLP, gestational hypertension or severe pre-eclampsia to establish diagnostic and treatment protocols in the local context.

CONCLUSION

This case report reiterates the need to classify women with hypertension in pregnancy with clinical and biochemical evidence of HELLP in accordance to the diagnostic criteria (Tennessee and Mississippi classifications). This facilitates early diagnosis, close supervision and monitoring and early intervention to optimize the maternal and perinatal outcome. In light of diagnostic dilemma that emerged from our report, we recommend considering the diagnosis of partial HELLP in the background of preeclampsia that warrants aggressive treatment like complete HELLP syndrome.

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REFERENCES

1. Shehata N, Burrows R, Kelton JG. Gestational thrombocytopenia. Clin Obstet Gynecol. 1999;42:327-34.
2. Burrows RF, Kelton JG. Thrombocytopenia at delivery: a prospective survey of 6,715 deliveries. Am J Obstet Gynecol. 1990;162:731-4.
3. Magann EF, Martin JN. Twelve steps to optimal management of HELLP syndrome. Clin Obstet Gynecol. 1999;42:532-50.
4. Sullivan CA, Martin JN. Management of the obstetric patient with thrombocytopenia. Clin Obstet Gynecol. 1995;38:521-34.
5. Rath W, Faridi A, Dudenhausen JW. HELLP syndrome. Peri-nat Med. 2000;28:249-60.
6. Veena HC, Manjunatha S, Itagi V, Taklikar RH, Patil RS. The hemostatic mechanisms in PIH. Indian J Appl Basic Med Sci. 2015;17:40-4.
7. Silver R, Berkowitz R, Bussel J. Thrombocytopeniain pregnancy. Practice bulletin, No 6. Chicago: American College of Obstetrics and Gynecology; 1999.
8. McCrae KR. Thrombocytopenia in pregnancy. Hematology Am Soc Hematol Educ Program. 2010:397-402.
9. Sibai BM. Diagnosis, controversies, and management of the syndrome of hemolysis, elevated liver enzymes, and low platelet count. Obstet Gynecol. 2004;103:981-91.
10. Weinstein L. Syndrome of haemolysis, elevated liver enzymes and low platelet count: a severe consequence of hypertensionin pregnancy. Am J Obstet Gynecol. 1982;142:159-67.
11. Sibai BM. Treatment of hypertension in pregnant woman. N Eng J Med. 1996;335:257-60.
12. Sibai BM. The HELLP syndrome (haemolysis, elevated liver enzymes, and low platelets): much ado about nothing. Am J Obstet Gynecol. 1990;162:311-6.
13. Abramovici D, Friedman SA, Mercer BM, Audibert F, Kao L, Sibai BM. Neonatal outcome in severe preeclampsia at 24 to 36 weeks' gestation: does the HELLP hemolysis, elevated liver enzymes, and low platelet count) syndrome matter? Am J Obstet Gynecol. 1999;180:221-5.
14. Abbade JF, Peracoli JC, Costa Araujo RA. Sao Paulo Med J/Rev Paul Med. 2002;120(6):180-4.
15. Liu CM, Chang SD, Chang PJ, Cha AS. Comparison of maternal and perinatal outcomes in Twainese women with complete and partial HELLP syndrome and women with severe preeclampsia without HELLP. Obstet Gynaecol. 2006;32(6):550-8.
16. Rakshit A, Lahiri S, Biswas SC, Dey R, Roy BR, Saha MM. A study to detect HELLP syndrome and partial HELLP syndrome among pre-eclamptic mothers and their impact in fetomaternal outcome Al Ameen J Med Sci. 2014;7(1):20-5.
17. Martin JN, Owens MY, Keiser SD, Parrish MR, Tam Tam KB, Brewer JM, et al. Standardized Mississippi protocol treatment of 190 patients with HELLP syndrome: slowing disease progression and preventing new major maternal morbidity. Hypertens Pregnancy. 2012;31:79-90.
18. Woudstra DM, Chandra S, Hofmeyr GJ, Dowswell T. Corticosteroids for HELLP (hemolysis, elevated liver enzymes, low platelets) syndrome in pregnancy. Cochrane Database Syst Rev. 2010;8(9):CD008148.

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