RESEARCH ARTICLE

Platelet count estimation in pregnancy-induced hypertension and assessment of the degree of thrombocytopenia

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ABSTRACT

Background: Gestational thrombocytopenia is the most common cause of thrombocytopenia and pregnancy-induced hypertension (PIH) is the second common cause. The degree and the incidence of thrombocytopenia depend on the severity of the disease process. Lower is the platelet count, greater will be the maternal and fetal morbidity and mortality. Early diagnosis and assessment of severity of PIH is necessary to prevent complications such as HELLP syndrome and maternal and fetal morbidity and mortality. Therefore, platelet count plays an important role in the functional evaluation of the hemostatic system. **Aims and Objectives:** The aims of the study were as follows: (i) To estimate the platelet count in PIH and (ii) to assess the degree of thrombocytopenia in PIH based on platelet count. **Materials and Methods:** The study group consisted of 120 pregnant women, of which 90 were diagnosed to be with PIH, with varying severity. Thirty subjects were control group with no signs of hypertension. Blood pressure (BP) was recorded and platelet count was assessed by peripheral smear method (PM) in all 120 pregnant women. **Results:** Platelet numbers done by peripheral smear were found to be 2.56 ± 0.89 lac/mm^3 in mild PIH, 1.95 ± 1.12 lac/mm^3 in pre-eclampsia, 1.34 ± 0.91 lac/mm^3 in eclampsia, and 3.13 ± 0.96 lac/mm^3 in control group. The platelet count by the automatic analyzer was 2.47 ± 0.10 lac/mm^3 in mild PIH, 1.82 ± 1.02 lac/mm^3 in pre-eclampsia, 1.29 ± 0.83 lac/mm^3 in eclampsia, and 2.92 ± 0.98 lac/mm^3 in control group. This shows that there is a decrease in platelet count with increase in severity of the disease. No statistical significant difference seen in both methods. **Conclusion:** Platelet count serves as an important tool for diagnosing the severity of the disease. Estimation of platelets from PM is a simple, rapid, easier, and cheaper method which can be used even at rural setup where automated counters are not available.

KEY WORDS: Pregnancy-induced Hypertension; Platelet Count; Peripheral Smear; Thrombocytopenia

INTRODUCTION

Pregnancy-induced hypertension (PIH) is one of the disorders that complicate pregnancy.[1] PIH is defined as hypertension that occurs in pregnancy after 20 weeks of gestation but disappears after delivery.[2]

PIH causes complications in 12–22% of all pregnancies.[3]

PIH is classified into:[2]
(i) Mild PIH,
(ii) Pre-eclampsia, and
(iii) Eclampsia.

Mild PIH is defined as blood pressure (BP) of 140/90 mmHg which returns to normal by 12 weeks of postpartum.

Pre-eclampsia is the presence of hypertension (BP >140/90) and significant proteinuria (300 mg/24 h) and/or edema.

Eclampsia is the occurrence of convulsion or coma unrelated to other cerebral conditions with signs and symptoms of pre-eclampsia.

There is a gradual rise in the incidence of PIH over the past few decades. Women with increased BP during pregnancy have significantly increased maternal and fetal morbidity and mortality. Hypertensive disorders of pregnancy are responsible for significant maternal and perinatal morbidity, and they are the second leading cause of maternal morbidity due to embolism.[3]

There are several important pathophysiologic changes in PIH. These changes include hemodynamic changes due to alterations in blood volume and peripheral vascular resistance, alterations in hemostatic mechanism, and abnormal renal functions.[4] Coagulation abnormalities exist in only a minority of patients of severe preeclampsia, mild thrombocytopenia is the most common of these which affects <10% of the cases.[4]

Gestational thrombocytopenia is the most common cause of thrombocytopenia and pregnancy-induced hypertension (PIH) is the second common cause.[5-7] The degree and the incidence of thrombocytopenia depend on the severity of the disease process.[8] Lower is the platelet count, greater will be the maternal and fetal morbidity and mortality. Early diagnosis and assessment of severity of PIH is necessary to prevent complications such as HELLP syndrome and maternal and fetal morbidity and mortality. Therefore, platelet count plays an important role in the functional evaluation of the hemostatic system. As the cause of PIH is not fully established, it remains as a disease of theories.[7]

Placental implantation with abnormal trophoblastic invasion of uterine vessels is a major cause of hypertension which is associated with pre-eclampsia syndrome.[9,10] Various studies have shown that the degree of incomplete trophoblastic invasion of the spiral arteries is directly correlated with the severity of subsequent maternal hypertension. This incomplete invasion leads to placental hypoperfusion which results in release of systemic vasoactive compounds that cause an exaggerated inflammatory response, vasoconstriction, endothelial damage, capillary leak, hypercoagulability, and platelet dysfunction, all of these contribute to organ dysfunction and the various clinical features of the disease.[4]

Thrombocytopenia is the most common hematological abnormality found during pregnancy.[1] This can be detected by various tests such as prothrombin time, partial thromboplastin time, fibronectin level, decrease antithrombin level, α_2 antitrypsin level, and decrease in circulating free placental growth factor and vascular endothelial growth factor. These tests are more sensitive but expensive, time consuming, require well-equipped hospital, and not suitable for routine purpose. However, platelet estimation by smear method which is a daily routine laboratory test is easy, rapid, and cheap and can be done at any setup with no established laboratories. As the accurate count of platelets by automated cell counters is not feasible at all hospitals or setups, platelet estimation from peripheral blood smear is used in this study as an alternative method to estimate the degree of thrombocytopenia in PIH patients.[3]

MATERIALS AND METHODS

Ethical clearance was obtained from the Human Ethical Committee of Navodaya Medical College and Research Centre, Raichur.

The study group comprises 120 pregnant women, of which 90 were diagnosed to be with PIH, with varying severity of PIH.

Thirty subjects were control group with no signs of hypertension and three other groups, of which 30 were diagnosed with signs of mild PIH, 30 with pre-eclampsia, and 30 with eclampsia.

The subjects were clinically examined, proper and relevant history were noted, recording of vital signs was done which included recording of supine BP following standard protocol, for assessing of platelets, capillary blood was used which was drawn under complete aseptic precautions and smears were prepared immediately and stained using Leishman's stain following standard protocol.

Manual Method

Platelet count by study of blood smear.

Principle: A drop of blood is spread on the slide to prepare a thin smear and it is stained. Estimation of platelets is made by two methods.

Estimation of platelets is done by counting the platelets per 1000 red cells. The normal ratio of platelet to red blood cell should be 1:20.

In traditional method, platelets are counted in 10 oil immersion field and the average platelet count is taken for calculation.[11]

The total number of platelets in lac/mm³ is calculated by multiplying average number of platelets by 20,000.

Automated Analyzer[12]

Automated platelet counting is done with a Coulter counter. In this counter, the particles that are between 2 and 10 fL are

counted as platelets. A platelet graph is plotted according to the size distribution of the platelets counted.

RESULTS

The study titled "Estimation of platelet count in PIH and assessment of degree of thrombocytopenia" was conducted in the department of physiology.

This cross-sectional study consisted of 30 control and 90 PIH patients.

The study group was divided into four groups: Group I – control, Group II – mild PIH, Group III – pre-eclampsia, and Group IV – eclampsia.

BP was recorded in all the groups. Peripheral blood smear was prepared and the platelet estimation was done and automated platelet count of all the groups was obtained. The obtained data were tabulated.

BP

By *post hoc* analysis, it was observed that there was a significant difference of all groups from control group in systolic BP (SBP) (122.66 ± 4.82), where it was found that there was high SBP in eclampsia group (160.51 ± 13.52 mmHg) as compared to mild PIH (139.06 ± 8.81 mmHg) and pre-eclampsia (159.65 ± 15.69 mmHg). There was a significant difference of all groups from control group in diastolic BP (DBP) (82.4 ± 3.49) which, in turn, indicates that there was high DBP in eclampsia (111.67 ± 9.71 mmHg) and pre-eclampsia (111.24 ± 6.87 mmHg) as compared to mild PIH (91 ± 3.81 mmHg) and the values of pre-eclampsia and eclampsia were almost equal. There was a significant difference of all groups from the

control group in mean arterial pressure (MAP) (95.82 ± 2.74), there was high MAP in eclampsia (127.95 ± 9.38 mmHg) and pre-eclampsia (127.37 ± 8.47 mmHg) as compared to mild PIH (107.02 ± 3.47 mmHg) and the values of pre-eclampsia and eclampsia were found to be almost equal [Table 1].

Comparison of Platelet Count in Control and Study Groups by Peripheral Smear Method (PM) and Automated Method (AM)

There was no significant difference in platelet count by manual method and automated analysis in the control group ($P = 0.095$), pre-eclampsia group ($P = 0.191$), mild PIH group ($P = 0.192$), and eclampsia group ($P = 0.429$). Mean values of platelet count by PM and AM are shown in Table 2. The results show that there is a negative correlation between the platelet count and SBP, DBP, and pulse pressure.

DISCUSSION

SBP in all the groups ($P < 0.01$) showed statistically significant difference when compared to the control group. SBP and DBP show a highly significant statistical difference in pre-eclampsia and eclampsia when compared to the mild PIH and control group. This indicates the severity of the disease. Although the precise cause of pre-eclampsia is not completely understood, ischemia of the placenta and subsequent release of toxic factors by the placenta are believed to play a role in causing many of the manifestations of this disorder, including hypertension in the mother. Substances released by the ischemic placenta, in turn, cause dysfunction of vascular endothelial cells throughout the body, including the blood vessels of the kidneys. The raised SBP and DBP in the PIH patients are due to the maternal endothelial cell activation/endothelial dysfunction which is due to release of

Table 1: The mean±standard deviation values of BP in different groups

Group	Systolic BP	Diastolic BP	Mean arterial pressure
Control (Group I)	122.66±4.82	82.4±3.49	95.82±2.74
Mild pregnancy-induced hypertension (Group II)	139.06±8.81**	91±3.81**	107.02±3.47**
Pre-eclampsia (Group III)	159.65±15.69**	111.24±6.87**	127.37±8.47**
Eclampsia (Group IV)	160.51±13.52**	111.67±9.71**	127.95±9.38**
F-value	74.98	147.51	161.68
P-value	0.0001	0.0001	0.0001

**$P<0.05$. BP: Blood pressure

Table 2: Comparison of platelet count by manual method and automated analysis

Group	Manual method (lac/mm³)	Automated analysis (lac/mm³)	95% confidence interval of difference	*t*-value	Significance
Group I	3.13±0.96	2.99±1.02	−0.025–0.296	1.726	*P*=0.095
Group II	2.56±0.89	2.47±0.10	−0.045–0.213	1.34	*P*=0.192
Group III	1.95±1.12	1.82±1.02	−0.0706–0.337	1.337	*P*=0.191
Group IV	1.34±0.91	1.29±0.83	−0.0733–0.168	0.802	*P*=0.429

placental antiangiogenic factors and other multiple factors in plasma [8,13,14]

Platelet Count in PIH

There was a statistically significant difference in the platelet count in all the study groups when compared to the control group. And also, there was a statistically significant difference in platelet count in between the study groups. The platelet count was very much low in the IV group (eclampsia) when compared to the other two groups. This indicates that the severity of the disease increases with increased degree of thrombocytopenia. The study conducted by Mohapatra *et al.* and Dube *et al.* showed the same relation of platelet count with the severity of PIH.[15,16]

Activation of coagulation system in small vessels and increased platelet aggregation is seen in PIH. Therefore, it is clear that PIH is one of the causes of thrombocytopenia and the platelet count increases back to normal rapidly after the delivery.[17] There are studies which suggest that the platelet is stored in the areas of endothelial damage and caused thrombocytopenia.[15,18,19] Hence, thrombocytopenia in PIH is due to vascular endothelial damage, increased platelet activation, and increased platelet turnover or consumption.

The platelet estimation was done in mild PIH, pre-eclampsia, eclampsia, and the control group; the platelet estimation was compared with the automated platelet count in all the groups. There were no statistically significant differences between PM when compared with the AM, control ($P = 0.095$), Group II ($P = 0.192$), pre-eclampsia ($P = 0.191$), and eclampsia group ($P = 0.429$). A similar study was done by Mohapatra *et al.* and showed that there was no significant difference between the platelet estimation and the AM.[15]

Correlation of Platelet Count and BP

The correlation analysis between the platelet count and BP in the study group has shown that there is inverse relation between both. When the value of platelet number estimation was compared between the control group with pre-eclampsia and eclampsia, a significant decrease in platelet number was observed as the mean BP increased. That is platelet count decreases with the severity of the disease. Our study results go in accordance with the studies conducted by Mohapatra *et al.* and Annam *et al.* who have shown the inverse relationship between the platelet count and BP that indicate the severity of the disease. They have concluded that platelet count is an early method of assessing the severity of the disease.[15,17]

As platelet count is an important diagnostic tool, the platelet estimation by PM which is rapid, economical, and easier to perform at any setup can be very useful in the diagnosis of PIH, assessing severity of the disease, and also to prevent complications of PIH by early reference of the patient to the institutional management and thus can reduce the morbidity and mortality of both mother and the baby.

Strength of our study is that severity of PIH is assessed by simple and easy platelet counting by PM which can be done at any setup.

Limitation of our study is that other tests which are more sensitive such as prothrombin time, thromboplastin time, and fibronectin level are not done as they are more time consuming and require well-equipped laboratory facilities.

CONCLUSION

The results of this study suggest that as there is an inverse relationship between the platelet count and BP, platelet estimation which is a reliable, rapid, easy, and cheaper can be used for the assessment of severity of the disease depending on the degree of thrombocytopenia. Thus, platelet estimation can be helpful in preventing the occurrence of PIH complications such as eclampsia, HELLP, and disseminated intravascular coagulation by early referral of the patient for the institutional management.

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