

## **Study of Mean Platelet Volume in Gestational Hypertension and Normal Pregnancy**

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### **Abstract**

**Introduction:** Gestational hypertension (GH) previously known as pregnancy induced hypertension (PIH) is seen in 10 to 12 % of Indian women. Platelets play a major role in the disease process of Gestational Hypertension. Mean platelet volume is one of the markers of platelet activation and can be easily measured routinely as a part of complete blood count. Studies relating mean platelet volume (MPV) and Gestational Hypertension are few and reports are contradictory. This study was done to know variations in MPV from first trimester to third trimester during pregnancy in healthy pregnant women and in Gestational Hypertension.

**Methods:** In our study, 50 cases of gestational hypertension and 50 women with normal pregnancy were included. MPV was measured during first trimester and third trimester in both women with gestational hypertension and normal pregnancy. All pregnant women were followed up to know the maternal and neonatal outcome.

**Results:** MPV was significantly increased from first trimester to third trimester in women with gestational hypertension than in normal pregnant women.

**Conclusion:** MPV is one of the markers of platelet activation that can be increased even before the onset of clinical symptoms. MPV is done routinely in all pregnant women. Even though MPV was within normal limits, there is an increase in MPV from first trimester to third trimester in gestational hypertension. So any increase in MPV may be considered as a caution and patients can be monitored more carefully to avoid adverse outcome. Further studies are needed to explore the usefulness of MPV as a marker of gestational hypertension.

**Keywords:** Gestational hypertension, pregnancy induced hypertension, mean platelet volume

### **1.Introduction**

Hypertensive disorders represent the most common medical complications of pregnancy and a major cause of maternal and perinatal mortality and morbidity worldwide[1]. The 4 common forms of hypertension are gestational hypertension, pre-eclampsia, eclampsia and chronic essential hypertension.

Gestational hypertension (GH) previously known as Pregnancy Induced Hypertension (PIH) is diagnosed if blood pressure is >140/90mmHg without proteinuria after 20 weeks of pregnancy[2,3].

The etiology and pathogenesis of GH remain enigmatic and controversial. The accepted theory is endothelial dysfunction and platelet

activation. Platelet activation results in release of granule contents and coagulation pathway gets activated which leads to various clinical and biochemical manifestations of the disease[4].

Mean platelet volume (MPV) is one of the indicators of the platelet activation and can be easily measured by automated analysers[5]. It is usually routinely done in first and third trimesters during antenatal check-up as a part of complete blood count.

This study was done to find if there is any significance in variation of mean platelet volume from 1<sup>st</sup> trimester to 3<sup>rd</sup> trimester in Gestational Hypertension and normal pregnancy.

### 1.1 Objectives of the study

To compare 1<sup>st</sup> and 3<sup>rd</sup> trimester variations of mean platelet volume in Gestational Hypertension and normal pregnancy

## 2. Materials and Methods

It is a comparative and case control study. Institutional ethical committee approval was obtained. Informed consent was taken from both cases and controls. Cases and controls were from patients who came to Obstetrics and Gynecology outpatient department of Vydehi Institute of Medical Sciences and Research Centre for routine antenatal check-up.

Cases included in the study were 50 women with systolic blood pressure of  $\geq 140$ mmHg and/or diastolic blood pressure of  $\geq 90$ mmHg after 20 weeks of pregnancy and without proteinuria (or urine protein of  $<300$ mg/dl for 24 hours)[6]. Women with essential hypertension, diabetes mellitus, kidney diseases, cardiac diseases, twin pregnancy and fetal anomalies were excluded from the study.

Controls were 50 normal pregnant women after 20 weeks included in the study. Institutional ethical committee approval was obtained.

All the participants underwent complete general physical examination, systemic examination and routine obstetric examination.

Vital parameters like pulse rate, blood pressure, temperature were checked. If blood pressure was found to be  $\geq 140/90$ , the subject was made to lie down for 30 minutes and rechecked again to confirm the diagnosis of gestational hypertension.

MPV was measured during first trimester and third trimester using COULTER A<sup>C</sup>T5 diff Autoloader (AL) hematology analyser. Basic investigations including complete blood count, RBS, VDRL, HIV/HBsAg, urine routine and urine for protein were done for both the groups. MPV was collected retrospectively from cases after diagnosis was made.

The normal values are[7]:

**Table 1: Mean platelet volume (MPV) (whole blood)**

Units	Non-pregnant Adult	First Trimester	Second Trimester	Third Trimester
$\mu\text{m}^3$	6.4 – 11	7.7 - 10.3	7.8 - 10.2	8.2 - 10.4
fl	6.4 – 11	7.7 - 10.3	7.8 - 10.2	8.2 - 10.4

### 2.1 Statistical analysis

Descriptive and inferential statistical analysis has been carried out in the present study. Student t test (two tailed, independent) has been used to find the significance of study parameters on continuous scale between two groups (Inter group analysis).

## 3. Results

Maximum numbers of subjects were in the age group of 21-25yrs in both groups. Mean age for cases was  $24.28 \pm 4.19$  and for controls it was  $24.19 \pm 3.45$ .

**Table 2: Blood pressure (mmHg)**

	Mean age(years)	Mean systolic blood pressure (mmHg)	Mean diastolic blood pressure (mmHg)
Controls	$24.19 \pm 3.45$	$112.92 \pm 7.20$	$74.44 \pm 6.04$
Cases	$24.28 \pm 4.19$	$143.44 \pm 11.82$	$95.28 \pm 8.54$

**Table 3: Mean Platelet Volume (MPV)**

Mean Platelet Volume(MPV)	Cases	Controls	P value
1st trimester	$7.75 \pm 0.78$	$8.06 \pm 1.00$	0.097+
3 <sup>rd</sup> trimester	$8.83 \pm 1.34$	$8.20 \pm 1.17$	0.015*
Difference	1.07	0.14	$<0.001^{**}$
P value	$<0.001^{**}$	0.460	-

MPV increased from first trimester to third trimester significantly by 1.07fl in cases where as there was no significant change in controls.

## 4. Discussion

Hypertensive disorders are the most common medical problems of pregnancy that complicate 10% of all pregnancies and can result in severe complications for the mother such as eclampsia, placental abruption, preterm delivery, the syndrome of Hemolysis, Elevated Liver enzymes and Low Platelets (HELLP) and ultimately even neonatal and maternal death.[1,8] The hypertensive disorders are classified as gestational hypertension (PIH), pre-eclampsia, eclampsia and chronic essential hypertension according to National High Blood Pressure Education Program Working Group in 2000.[6]

Gestational Hypertension (GH) is the most frequent cause of hypertension during pregnancy with incidence being 6-7% in nulliparous and 2-4% in multiparous women. Majority of cases of GH develop after 37wks of gestation.[1]

The accepted theory for pathogenesis is endothelial dysfunction and platelet activation. In response to endothelial injury, platelets get activated and release contents of alpha granules like thromboxane A<sub>2</sub> and serotonin leading to vasoconstriction and activation of coagulation. This secondary activation of coagulation combined with a decrease in fibrinolysis and impaired fibrin clearance will interfere with the microcirculation of placenta, liver, CNS and kidneys. This leads to development of clinical and biochemical features of the disease.[4]

Platelet activation is measured in terms of reduced platelet count, increased mean platelet volume, elevated  $\beta$ -thromboglobulin and platelet factor 4.[5] These are elevated in PIH much before the onset of clinical symptoms and hence may be useful as predictors for the onset of the disease and also indicate the severity.[4,9,10]

Mean platelet volume (MPV) is one of the indicators of the platelet activation and can be easily measured by automated analysers. It is routinely done during antenatal checkup.[5]

Few studies have been done regarding the feasibility of using MPV as markers in PIH and have shown contradictory results. They have shown that there is macrothrombocytosis[11] and increase in mean platelet volume in patients with moderate or severe hypertension in pregnancy.[12]Whereas another study showed that there was no change in the mean platelet volume in patients with mild to moderate hypertension.[13]

In a study done by Ioannis *et al*[14]found that there was no change in mean platelet volume in normotensive as well as PIH group during 3<sup>rd</sup> trimester . But another study done by Singer *et al*[12] showed that there is an increase in mean platelet volume in patients with moderate or severe hypertension in pregnancy. Giles and Inglis[11] observed that there was significant macrothrombocytosis (MPV>10.4) in hypertensive pregnant women with/without edema and without proteinuria. A study done by Yin[15] also showed that mean platelet volume in PIH was higher than that of controls.

In this study, MPV was measured during 1<sup>st</sup> trimester and 3<sup>rd</sup> trimester in gestational hypertension and normal pregnant women.

In the present study even though mean platelet volume was within normal limits, there was a significant increase in mean platelet volume from first trimester to third trimester in cases (1.07) compared to controls (0.14). But our study reported that MPV was increased more than the normal value (>10.4) in 6 cases of which 4 of them had systolic blood pressure >150mmHg and 2 of them had diastolic blood pressure >90mmHg. This indicates the association of MPV with severe GH cases. A study done by Dadhich *et al*[16]showed similar results that there was a significant increase in MPV in preeclampsia patients (44.5%) with duration of gestation as blood pressure increased. The increase was more significant as severity of preeclampsia increased (46.26% in mild preeclampsia vs51.02% in severe preeclampsia).

This suggests the role of platelets in pathogenesis of gestational hypertension. Hence any

increase in MPV from 1<sup>st</sup> trimester to 3<sup>rd</sup> trimester may be noted and pregnant women can be monitored more carefully. This helps in early diagnosis so that proper management line can be chosen and maternal and neonatal morbidity and mortality can be prevented.

## 5. Conclusion

We found that although MPV was in normal limits, it significantly increased from first to third trimester in GH compared to normal pregnant women. MPV has increased more than normal in few cases with severe forms of GH. This emphasizes the role of platelets in the pathogenesis of the disease.

MPV is one of the markers of platelet activation that can be increased even before the onset of clinical symptoms. It is done routinely as a part of antenatal check up. Hence any increase in MPV from 1<sup>st</sup> trimester to 3<sup>rd</sup> trimester may be considered as a caution and patients can be monitored more carefully to avoid adverse outcome.

Further studies are needed to explore the usefulness of MPV as a marker in GH. We found that MPV was raised in severe gestational hypertension and as we could not divide the cases based on severity, further studies are needed to find the association of MPV to various grades of hypertension.

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## References

- [1] Steven G. Gabbe, Jennifer R. Niebyl, Joe Leigh Simpson. *Obstetrics: Normal and problem pregnancies*. 5<sup>th</sup> ed. Philadelphia: Churchill Livingstone; 2007. P. 863-66.
- [2] Gary CunninghamF, Kenneth J. Leveno, Steven L. Bloom, John C. Hauth, Dwight J. Rouse, Catherine Y. Spong. *William's obstetrics*. 23<sup>rd</sup> edition. United States of America: McGraw Hill companies; 2010. 706-708.
- [3] Sharma Anjana, Mahendra Poonam, Bisht Shradha. Management of pregnancy induced hypertension. *Int J of Res in Ayu and Pharm* 2010 Nov-Dec; 1(2):390-398.
- [4] Veronique C. Balleger, Bernard Spitz, Luc A. DeBaene, Andre F. Van Assche, Melanie Hidajat, Arnold M. Criel. Platelet activation and

- vascular damage in gestational hypertension. *Am J Obstet Gynecol.* 1992; 166:629-33.
- [5] Lazarov R, Konijnenberg A, van der Post JA, Sturk A, Boer K. Preeclampsia not (yet) predictable from the blood platelet count.
- [6] Report of the National High Blood Pressure Education Program Working Group on High Blood Pressure in Pregnancy. *Am J Obstet Gynecol.* 2000 Jul; 183(1):S1-S22.
- [7] Abbassi-Ghanavati M, Greer LG, Cunningham FG. Pregnancy and laboratory studies: a reference table for clinicians. *Obstet Gynecol.* 2009 Dec; 114(6):1326-31.
- [8] Sibai BM, Caritis SN, Thom E. Prevention of preeclampsia with low-dose aspirin in healthy, nulliparous pregnant women. The National Institute of Child Health and Human Development Network of Maternal-Fetal Medicine Units. *N Engl J Med* 1993; 329:1213-8.
- [9] Hutt R, Ogunniyi SO, Sullivan MH, Elder MG. Increased platelet volume and aggregation precede the onset of preeclampsia. *Obstet Gynecol.* 1994 Jan;83(1):146-9.
- [10] Ozgur Dundar, Pinar Yoruk, Levent Tutuncu, Alev Akyol Erikci, Murat Muhcu, Ali Rustu Ergur, et al. Longitudinal study of platelet size changes in gestation and predictive power of elevated MPV in development of pre-eclampsia. *Prenat Diagn.* 2008 Nov; 28(11):1052-56.
- [11] Giles C., Inglis T. C. M.. Thrombocytopenia and macrothrombocytosis in gestational hypertension. *BJOG.* 1981 Nov; 88(11):1115-19.
- [12] Singer CR, Walker JJ, Cameron A, Fraser C. Platelet studies in normal pregnancy and pregnancy-induced hypertension. *Clin Lab Haematol.* 1986; 8(1):27-32.
- [13] Walker JJ, Cameron AD, Bjornsson S, Singer CR, Fraser C. Can platelet volume predict progressive hypertensive disease in pregnancy?. *Am J Obstet Gynecol.* 1989 Sep; 161(3):676-9.
- [14] Ioannis Karalis, Sunil k. Nadar, Eman Al Yemeni, Andrew D. Blann, Gregory Y.H. Lip. Platelet activation in pregnancy induced hypertension. *Thromb Res* 2005; 116(5):377-383.
- [15] Yin SM, Li YQ, Xie SF, Ma LP, Wu YD, NieDN, et al. Study on the variation of platelet function in pregnancy induced hypertension and gestational diabetes mellitus. *Zhonghua Fu Chan KeZaZhi.* 2005 Jan; 40(1):25-8.
- [16] Dadhich S, Agrawal S, Soni M, Choudhary R, Jain R, Sharma S, et al. Predictive value of platelet indices in development of preeclampsia. *J South Asian Feder Obst Gynaec* 2012; 4(1):17-21.