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Original Research Article

## Platelet count and MPV in women with PIH in their third trimester

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### ABSTRACT

**Background:** One of the most common and potential life-threatening complications of pregnancy is pregnancy induced hypertension. Though platelet count during pregnancy is within the normal non-pregnant reference values, there is a tendency for the platelet count to fall in late pregnancy. The frequency and intensity of maternal thrombocytopenia varies and is dependent on the intensity and severity of PIH.

**Methods:** This prospective study was conducted in the Department of obstetrics and gynecology in Jhalawar medical college from January 2018 to April 2018. Total 120 pregnant women during third trimester (32-40 weeks) aged 18 to 35 years were selected. Among them 63 were preeclamptic patients and 63 were healthy normotensive control. Subjects and healthy pregnant women (control) visiting the Obstetrics and Gynecology department of Jhalawar Medical College were registered in the study and followed during their pregnancy. Both, subjects and control participants were subject to platelet count manually and MPV was determined by an automated analyser (sysmex XN-1000™) performed using standard methods on.

**Results:** The mean platelet count of the subject group ( $131.4937 \pm 62.05999$  lakh/mm<sup>3</sup>) was significantly lower than that of the control group ( $324.9683 \pm 230.78764$  lakh/mm<sup>3</sup>). This shows that there is thrombocytopenia found in patients with P.I.H in their third trimester. On the other hand, the p value of “mean platelet volume” in patients with preeclampsia was ( $p < 0.0001$ ) which shows that there is no significant difference in MPV of cases ( $7.1438 \pm 2.62$  femtolitre) and control ( $7.8976 \pm 3.08$  femtolitre) ( $p > 0.142$ ), regular monitoring of platelet counts in women with Pregnancy Induced Hypertension must be subject of the management protocols.

**Conclusions:** In present study we observed that the number of thrombocytopenic subjects was higher in cases of preeclampsia as compared to the control group. These extrapolations indicate that there might be some important mechanism which interferes with platelets life span thus reducing the number of functional platelets in circulation. The platelet count has an association at prediction of increasing grade of PIH. There is an inverse relationship between the severity of PIH and platelet count. The depleted platelet counts are concluded to be consistently associated with clinical groups of severe preeclampsia and the risk of consumptive coagulopathy.

**Keywords:** fL (femtolitre), Hypertension, MPV (Mean platelet volume), Platelet count, Pregnancy

### 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 woman with severe preeclampsia may developed, 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 releases 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 increases 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. The aim of the study was to see the platelet count in pregnancy induced hypertension

(PIH). It was a prospective study. This study is aimed to investigate the relationship between platelet count and MPV (Mean platelet volume) in pregnancy induced hypertension patients in their 3rd trimester. To study Platelet count and mean platelet volume in cases of Pregnancy induced hypertension and to correlate and compare the values with Normotensive pregnant subjects. All the necessary information and data were collected by interviewing the patients or their attendants on a pre-designed data collection sheet. Blood sample were collected from the patients and required investigations were done. Although there is chance of development of disseminated intravascular coagulation (DIC) and Liver Failure in patients with low platelet count, but in present study there is no such incidence.

## METHODS

This present study was carried out at the Obstetrics and Gynaecology department Jhalawar medical college Jhalawar district, Rajasthan 2018 (September). The preliminary data in regard to name, age, sex, registration number, obstetric, menstrual, and family history, general and systemic examination and investigations were recorded in a proforma after getting informed consent from the patients. The subjects for this study included consecutively-recruited Sixty-Three (63) pregnant women with hypertension (PIH) and proteinuria. Sixty-Three (63) subjects without PIH served as a control. Two millilitres of blood sample was drawn aseptically using the 5ml syringe from the median ante-cubital vein of all the subjects and control participants into EDTA-ant coagulated tubes. Platelets were counted manually under high field microscope by sending samples to the pathology department at Jhalawar medical college.

### Inclusion criteria

- Age 18-35 years,
- Preeclamptic women whose blood pressure was normal during first 20 weeks of gestation
- No previous history of hypertension and all the case were in the third trimester of pregnancy.
- BP>140 /90 with proteinuria (proteinuria +1, +2, +3 or +4) on dipstick test.

### Exclusion criteria

- Subjects with haemorrhagic disorders,
- Sepsis,
- Functional uterine bleeding,
- Placental abruption or previa,
- Diabetes, respiratory, circulatory, renal and hepatic disorders,
- Known cases of hypertension
- Gestational thrombocytopenia
- Immune thrombocytopenia
- Subjects taking drugs which can affect platelet count were excluded from the study.

### Statistical analysis

Statistical analysis was performed with the SPSS, Trial version 23 for Windows statistical software package (SPSS inc., Chicago, il, USA) and Primer. The Categorical data were presented as numbers (percent) and were compared among groups using Chi square test. The quantitative data were presented as mean and standard deviation and were compared using by students t-test and ANOVA Test .Probability P value 25 years subjects dominated in severe preeclampsia group.

### RESULTS

In present study total no of 126 patients were taken, divided into two groups. Each group containing 63 patients. The groups were divided into cases (63 patients) and cases (63 patients).

The age group which we took for present study was between 18 to 35 years of age. The mean age for cases

was 27.50 and the mean age for control group was 28.36 with a standard deviation of 3.1 and 2.7 respectively. The results showed that there was no significant difference in age between cases and control ( $p>0.05$ ). It means study is age matched (Table 1).

**Table 1: Distribution of age according to groups.**

	Group	N	Mean age	Std. deviation	T value	P value
Age	Case	63	27.50	3.18	1.619	0.108
	Control	63	28.36	2.74		

The mean platelet count of the subject group ( $131.4937\pm 62.05999$ ) was significantly higher when compared to that of the control group ( $324.9683\pm 230.78764$ ). The P value is  $<0.0001$  which is  $<0.05$ . This shows that there is significant co-relation between PIH and platelet count which is with increase in B.P of a patient, platelet count decreases (Inverse relationship) (Table 2).

**Table 2: Distribution of platelets according to groups.**

	Group	N	Mean platelet (lakh/mm <sup>3</sup> )	Std. deviation	T value	P value
Platelets	Case	63	131.4937	62.05999	6.426	$<0.0001$
	Control	63	324.9683	230.78764		

**Table 3: Distribution of MPV according to groups.**

	Group	N	Mean platelet volume (femtolitre)	Std. Deviation	T value	P value
MPV	Case	63	7.1438	2.62068	1.479	0.142
	Control	63	7.8976	3.08140		

When compared mean platelet volume of cases and control group. The mean platelet volume of cases and control group was found to be ( $7.1438\pm 2.62068$ ) and ( $7.8976\pm 3.08140$ ) respectively.

The p value was found to be  $p>0.142$ , which is  $> 0.05$  and thus no significant relationship was found in MPV of cases and controls. Although various previous studies have shown inverse relationship of MPV between cases and controls (Table 3). Regular monitoring of platelet counts in women with Pregnancy Induced Hypertension must be subject of the management protocols.

There is definite statistical difference in values of platelet count in PIH groups when compared with normotensive pregnant women. As PIH is known to land in consumptive coagulopathy, the present study concludes and suggests that the estimation of platelet count offer early, simple, rapid assessments of the disease for its severity and the risk of complications. Therefore, these tests may be considered as screening tests to be routinely performed in antenatal workup of women with PIH.

### DISCUSSION

Hypertensive disorders complicating pregnancy form one of the deadly triads of maternal death along with sepsis and haemorrhage. Pregnancy induced hypertension has been divided into three categories:

- Hypertension alone
- Pre-eclampsia
- Eclampsia.

Pre-eclampsia is defined as hypertension (BP $>140/90$ ) plus proteinuria (300mg or more of urinary protein for 24hrs or 100mg/dl or more in at least two random urine specimens collected 6 or more hours apart).

Importantly the differentiation between mild and severe pre-eclampsia cannot be rigidly pursued because apparently mild disease may progress rapidly to severe disease and one of the signs of severity is thrombocytopenia associated with pre-eclampsia. Maternal thrombocytopenia can be induced acutely by

pre-eclampsia or may follow the progressive course. The frequency and intensity of maternal thrombocytopenia have shown different results in different studies. The cause of thrombocytopenia is not known; platelet aggregation is increased in pre eclamptic women (Torres and associates). Immunological processes or simply platelet deposition at sites of endothelial damage may be the cause (Pritchard and colleagues). Samuel and colleagues performed direct and indirect anti globulin test and found that platelet bound and circulating platelet bindable immunoglobulin were increased in pre-eclampsia women and their neonates. They interpreted these findings to suggest platelet surface alterations. Burrows and colleagues reported that platelets from pre eclamptic women were more likely to have platelet associated IgG, even if thrombocytopenia didn't develop. Although, they believed this mechanism implied an autoimmune processing could also be bound to platelets damaged by any mechanism.

Kelton showed that thrombocytopenia with preeclampsia was frequently associated with a prolonged bleeding time. This was true even with normal platelet level. They attributed this to impaired thromboxane synthesis. Kilby and associates and Barr and colleagues found increased intracellular free calcium concentration in platelets from preeclamptic women. Louden and colleagues interpreted this and other evidence to mean that platelets from preeclamptic women are exhausted, that is, platelet aggregation and release are decreased.

The clinical significance of thrombocytopenia, in addition to the obvious impairment in coagulation, is that it reflects the severity of the pathological process. In general, the lower the platelet count, the greater are maternal and fetal morbidity and mortality (Leduc and coworkers, Verhaeghe and colleagues). The addition of elevated liver enzymes to this clinical picture is even more ominous. Weinstein referred to this combination of events as the HELLP syndrome that is, haemolysis (H), Elevated liver enzymes (EL) and low platelets (LP). This study presents data on the platelet count investigated for twenty pregnant women with pregnancy induced hypertension. Significant lower platelet count was observed among pregnant women with PIH compared to individuals from control group.

Preeclampsia is one of the major health problems during pregnancy. It complicates 3%-8% of pregnancies and causes marked increase in perinatal, maternal morbidity, and mortality.<sup>13-16</sup> Although the exact pathophysiology of preeclampsia is not completely understood, certain factors have been attributed to it, which include deficient trophoblastic invasion of the maternal vascular bed with subsequent reduction of placental blood flow.<sup>17,18</sup> Placental under perfusion initiates widespread systemic, maternal endothelial dysfunction, and increased vascular permeability.<sup>19</sup> Coagulation system is activated by the contact of platelets with the injured endothelium leading to increase in consumption as well as bone marrow

production of platelets.<sup>20</sup> Various indices are used to measure platelet functions, for example, the platelet count (PC), mean platelet volume (MPV), the PC to MPV ratio, and platelet distribution width (PDW); PDW measures platelet size distribution.<sup>21</sup> The utility of different platelets indices as predictors of preeclampsia has been studied previously however, reports in this regard are controversial.<sup>22-33</sup> "Occurrence of hypertension (systolic blood pressure  $\geq 140$  mm hg or diastolic blood pressure  $\geq 90$  mm hg) in the second half of pregnancy (after 20 weeks of gestation) and proteinuria (presence of 300 mg or more of protein in 24 hour urine sample or  $\geq 2+$  on dipstick)".<sup>34</sup> Preeclampsia cases were considered mild or severe according to the diastolic blood pressure of  $< 110$  or  $\geq 110$  mm hg, respectively.<sup>35</sup>

There was no significant difference in PDW in the current study. This goes with the previous findings, where Doğan et al found no significant difference in PDW among women with severe preeclampsia, mild preeclampsia, and healthy controls.<sup>26</sup> Nonetheless, a significantly higher level of PDW has recently been observed among women with preeclampsia.<sup>24,29,36</sup>

Although, the current study showed no difference in the MPV between the cases and the controls, the utility of MPV in predicting preeclampsia has been shown in several studies.<sup>24,26-28,30-32</sup> It is worth to mention that Yavuzcan et al observed no significant difference in the MPV between women with severe preeclampsia, healthy pregnant women, and healthy non-pregnant women.<sup>33</sup> It is noteworthy that some researchers failed to confirm PC and MPV as predictors of preeclampsia, probably because of the differences in the methods and/or equipment used to obtain hemogram.<sup>37,38</sup>

Aside from platelet parameters, there are several biomarkers of preeclampsia including soluble endoglin (seng) or soluble fms-like tyrosine kinase-1 (sflt-1).<sup>39</sup> One limitation of this study is the lack of a comparative analysis between platelet parameters and seng/sflt-1. However, platelet parameters are simple laboratory markers and easy to check during antenatal care.

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