

A Hospital-Based Assessment of Platelet Counts in Pregnancy: A Case-Control Study

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Abstract

Aim: The present study was conducted to assess the Platelet Count in the Pregnant Women.

Methods: The present study was planned in Department of Pathology for a period of 12 months. In the present study 200 samples received for the platelet estimation were enrolled in the present study. All the patients were informed consents.

Results: The demographic data obtained for the subjects showed that the most of the control group aged within 21 to 25 years (42%) while the majority of the pregnant women (42%) were within 26-30 years. There was a statistically significant difference in age and parity, between the cases and the control groups ($p < 0.0001$). Although, there was an increase in mean platelet count (PLT) in the pregnant women when compared with the non-pregnant group ($224.56 \pm 68.72 \times 10^9$ cell/L) versus $216.94 \pm 53.27 \times 10^9$ cell/L, the MPV, PDW, and platelet larger cell ratio were slightly lower in pregnant women than the non-pregnant group. These changes were not statistically different between the two study groups ($p > 0.05$). The mean PLT count in the first, second and third trimester of pregnancy ($226.64 \pm 44.26 \times 10^9$ cell/L, $236.34 \pm 82.38 \times 10^9$ cell/L and $210.44 \pm 62.08 \times 10^9$ cell/L) did not differ significantly from the mean PLT count of the non-pregnant women ($216.94 \pm 53.27 \times 10^9$ cell/L) ($p = 0.180$). Moreover, the mean MPV of pregnant women were lowest in 3rd trimester and significantly differ from pregnant women in their 1st, 2nd trimester and the control group ($p = 0.032$). There was no significant association between pregnancy status and thrombocytopenia.

Conclusion: Based on these findings, it can be inferred that platelet count and MPV decreases while PDW increase with the progression of gestation age compared to the non-pregnant women.

Keywords: Platelet Count, Pregnant Women, Mean Platelet Volume

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Introduction

The normal distribution of platelet counts at the time of delivery and the absence of health problems in the mother and infant suggested that gestational thrombocytopenia was the result of lower platelet counts that occur in all women during pregnancy. [1,2,4] The current thinking is that platelet counts in all women begin to decrease in the mid-second to third trimester and continue to decrease until the time of delivery. [3]

Hypertensive disorders of pregnancy (HDP) represent a group of conditions associated with high blood pressure during pregnancy, proteinuria and in

some cases convulsions. The most serious consequences for the mother and the baby result from pre-eclampsia and eclampsia. [5] These are associated with vasospasm, pathologic vascular lesions in multiple organ systems, increased platelet activation and subsequent activation of the coagulation system in the micro-vasculature. It is a multisystem disease and many theories are proposed for pathophysiology. So there is a constant search for better prognostic factors to predict the progression and severity of disease. The fall in platelet count is most frequently found in

preeclampsia and is probably due to consumption during low grade intravascular coagulation. [6]

The platelet count in pregnancy is slightly lower than in non-pregnant women. [7] Most studies report a reduction in platelet count during pregnancy, resulting in levels about 10% lower than pre-pregnancy level at term. [8,9] The majority of women still have levels within the normal range; however, if pre-pregnancy levels are border-line, or there is a more severe reduction, the level may fall below the normal range. The mechanisms for this are thought to be dilutional effects and accelerated destruction of platelets passing over the often scarred and damaged trophoblast surface of the placenta. [10] Platelet counts may also be lower in women with twin compared with singleton pregnancies, possibly related to greater increase of thrombin generation. [11] Thrombocytopenia is a common finding in pregnancy, occurring in 7–10% pregnancies. [12] It may be a diagnostic and management problem, and has many causes, some of which are specific to pregnancy.

Women with low platelet counts in pregnancy are generally less symptomatic due to the procoagulant state induced by increased levels of fibrinogen, factor VIII and von Willebrand Factor (VWF), suppressed fibrinolysis and reduced protein S activity. [13] Although most cases of thrombocytopenia in pregnancy are mild, with no adverse outcome for mother or baby, occasionally a low platelet count may be part of a complex disorder with significant morbidity and may (rarely) be life-threatening.

The present study was conducted to assess the Platelet Count in the Pregnant Women.

Materials and Methods

The present study was planned in Department of Pathology, Narayan medical college and hospital, Sasaram, Bihar, India for a period of 12 months. In the present study 200 samples received for the platelet estimation were enrolled in the present study. All the patients were informed consents.

Following was the inclusion and exclusion criteria for the present study.

Inclusion Criteria: The records of pregnant women with proved platelet abnormalities Normal pregnancy at 2nd- 3rd trimester.

Exclusion criteria: All cases with pre- existing hypertension other than PIH. Patients having co morbid conditions such as Severe anaemia, Diabetes mellitus, History of auto immune disorder, History of Idiopathic Thrombocytopenic Purpura, History of receiving drugs like aspirin, anti-coagulants etc.

Methodology

Relevant data were obtained from case files and compiled by a common proforma that included socio-demographic characteristics of mothers, obstetric history, signs and symptoms in mother at presentation, laboratory data, and maternal and perinatal outcomes. The data collection was followed by analysis of the collected data.

Clinical details of all cases were documented. Those cases with pre-existing hypertension, having associated co morbid diseases such as diabetes mellitus, auto immune disorders, ITP, neoplastic diseases, heart diseases and cases on anticoagulants were excluded from the study.

Results

Table 1: Association between age, parity and pregnancy status of study participants

Variables	Pregnancy Status		p-value
	Pregnant, n (%)	Non-Pregnant, n (%)	
Age (years)			
15-20	0 (0%)	20 (20%)	<0.0001
21-25	12 (12%)	42 (42%)	
26-30	42 (42%)	23 (23%)	
31-35	34 (34%)	8 (8%)	
36-40	12 (12%)	7 (7%)	
Parity			
0	0 (0%)	57 (57%)	<0.0001
1	29 (29%)	23 (23%)	
2	26 (26%)	12 (12%)	
3	23 (23%)	2 (2%)	
≥4	22 (22%)	6 (6%)	

In this study, 200 participants who met the inclusion criteria were successfully enrolled. Among which 100 were pregnant women (case group) and 100

non-pregnant women (control group). The demographic data obtained for the subjects showed that the most of the control group aged within 21 to

25 years (42%) while the majority of the pregnant women (42%) were within 26-30 years. There was a statistically significant difference in age and parity,

between the cases and the control groups ($p < 0.0001$).

Table 2: Comparison of platelet indices between non-pregnant and pregnant women

Platelet Indices	Non-pregnant Women	Pregnant Women	p-value
Platelet count ($\times 10^9$ cells/L)	216.94 \pm 53.27	224.56 \pm 68.72	0.282
Mean platelet volume (fL)	10.27 \pm 1.85	9.74 \pm 3.63	0.290
Platelet distribution width	12.60 \pm 2.07	13.47 \pm 3.60	0.712
Platelet larger cell ratio	28.62 \pm 7.63	28.32 \pm 8.32	0.310

Although, there was an increase in mean platelet count (PLT) in the pregnant women when compared with the non-pregnant group (224.56 \pm 68.72 $\times 10^9$ cell/L) versus 216.94 \pm 53.27 $\times 10^9$ cell/L), the MPV, PDW, and platelet larger cell ratio were slightly lower in pregnant women than the non-pregnant group. These changes were not statistically different between the two study groups ($p > 0.05$).

Table 3: Comparison of platelet indices between non-pregnant and the various trimesters of pregnant women

Platelet Indices	Non-pregnant state	First trimester	Second trimester	Third trimester	p-value
Platelet count ($\times 10^9$ cells/L)	216.94 \pm 53.27	226.64 \pm 44.26	236.34 \pm 82.38	210.44 \pm 62.08	0.180
Mean platelet volume (fL)	10.27 \pm 1.85	10.42 \pm 0.88	10.32 \pm 0.90	8.72 \pm 5.70	0.032
Platelet distribution width	12.60 \pm 2.07	13.28 \pm 2.18	13.40 \pm 3.27	13.47 \pm 4.45	0.974
Platelet larger cell ratio	28.62 \pm 7.63	28.22 \pm 7.70	27.93 \pm 6.84	28.00 \pm 10.12	0.730

The mean PLT count in the first, second and third trimester of pregnancy (226.64 \pm 44.26 $\times 10^9$ cell/L, 236.34 \pm 82.38 $\times 10^9$ cell/L and 210.44 \pm 62.08 $\times 10^9$ cell/L) did not differ significantly from the mean PLT count of the non-pregnant women (216.94 \pm

53.27 $\times 10^9$ cell/L) ($p = 0.180$). Moreover, the mean MPV of pregnant women were lowest in 3rd trimester and significantly differ from pregnant women in their 1st, 2nd trimester and the control group ($p = 0.032$).

Table 4: Incidence and severity of thrombocytopenia in non-pregnant and pregnant women

Variables	Pregnancy Status		p-value
	Pregnant, n (%)	Non-Pregnant, n (%)	
Platelet Count			
Normal (150-400 $\times 10^9$ cells/L)	90 (90%)	88 (89%)	0.812
Mild (100-<150 $\times 10^9$ cells/L)	10 (10%)	12 (12%)	
Moderate (50-<100 $\times 10^9$ cells/L)	0 (0%)	0 (0%)	
Severe (<50 $\times 10^9$ cells/L)	0 (0%)	0 (0%)	

Out of the 200 subjects in this study, 90 (90%) and 88 (88%) of the case and control groups had normal platelet counts (150-400 $\times 10^9$ cells/L), respectively. Moreover, 10 (10%) and 12 (12%) of the case and control groups had mild thrombocytopenia (100-150 $\times 10^9$ cells/L), respectively. There was no significant association between pregnancy status and thrombocytopenia ($p = 0.812$).

Discussion

Mild thrombocytopenia in pregnant women at the time of delivery, described as gestational thrombocytopenia, has been well documented in three large studies during that past 24 years. [14-16]

The reduced, but symmetrical, distribution of platelet counts at delivery suggests that a shift from normal values to lower platelet counts occurs in all women. [15-17] These observations have led to the common belief that a physiologic change associated with pregnancy causes platelet counts to gradually decrease. Platelets are responsible for blood clotting and play a significant role during pregnancy as well. The average or normal platelet count of a non-pregnant woman may range between 150,000 and 400,000 per microlitre of blood. The platelet count of a woman may drop to 116,000 per microlitre of blood during pregnancy. However, if it is less than 116,000, the condition is known as

thrombocytopenia. Thrombocytopenia, or a low blood platelet count, is encountered in 7-12% of all pregnancies. [18]

The demographic data obtained for the subjects showed that the most of the control group aged within 21 to 25 years (42%) while the majority of the pregnant women (42%) were within 26-30 years. There was a statistically significant difference in age and parity, between the cases and the control groups ($p < 0.0001$). Although, there was an increase in mean platelet count (PLT) in the pregnant women when compared with the non-pregnant group ($224.56 \pm 68.72 \times 10^9$ cell/L) versus $216.94 \pm 53.27 \times 10^9$ cell/L), the MPV, PDW, and platelet larger cell ratio were slightly lower in pregnant women than the non-pregnant group. These changes were not statistically different between the two study groups ($p > 0.05$). The mean PLT count in the first, second and third trimester of pregnancy ($226.64 \pm 44.26 \times 10^9$ cell/L, $236.34 \pm 82.38 \times 10^9$ cell/L and $210.44 \pm 62.08 \times 10^9$ cell/L) did not differ significantly from the mean PLT count of the non-pregnant women ($216.94 \pm 53.47 \times 10^9$ cell/L) ($p = 0.180$). In pregnancy, increased platelet destruction may be mediated by immunological mechanisms, abnormal platelet activation, or platelet consumption. Increased destruction or utilization of platelets during pregnancy occurs in microangiopathies (affecting small blood vessels) such as thrombotic thrombocytopenic purpura, haemolytic uraemic syndrome, haemolysis, elevated liver enzymes, low platelet (HELLP) syndrome, and pre-eclampsia. [19] Another study agrees with the physiologic findings in pregnancy where platelet counts decrease possibly due to haemodilution, which majorly occurs in the third trimester. [20] However, the difference in platelet count reported in our study compared to others could be attributed to the nature of sampling techniques or variation in laboratory analytical protocols, as these might have impacted on the results. [21]

Moreover, the mean MPV of pregnant women were lowest in 3rd trimester and significantly differ from pregnant women in their 1st, 2nd trimester and the control group ($p = 0.032$). There was no significant association between pregnancy status and thrombocytopenia. Findings from this study showed non-significant decreased levels of MPV, PCT and PDW in pregnant women compared to the control subjects ($p > 0.05$). These changes might be related to the blood volume expansion and hemodilution that occurs during pregnancy. During pregnancy, platelet count decreases gradually from the 1st till the 3rd trimester. In addition, hemodilution has been shown to accelerate platelet consumption which may contribute to a decline in platelet count throughout gestation. [22] However, there was a decrease in platelet count among subject in their 3rd trimester when compared with the control group. These are

similar to the findings by Babah et al [23] where they reported a statistically non-significant increase in platelet count in the first to the third trimester of pregnancy. There was a significant increase in mean platelet volume among subjects in the first and second trimester when compared with the control group. But a significant decrease was observed in the third trimester when compared with the control group. Mean platelet volume (MPV) and platelet distribution width (PDW) were reported to increase during platelet activation. [24]

In normal pregnancy, there is often an increase in platelet aggregation and a decrease in the number of circulating platelets with gestation. As the platelet lifespan decreases, the MPV increases minimally during pregnancy. [25] Thus, the MPV is an accurate measure of platelet size and its considered a biomarker of platelet function. Hence, larger platelets with higher MPV counts are reactive and raise higher amounts of the prothrombotic factor thromboxane A₂, increasing the tendency to thrombosis. [26] The PDW values appeared to have reduced in the three groups of pregnant women, which were statistically non-significant. On the contrary, an increase in PDW was reported by Omorogiuwa and Aigborhuan [27], which was attributed to physiologic compensation for the decreasing platelet count and volume with progression in gestation age.

Conclusion

Based on these findings, it can be inferred that platelet count and MPV decreases while PDW increase with the progression of gestation age compared to the non-pregnant women. Clinically platelet indices can be a useful screening test for early identification of preeclampsia and eclampsia. Platelet count is a simple, low cost, and rapid routine screening test.

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