

## Original Research Article

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# A comparison of platelet count in severe preeclampsia, mild preeclampsia and normal pregnancy

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## ABSTRACT

**Background:** Preeclampsia, the most common of hypertensive disorders of pregnancy is an idiopathic multisystem disorder affecting 2 – 10% of all pregnancies and together they form one member of the deadly triad, along with hemorrhage and infection that contribute greatly to the maternal morbidity and mortality rates. The identification of this clinical entity and effective management play a significant role in the outcome of pregnancy. Platelet count is emphasized to play a significant role in hemostasis mechanism of preeclampsia and the degree of thrombocytopenia increases with severity of preeclampsia. This study was conducted to find correlation of platelet count in severe preeclampsia, mild preeclampsia and normal subjects.

**Methods:** Total 140 subjects, 70 control and 70 cases were enrolled in the study. Samples for platelet count were collected and estimation was carried out by the auto-analyzers. The statistical evaluation is done using SPSS version 22 along with Anova and student t-test.

**Results:** The mean platelet count was significantly lower ( $p < 0.05$ ) in mild and severe preeclampsia than that in the normal pregnancy. Decreased platelet count in severe preeclampsia was significant compared to that in mild preeclampsia.

**Conclusions:** The frequency of thrombocytopenia was found to be directly related with the severity of disease, so platelet count can be used as a simple and cost effective tool to monitor the progression of preeclampsia, thereby preventing complications to develop during the gestational period.

**Keywords:** Preeclampsia, Platelet count, Thrombocytopenia

## INTRODUCTION

Pregnancy is a physiological process but can induce hypertension in normotensive women or aggravate already existing hypertension.

Preeclampsia, the most common of hypertensive disorders of pregnancy is an idiopathic multisystem disorder affecting 2-10% of all pregnancies and together they form one member of the deadly triad, along with haemorrhage and infection that contribute greatly to the maternal morbidity and mortality rates.<sup>1</sup> The

identification of this clinical entity and effective management play a significant role in the outcome of pregnancy. Normal pregnancy is associated with impressive changes in the haemostatic mechanism to maintain placental function during pregnancy and to prevent excessive bleeding in delivery. The combined changes of increase coagulation factors and suppression of fibrinolytic activity are defined as hypercoagulable state or prothrombotic state.<sup>2,3</sup> It usually occurs in the last trimester of pregnancy and more commonly in primiparas. It is characterized by maternal endothelial dysfunction presenting clinically with hypertension and proteinuria, and results in hypercoagulable state and may

lead to acute renal failure (ARF), pulmonary oedema and approximately 10% of women with severe preeclampsia may develop hemolysis, elevated liver enzyme and low platelet count referred to as HELLP syndrome.<sup>4</sup>

The endothelial dysfunction develops because of the formation of uteroplacental vasculature insufficient to supply adequate blood to the developing fetus resulting in fetoplacental hypoxia leading to imbalances in the release and metabolism of prostaglandins, endothelin and nitric oxide by placental and extra placental tissue. These as well as enhanced lipid peroxidation and other undefined factors contribute to the hypertension platelet activation and systemic endothelial dysfunction.<sup>5</sup>

Many haemostatic abnormalities have been reported in association with hypertensive disorder of pregnancy. Thrombocytopenia is most common of these abnormalities.<sup>6</sup> The degrees of thrombocytopenia increases with severity of disease.<sup>7</sup> Thrombocytopenia in preeclampsia is attributed to various causes including increased platelet consumption due to disseminate intravascular coagulopathy and/or immune mechanism.<sup>8</sup>

Most of the studies observed significant decrease in platelet count during normal pregnancy.<sup>9</sup> There is a significant decrease in platelet count especially during second and third trimesters. Thrombocytopenia can result from decrease in platelet production or accelerated platelet destruction. The various mechanisms of thrombocytopenia in pregnancy explained by different workers are as under:

- Hemodilution in late pregnancy.<sup>10</sup>
- Decreased platelet survival time during normal pregnancy.<sup>11</sup>
- Plasma beta thromboglobulin and platelet factor 4 levels, both reflecting platelet activation, were significantly increased during normal pregnancy, indicating an increase in platelet activation, and supporting the hypothesis that there is an increased turnover of platelets during the progression of normal pregnancy.<sup>12</sup>

Hence the study is aimed to analyse the utility of platelet count in pre-eclampsia so as to prevent complication, early detection, careful monitoring and appropriate management to reduce the morbidity and mortality of both mother and child.

## METHODS

This comparative prospective study was conducted at the J.K Hospital associated with L.N Medical College and research centre, Bhopal during the period of one year. Cases of preeclampsia will be categorized on the basis of blood pressure based upon classification according to the scheme the National High Blood Pressure Education Program (NHBPEP) (2000) criteria<sup>13</sup>

- Mild preeclampsia: Patient having systolic blood pressure between 140-160 mmHg, diastolic blood pressure between 90-110 mmHg and proteinuria upto 1+
- (Severe preeclampsia: Patient having systolic blood pressure between >160 mmHg, diastolic blood pressure >110 mmHg plus one or more of the following criteria: proteinuria >1+, headache, visual disturbance, upper abdominal pain, oliguria (<400 ml/24 hours), serum creatinine elevated >1.2 mg/dl, marked elevation of serum transaminase AST or ALT, fetal growth restriction and pulmonary edema.

The study includes total 140 subjects, 70 normotensive pregnant women without any complication and 70 pregnant women with signs and symptoms of preeclampsia in third trimester of gestation.

All the subjects were undergone blood investigations, i.e. complete blood cell count for Platelet count using EDTA anticoagulant blood and analyzed on Mindray, Automated Hematology Analyzer". The test was conducted within 1 hour of sample collection maintaining at room temperature to minimize variation due to sample aging. All consenting Gestation age and gestation matched normal pregnant women in 3rd trimester would constitute the control.

Exclusion Criteria include subjects having history of essential hypertension, with known liver disease, renal disorder, hydatidiform mole, with known bleeding disorder, on anticoagulant therapy, with established DIC, idiopathic thrombocytopenic purpura, history of illicit drug use, any associated inflammatory disease or sepsis, any associated malignancy. Thrombocytopenia was defined as platelet count <150x10<sup>9</sup>/L.

## Statistical analysis

The statistical software namely statistical package for the social sciences (SPSS) version 22 is used for analysis of data. Analysis of variance (ANOVA) is used to compare the variables. Data is expressed as mean±standard deviation. The p-value was calculated for each parameter and p<0.05 is considered statistically significant. Student t-test was used for doing comparison of Platelet Count in Severe Preeclampsia, Mild Preeclampsia and Normal Pregnancy. Bar diagram were used for graphical representation of this data.

## RESULTS

In the present study, we have studied platelet count in total 70 cases of preeclampsia in 3rd trimester of pregnancy. It included 31 (44.30%) cases of mild pre-eclampsia, 39 (55.70%) cases of severe pre-eclampsia. (Table 1). Similarly, 70 normotensive age and gestation matched pregnant women in 3<sup>rd</sup> trimester were also studied as controls.

**Table 1: Age wise distribution of patients in cases and controls.**

Age group (Years)	Control (%)	Cases		
		Mild PE (%)	Severe PE (%)	Total cases (%)
20-24	33 (47.5)	8	21	29 (41.42)
25-29	28 (41.25)	15	14	29 (41.42)
30-34	6 (7.5)	7	3	10 (14.28)
35-40	3 (3.75)	1	1	12 (2.85)
Total	70 (100)	31 (44.30)	39 (55.70)	70 (100)
Mean age± SD	25.17± 3.85	26.19± 3.72	24.28± 3.41	25.12± 3.675
Range	20-35	20-35	20-35	20-35

In the present study, mean age of the cases was  $25.12 \pm 3.65$  years. Maximum 68 (82.82%) cases were between 20-29 years of age. There was only one case of mild preeclampsia and severe preeclampsia each above 35 years of age as shown in Table 1.

The mean gestational age in cases was  $33.55 \pm 3.93$  weeks. In subgroups of cases, the mean gestational age was  $33.94 \pm 3.54$  and  $33.30 \pm 4.43$  weeks in mild pre eclampsia and severe pre eclampsia respectively as shown in Table 2. Statistically these differences were found to be insignificant ( $p = 0.197$ ;  $p > 0.05$ ).

**Table 2: Distribution of cases and controls in relation to gestational age.**

Diagnosis	No. of patients	Gestational age (weeks)	
		Range	Mean±SD
Cases	70	28-40	$33.55 \pm 3.93$
Mild PE	31	28-40	$33.94 \pm 3.54$
Severe PE	39	28-40	$33.30 \pm 4.23$
Controls	70	28-40	$34.66 \pm 3.70$

The distribution of cases according to parity showed that 41 (58.60%) cases were primiparous and 29 (41.40 %) cases were multiparous whereas in controls 40 (57.20%) women were primiparous and 30 (42.80%) women were multiparous as shown in Table 3.

**Table 3: Distribution of parity in cases and controls.**

Diagnosis	No. of patients (%)	Parity	
		Primi (%)	Multi (%)
Cases	70 (100)	41 (58.60)	29 (41.40)
Mild PE	31 (100)	16 (51.60)	15 (48.40)
Severe PE	39 (100)	25 (60.10)	14 (39.90)
Controls	70 (100)	40 (57.20)	30 (42.80)

The difference in parity in cases and controls was found statistically insignificant ( $p=0.572$ ,  $p>0.05$ ). When compared among the subgroups of cases, in severe pre eclampsia out of 39 cases 25 were primiparous and 14 were multiparous. Thus, severe pre eclampsia was found to be more common in primiparous women as compared

to that in mild pre eclampsia and statistically this difference was found to be significant ( $p<0.05$ ) (Table 3).

**Table 4: Mean platelet count in cases and controls.**

Diagnosis	No. of patients	Platelet count ( $\times 10^9/L$ )	
		Range	Mean±SD
Cases	70	24-366	$168 \pm 74.22$
Mild PE	31	62-366	$197.29 \pm 73.65$
Severe PE	39	24-253	$145.25 \pm 66.96$
Controls	70	76-450	$229.61 \pm 73.27$

In the present study, the mean platelet count in cases was found to be  $168 \pm 74.22 \times 10^9/L$  with a range of 24-366  $\times 10^9/L$  while in controls the mean platelet count was  $229.61 \pm 73.27 \times 10^9/L$  with a range of 76 - 450  $\times 10^9/L$  as shown in Table 4. Thus, there was decrease in mean platelet count in cases as compared to that in controls and this difference was statistically significant ( $p=0.000$ ,  $<0.05$ ). The mean platelet count in various subgroups was  $197.29 \pm 73.65 \times 10^9/L$  in mild preeclampsia and  $145.25 \pm 66.96 \times 10^9/L$  in severe preeclampsia (Table 4, Figure 4). Thus, the platelet count was found to decrease with the progression of disease. On statistical analysis, when the mean platelet count in different subgroups of cases were compared with that in the controls, the decrease in platelet count in mild preeclampsia ( $p=0.043$ ,  $<0.05$ ) and in sever preeclampsia ( $p<0.05$ ) was significant.

**Table 5: Distribution of cases and controls according to the level of platelet counts.**

Diagnosis	Platelet count ( $\times 10^9/L$ )			
	>150	100-150	<100	Total
Cases no. (%)	40 (57.14)	17 (24.28)	13 (18.57)	70 (100)
Mild PE	22	7	2	31
Severe PE	18	10	11	39
Controls no. (%)	61 (87.14)	7 (10.06)	2 (2.8)	70 (100)

The comparison of platelet count amongst the subgroups of cases showed the decrease in platelet count in severe pre eclampsia was significant ( $p=0.002$ ,  $<0.05$ ) when compared with that in mild preeclampsia. In the present study the cases and controls were distributed according to the levels of platelet count into three categories as normal ( $>150 \times 10^9/L$ ), low ( $100-150 \times 10^9/L$ ), and very low ( $<100 \times 10^9/L$ ) platelet counts. 40 (57.14%) cases were found to have normal platelet counts while 17(24.28%) cases had low platelet counts and 13(18.57%) cases had very low platelet counts. Similarly, in controls 61 (87.14%) women were found to have normal platelet counts, 7 (10.06%) women had low platelet counts and only 2 (2.8%) women had very low platelet counts, as shown in Table 5. In total 70 cases of pre eclampsia, thrombocytopenia was seen in 30(42.85%) cases. There were only 9 (12.85%) case with thrombocytopenia in mild pre eclampsia group whereas in sever pre eclampsia

21 (30%) cases showed thrombocytopenia. In the controls, only 9 (12.85%) women showed thrombocytopenia.

## DISCUSSION

In the present study, mean age of the cases was  $25.12 \pm 3.65$  years. Maximum 68 (82.82%) cases were between 20-29 years of age (Table 2). It appears that as far as age is concerned, there is no or little difference between normal healthy pregnant women and patients with different degrees of severity of pregnancy induced hypertension. But it was clear that most patients in normal pregnant control group and patients with pregnancy induced hypertension were in age ranging between 21 to 29 years. Jaleel et al and Kumar et al also found maximum cases between 21-30 years of age, similar to the present findings.<sup>14,15</sup> Younger age of occurrence of preeclampsia testifies the early age of marriage and pregnancy in our country as compared to western countries.

The mean gestational age observed in cases in the present study was  $33.55 \pm 3.93$  weeks. In subgroups of cases the mean gestational age in mild preeclampsia and severe preeclampsia were found to be  $33.94 \pm 3.54$  and  $33.22 \pm 4.23$  weeks respectively. Statistically, these differences were found to be insignificant (Table 3). Priyadarshini et al, Jahromi et al also observed similar findings with that of present study, their findings were also statistically not significant.<sup>16,17</sup>

The present study observed, 41 (58.60%) cases and 40 (57.20%) controls were primiparous and there was no significant difference in the parity of cases and controls (Table 3). However, in subgroups of cases the present study observed, the severe pre-eclampsia (64.10% cases) were more frequent in primiparous women as compared to that in mild preeclampsia (51.60% cases). Chaware et al observed that in mild pre-eclampsia 52% cases were primigravidae, 28% patients were second gravidae and 20% cases were third or more gravidae.<sup>18</sup> Similarly Sameer et al observed patients of mild pre eclampsia and severe pre-eclampsia were all more frequent in primiparous women accounting for 66.03% and 65.51% cases respectively.<sup>19</sup> In present study severe pre-eclampsia 67.5% patients were primigravidae, followed by 22.5% cases of second gravidae and 10% of third or more gravidae. The present findings correlate with authors in respect of severe preeclampsia.

In the present study, the mean platelet count in cases was found to be  $168 \pm 74.29 \times 10^9/L$  while in controls it was  $229.61 \pm 73.27 \times 10^9/L$  (Table 4). This decrease in platelet count in cases was statistically significant as compared to that in controls. Chauhan et al observed platelet count in cases as  $157.18 \pm 56.66 \times 10^9/L$  and in controls as  $222.93 \pm 97.94 \times 10^9/L$ .<sup>20</sup> Meshram DP observed platelet count  $242 \pm 62 \times 10^9/L$  in control group and  $160 \pm 51 \times 10^9/L$  in preeclampsia.<sup>21</sup> These authors also found significantly

decreased platelet counts in pre eclampsia as compared to that in controls, similar to the present findings. In present study, subgroups of cases, the platelet count was  $197.29 \pm 73.65 \times 10^9/L$  in mild preeclampsia, and  $145.23 \pm 66.91 \times 10^9/L$  in sever preeclampsia (Table 4). The present study also found thrombocytopenia in 30 (42.85%) cases with only 9 (12.85%) cases having thrombocytopenia in mild preeclampsia patients and 21 (30%) cases in severe preeclampsia (Table 5). Vrunda et al found thrombocytopenia in 10 (25%) cases of mild PIH and 20 (62.5%) cases of severe PIH.<sup>22</sup> Mohapatra et al found thrombocytopenia in 2 (6.6%) cases of mild PIH and 18 (60%) cases of preeclampsia.<sup>23</sup> Thus, the platelet count was found to decrease with the severity of disease. This gradually reduced platelet counts in patients of mild pre eclampsia to severe pre eclampsia were comparable to those reported by other authors as shown in Table 6. On comparing the subgroups of cases, the number of thrombocytopenia cases was more in patients with severe pre-eclampsia followed by that in mild pre eclampsia. Thus, the frequency of thrombocytopenia cases was also found to be directly related with the severity of disease.

**Table 6: The findings of various authors in cases and controls in respect to the normal, low and very low platelet counts.**

Author (Years)	Platelet counts ( $\times 10^9/L$ )		
	Control	Mild PE	Severe PE
Srivastava et al <sup>24</sup>	194.4	179.7	164.2
Jambulkar et al <sup>25</sup>	238	230	170
Ammar K et al <sup>26</sup>	252.6	183.85	134.88
Present study	$229.61 \pm 73.27$	$197.29 \pm 73.65$	$145.23 \pm 66.91$

The mechanism of thrombocytopenia in pre eclampsia is variously explained as under:

It may be due to increased consumption of platelets with increased megakaryocytic activity to compensate it. Platelets adhere to areas of damaged vascular endothelium resulting in secondary destruction of platelets (O'Brien et al).<sup>27</sup>

- Prostacyclin is an important eicosanoid that exerts strong inhibition of platelet aggregation. There is continuous availability of this eicosanoid from blood vessels which keeps circulating platelets in a dispersed and disaggregated form (O'Brien et al).<sup>27</sup> Deprivation of this prostacyclin makes the circulating platelets even more vulnerable to aggregation. Removal of aggregated platelets might be responsible for thrombocytopenia often observed in pregnancy induced hypertension (FitzGerald et al).<sup>28</sup>
- Platelets from severely preeclamptic patients showed less response than normal to a variety of aggregating agents suggesting that platelets may have undergone previous aggregation in the microcirculation (Whigham et al 1978).<sup>29</sup>

- Recent studies have documented that increased plasma levels of sFlt1- soluble vascular endothelial cell growth factor (VEGF) receptor type 1 as well as endoglin, an endothelial cell-derived member of the tumor growth factor-2 (TGF-2) receptor family (Venkatesha et al), are present in patients intended to develop preeclampsia as early as the late first trimester.<sup>30</sup> Increased levels of soluble fms-like tyrosine kinase-1 (sFlt1) and endoglin mRNA is present in preeclamptic placentae, suggesting this is the source of these proteins (Kita et al).<sup>31</sup> sFlt1 binds and neutralizes VEGF and placental growth factor (PLGF), another important VEGF and placental growth factor (PLGF), another important VEGF family member whose levels normally increase during pregnancy, whereas endoglin blocks the binding of TGF-2 to endothelial cells. (Young et al).<sup>32</sup> These types of pregnancies are also associated with qualitative alterations suggesting increased platelet turnover.

## CONCLUSION

The frequency of thrombocytopenia was found to be directly related with the severity of disease, so platelet count can be used as a simple and cost effective tool to monitor the progression of preeclampsia, thereby preventing complications to develop during the gestational period.

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