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ORIGINAL RESEARCH

PLATELET INDICES AS PROGNOSTIC MARKERS ACROSS THE SPECTRUM OF PREGNANCY-INDUCED HYPERTENSION: A PROSPECTIVE COMPARATIVE STUDY

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ABSTRACT

Background: Pregnancy-induced hypertension (PIH) complicates 6–10% of pregnancies and remains a leading cause of maternal and perinatal morbidity. Platelet activation is central to its pathophysiology through endothelial injury and consumptive coagulopathy. Mean platelet volume (MPV), platelet distribution width (PDW) and platelet-large cell ratio (P-LCR) are morphometric indices generated automatically during a routine complete blood count at no additional cost. This study evaluated their behaviour across the PIH severity spectrum. Methods: A prospective comparative study was conducted at a tertiary Armed Forces hospital in New Delhi between May 2016 and May 2018. Seventy-three women with PIH (gestational hypertension n = 29, preeclampsia n = 37, eclampsia n = 7) and 70 normotensive pregnant women beyond 20 weeks of gestation were enrolled (N = 143). Platelet count, MPV, PDW and P-LCR were measured on a Sysmex KX-21 analyser, with platelet counts independently verified on Leishman-stained smears by two pathologists blinded to clinical details. Between-group comparisons used the Mann–Whitney U test. Results: Mean platelet count was lower in PIH than in controls (155.3 ± 88.5 versus 222.0 ± 73.2 × 10^3/µL; $p < 0.001$). All three indices were higher in every PIH subgroup ($p < 0.001$ for each): MPV 11.90 ± 1.61 versus 10.30 ± 1.57 fL, PDW 17.67 ± 3.89 versus 14.26 ± 3.26 fL, and P-LCR 40.21 ± 9.82% versus 29.02 ± 10.49%. Values rose stepwise with severity, peaking in eclampsia (MPV 12.91 fL, PDW 21.01 fL, P-LCR 47.74%). In gestational hypertension the platelet count did not differ from controls ($p = 0.372$) although all three indices were already elevated ($p < 0.001$). Using the laboratory reference limits, P-LCR exceeded its threshold in 89.0% of PIH cases and PDW in 86.3%, against 48.6% for MPV. Conclusion: MPV, PDW and P-LCR rise in graded fashion across the PIH spectrum and are elevated in gestational hypertension at a stage when the platelet count is still normal, identifying a window of subclinical platelet activation that the count alone does not reveal. Because these indices are byproducts of a test already performed antenatally, they are attractive adjuncts to risk stratification. Their diagnostic performance cannot be judged from this study, however, since specificity was not assessed and the reference limits applied were not derived from a pregnant population; threshold derivation in pregnancy with formal receiver operating characteristic analysis is the necessary next step.

Keywords: Pregnancy-induced hypertension; Preeclampsia; Eclampsia; Mean platelet volume; Platelet distribution width; Platelet-large cell ratio; Prognostic markers.

INTRODUCTION

Pregnancy-induced hypertension (PIH) is defined as new-onset hypertension, systolic pressure of 140 mmHg or above or diastolic pressure of 90 mmHg or above, arising after 20 weeks of gestation in a woman previously normotensive [1]. It describes a clinical continuum running from gestational hypertension through preeclampsia to eclampsia, and complicates approximately 6–10% of pregnancies worldwide [2]. It remains among the leading causes of maternal death, through

abruptio placentae, cerebrovascular accident, multiorgan failure and disseminated intravascular coagulation, and exposes the fetus to growth restriction, prematurity and intrauterine death [2,3].

Platelets are central to this pathophysiology. Defective placentation and impaired trophoblast invasion provoke endothelial injury, platelet activation and accelerated peripheral consumption, and thrombocytopenia is consequently found in roughly half of women with preeclampsia [4,5]. The platelet count has therefore long been used as a bedside prognostic index in hypertensive pregnancy [6]. Counting platelets, however, captures only the end result of consumption. Automated impedance analysers additionally report morphometric indices that describe the population from which the count is drawn: mean platelet volume, which reflects average platelet size and, indirectly, metabolic and haemostatic activity [7]; platelet distribution width, which quantifies anisocytosis; and platelet-large cell ratio, the proportion of platelets exceeding 12 fL. Larger platelets are denser in granules, generate more thromboxane A_2 and aggregate more readily, so a rising MPV is widely interpreted as a marker of platelet activation and of accelerated thrombopoiesis under consumptive stress [8].

Several investigators have examined these indices in hypertensive pregnancy and have generally reported higher values in preeclampsia and eclampsia than in normotensive controls, with a gradient across severity categories [9,10,11]. Interest has been renewed by two recent studies: a multivariable analysis in which PDW and P-LCR remained independent predictors of preeclampsia after adjustment for age, gestational age, blood pressure and body mass index while the platelet count did not [12]; and a study correlating platelet indices with angiogenic markers, which found activated platelets to be a possible additional source of soluble fms-like tyrosine kinase-1 [13]. Together these suggest that the indices carry information about platelet biology that the count does not.

Despite this, the indices are seldom examined in routine antenatal practice in India, even though they are printed on every complete blood count report at no extra cost or effort. The present study was undertaken to characterise MPV, PDW and P-LCR prospectively across all three clinical subtypes of PIH at a tertiary Armed Forces hospital, to determine whether they vary in step with disease severity, and to establish whether they change before the platelet count does.

MATERIALS AND METHODS

Study design and setting

This prospective analytical comparative study was conducted jointly by the Departments of Pathology and Obstetrics and Gynaecology at Base Hospital Delhi Cantt, New Delhi, between 1 May 2016 and 1 May 2018.

The protocol was approved by the Institutional Ethics Committee (IEC No. 101/10/Oct/BH-2016, dated 27 October 2016) and the study was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from every participant. The study conducted according to the Declaration of Helsinki.

Participants

One hundred and forty-three pregnant women beyond 20 weeks of gestation were enrolled consecutively. Group I comprised 73 women with PIH (gestational hypertension n = 29, preeclampsia n = 37, eclampsia n = 7); Group II comprised 70 normotensive pregnant women.

Women beyond 20 weeks of gestation with a blood pressure of 140/90 mmHg or above, or proteinuria exceeding 300 mg in 24 hours, or oedema were enrolled as cases. Normotensive women beyond 20 weeks with no hypertensive disorder served as controls. Women were excluded if they had pre-existing diabetes mellitus, anaemia or a haemoglobinopathy, a haemorrhagic disorder, epilepsy, chronic hypertension or renal disease, or were taking any medication known to affect platelet count or function.

Cases were classified according to the criteria of the National High Blood Pressure Education Program Working Group [3]: gestational hypertension, blood pressure of 140/90 mmHg or above after 20 weeks without proteinuria in a previously normotensive woman; preeclampsia, hypertension with proteinuria exceeding 0.3 g in 24 hours or 1+ or more on dipstick; and eclampsia, new-onset generalised convulsions in a preeclamptic woman not attributable to another cause.

Laboratory methods

Under aseptic precautions, 3 mL of venous blood was collected into an EDTA vacutainer and processed within two hours. Platelet count, MPV, PDW and P-LCR were measured on a Sysmex KX-21 automated haematology analyser (Sysmex Corporation, Kobe, Japan). Every platelet count was independently confirmed on a Leishman-stained peripheral blood smear by two qualified pathologists blinded to clinical details, which guards against the spurious counts that platelet clumping and giant platelets can produce on impedance analysers.

The laboratory reference intervals applied were MPV 8.4–12.0 fL, PDW 8.0–14.0 fL and P-LCR 10–30%, and an index was recorded as elevated when it exceeded the upper limit.

Statistical analysis

Data were analysed in SPSS version 17.0 (SPSS Inc., Chicago, Illinois). Continuous variables are summarised as mean ± standard deviation. Normality was assessed by the Shapiro–Wilk test; platelet count and PDW departed significantly from normality ($p < 0.05$), and the Mann–Whitney U test was therefore used for all between-group comparisons of platelet parameters. Categorical variables were compared using the chi-square test. A two-sided p value below 0.05 was taken to denote statistical significance.

Twelve pairwise comparisons against the control group were performed (four parameters across three subgroups) without formal adjustment for multiplicity. A Bonferroni-corrected threshold would be 0.0042; every comparison reported as significant in this study has $p < 0.001$ and therefore survives correction, so the conclusions are unaffected. This is stated for transparency rather than as a correction applied.

No prospective sample size calculation was performed. A post-hoc adequacy assessment, supplied for completeness and to be read as descriptive rather than as justification, indicates that the observed differences between all PIH cases and controls correspond to standardised effect sizes of 0.82 for platelet count, 1.00 for MPV, 0.95 for PDW and 1.10 for P-LCR, for which 70 controls and 73 cases give power above 99.8% at a two-sided alpha of 0.05. Even the seven-woman eclampsia subgroup retained power above 98% against controls for each index, because the effect sizes at that end of the spectrum are very large (1.69 to 2.01). The sample was therefore adequate for the comparisons reported, though not, as noted in the Limitations, for threshold derivation.

RESULTS

Participant characteristics

All 143 enrolled women completed the study. The two groups were closely matched for age (Table 1), the largest stratum in each being 26–30 years. The difference in age distribution was not significant (chi-square 2.85, 4 degrees of freedom, $p = 0.583$); this test is supplied editorially, as the submitted draft asserted comparability without a formal comparison, and it should be regarded as approximate because two of the ten cells have expected counts below five, for which an exact test would be preferable. The mean age of the PIH group was 28.58 ± 4.48 years.

Table 1. Age distribution of study participants

Age group (years)	Controls (n = 70)	PIH cases (n = 73)	Total (N = 143)
16–20	2 (2.9)	1 (1.4)	3 (2.1)
21–25	25 (35.7)	19 (26.0)	44 (30.8)
26–30	29 (41.4)	32 (43.8)	61 (42.7)
31–35	11 (15.7)	15 (20.5)	26 (18.2)
> 35	3 (4.3)	6 (8.2)	9 (6.3)
Total	70 (100)	73 (100)	143 (100)

Values are n (%). Chi-square 2.85, df 4, p = 0.583.

Platelet count

The distribution of platelet counts by subgroup is shown in Table 2. Thrombocytopenia became both more frequent and more profound as disease severity increased. Among women with gestational hypertension, 23 of 29 (79.3%) had counts above 150 × 10³/µL, whereas no woman with eclampsia did, and four of the seven (57.1%) had counts below 50 × 10³/µL.

Table 2. Distribution of platelet counts across PIH subgroups

Platelet count (× 10³/µL)	Gestational HTN (n = 29)	Preeclampsia (n = 37)	Eclampsia (n = 7)
< 50	1 (3.4)	1 (2.7)	4 (57.1)
50–100	0 (0.0)	18 (48.6)	2 (28.6)
101–150	5 (17.2)	5 (13.5)	1 (14.3)
> 150	23 (79.3)	13 (35.1)	0 (0.0)

Values are n (%). HTN, hypertension.

Platelet count and platelet indices by severity

Mean platelet count fell progressively with severity while all three indices rose in parallel (Table 3). The finding of principal interest lies in the gestational hypertension column: the platelet count in this subgroup was statistically indistinguishable from that of controls (205.1 versus 222.0 × 10³/µL; p = 0.372), yet MPV, PDW and P-LCR were all already substantially and significantly raised (p < 0.001 for each). The dissociation persisted across the spectrum, with every index significant in every subgroup while the count became significant only from preeclampsia onwards.

Table 3. Platelet count and platelet indices by disease severity

Parameter	Control (n = 70)	Gestational HTN (n = 29)	Preeclampsia (n = 37)	Eclampsia (n = 7)
Platelet count (× 10³/µL)	222.0 ± 73.2	205.1 ± 66.3	135.3 ± 87.7	54.4 ± 31.2
p versus control	—	0.372	< 0.001	< 0.001
MPV (fL)	10.30 ± 1.57	11.70 ± 1.52	11.86 ± 1.72	12.91 ± 1.16
p versus control	—	< 0.001	< 0.001	< 0.001
PDW (fL)	14.26 ± 3.26	17.56 ± 3.47	17.12 ± 3.92	21.01 ± 4.30
p versus control	—	< 0.001	< 0.001	< 0.001
P-LCR (%)	29.02 ± 10.49	39.16 ± 10.62	39.60 ± 9.05	47.74 ± 8.13
p versus control	—	< 0.001	< 0.001	< 0.001

Values are mean ± standard deviation. Mann–Whitney U test. MPV, mean platelet volume; PDW, platelet distribution width; P-LCR, platelet-large cell ratio; HTN, hypertension. Pooled values for all 73 PIH cases, derived from the subgroup means and standard deviations, were: platelet count 155.3 ± 88.5 × 10³/µL, MPV 11.90 ± 1.61 fL, PDW 17.67 ± 3.89 fL and P-LCR 40.21 ± 9.82%.

Table 4. Proportion of PIH cases with elevated platelet indices

Index (upper limit)	All PIH	Gestational HTN	Preeclampsia	Eclampsia
MPV > 12 fL	35/72 (48.6)	13/29 (44.8)	16/36 (44.4)	6/7 (85.7)
PDW > 14 fL	63/73 (86.3)	25/29 (86.2)	31/37 (83.8)	7/7 (100.0)
P-LCR > 30%	65/73 (89.0)	24/29 (82.8)	34/37 (91.9)	7/7 (100.0)

The MPV denominator is 72 rather than 73 because one preeclampsia record contained a confirmed data-entry error and was excluded from that analysis only; PDW and P-LCR were available for all 73 cases. These proportions describe sensitivity alone. The corresponding proportions among the 70 controls were not determined, and the interpretation of this table depends on them; the point is developed in the Discussion.

DISCUSSION

In this cohort of 143 pregnant women, MPV, PDW and P-LCR were all significantly higher in every category of pregnancy-induced hypertension than in normotensive controls, and rose in graded fashion from gestational hypertension through preeclampsia to eclampsia. The platelet count fell along the same gradient. The observation of most practical interest is that the two do not move in step: in gestational hypertension the indices had already shifted substantially while the count remained normal.

Dissociation between count and indices

Platelet count in the gestational hypertension subgroup did not differ from controls ($p = 0.372$), yet MPV, PDW and P-LCR were all raised with p below 0.001. This is biologically coherent. Peripheral consumption is met initially by accelerated thrombopoiesis, and the young platelets released in compensation are larger and more heterogeneous than the population they replace. Platelet size and size dispersion therefore change while total platelet mass is still being maintained, and the count falls only when compensation fails. The indices are, on this reading, a measure of thrombopoietic strain rather than of platelet depletion, and it is precisely that distinction which gives them a potential early-warning role. The same pattern of dissociation was reported in the multivariable analysis of Li and colleagues, in which PDW and P-LCR predicted preeclampsia independently while platelet count did not [12].

Comparison with published series

The mean platelet count in the present PIH group ($155.3 \times 10^3/\mu L$) was significantly lower than in controls and lies within the range reported by Indian series (Table 5). The eclampsia value of $54.4 \times 10^3/\mu L$ is markedly lower than in the comparator studies. The most probable explanation is referral: as a tertiary Armed Forces centre this hospital receives a disproportionate share of severe and late-presenting cases, including women with features of HELLP syndrome, in whom profound thrombocytopenia is characteristic [14]. The normotensive and preeclampsia values, by contrast, sit comfortably within the published range, which argues that the difference is one of case mix rather than of measurement.

Table 5. Mean platelet count in the present study and in published Indian series ($\times 10^3/\mu L$)

Study	Normotensive	Preeclampsia	Eclampsia
Vrunda and Saila [4]	220	140	130
Dubey et al. [5]	230	190	180
Mohapatra et al. [6]	230	180	120
Annam et al. [9]	218	155	131
Present study	222	135	54

Values are rounded means as reported in the respective publications.

The mean age of the PIH group (28.58 ± 4.48 years) is slightly higher than that reported by Prakash and colleagues (24.75 ± 3.36 years) [15] and by Sandhya and colleagues [16], which plausibly reflects the demographic profile of a service population.

The individual indices

MPV rose from 10.30 fL in controls to 12.91 fL in eclampsia. Larger platelets carry more dense granules, generate more thromboxane A_2 and aggregate more readily, so an increase in mean size plausibly contributes to the vasospasm and microvascular thrombosis that characterise severe PIH [7,8]. The magnitude of the rise is in keeping with that reported by Alsheeha and colleagues [10] and by Wadhwa and colleagues [19].

PDW rose from 14.26 fL to 21.01 fL and quantifies anisocytosis, the simultaneous presence of large newly released platelets and small senescent ones that accompanies accelerated turnover. The gradient across severity categories

reproduces that reported by Freitas and colleagues [11] and by Karateke and colleagues [20], and PDW was the index most consistently abnormal at the mild end of the spectrum in this cohort.

P-LCR, the proportion of platelets exceeding 12 fL, rose from 29.02% to 47.74%. It is the most direct available surrogate for the release of large reticulated platelets from the marrow in response to peripheral consumption. Dionisio and Favero found a positive correlation between P-LCR and soluble fms-like tyrosine kinase-1, raising the possibility that these activated platelets are themselves a source of the antiangiogenic mediator rather than merely a marker of the process [13].

Why the apparent ranking of the three indices should not be relied upon

Table. shows P-LCR abnormal in 89.0% of cases, PDW in 86.3% and MPV in 48.6%, and it is tempting to read this as a ranking of usefulness. That reading is not safe, for two reasons that the present design cannot resolve. The first is the absence of specificity data. The reference limits applied were >14 fL for PDW and >30% for P-LCR, but the mean PDW of the normotensive control group in this study was 14.26 fL, which already exceeds its own threshold, and the mean control P-LCR was 29.02%, within one point of its threshold. Since roughly half of any group lies above its own mean, a substantial proportion of these healthy pregnant women would have been classified as abnormal by the same criteria. A cut-off placed at the centre of the reference population inflates apparent sensitivity while destroying specificity, and the higher yield of PDW and P-LCR over MPV, whose threshold of 12 fL sits about one standard deviation above the control mean, is at least partly an artefact of where the three thresholds happen to fall.

The second reason is that the three indices are not independent measurements. P-LCR is defined as the fraction of platelets exceeding 12 fL, and MPV is the mean of the same volume distribution; for any plausible platelet size distribution the two are monotonically related, and a comparison of their sensitivities at thresholds of 30% and 12 fL is close to comparing two points on a single curve. PDW is derived from the width of that same distribution. Treating the three as independent confirmatory markers therefore overstates the evidence they jointly provide.

None of this undermines the central finding. That all three indices differ highly significantly from control values, and do so in gestational hypertension before the count moves, rests on the comparison of continuous distributions between groups and is independent of where any threshold is drawn. What cannot be concluded from these data is which index performs best diagnostically, or what threshold should be used in practice. Both questions require pregnancy-specific reference intervals and receiver operating characteristic analysis in a larger sample.

CONCLUSION

MPV, PDW and P-LCR were elevated in every category of pregnancy-induced hypertension in this cohort and rose in graded fashion with clinical severity, reaching their highest values in eclampsia. The finding of most practical consequence is the dissociation seen in gestational hypertension, where all three indices were significantly raised while the platelet count remained indistinguishable from normal, indicating that subclinical platelet activation and thrombopoietic compensation are detectable before overt thrombocytopenia develops.

Because these indices are generated automatically alongside a complete blood count that is already performed as part of routine antenatal care, they cost nothing additional to obtain and require no new equipment. On the present evidence they are best regarded as a signal worth noticing when it appears, rather than as a validated screening test: this study establishes that the indices differ between groups, not how well they discriminate at the level of the individual woman. Establishing that will require pregnancy-specific reference intervals, formal receiver operating characteristic analysis in a larger multicentre sample, serial measurement from the first trimester, correlation with maternal and perinatal outcomes, and where available the measurement of immature platelet fraction and of angiogenic markers such as the soluble fms-like tyrosine kinase-1 to placental growth factor ratio. Complementary biochemical markers of severity, such as lactate dehydrogenase may usefully be assessed alongside them.

Limitations

The limitations of this study are substantial and constrain the conclusions accordingly. Specificity was not assessed, and the reference limits applied appear to have been derived from a general adult population rather than from pregnant women, in whom platelet size and dispersion may differ; this represents the most important limitation of the study and is discussed

above. The study was conducted at a single tertiary Armed Forces hospital, and therefore both the demographic profile and the severity distribution may not be representative of the wider obstetric population, as illustrated by the platelet counts observed in the eclampsia subgroup. The eclampsia subgroup comprised only seven women and was therefore too small for threshold derivation or receiver operating characteristic analysis, although the observed effect sizes at this end of the spectrum were sufficient for the group comparisons reported.

Measurement at a single time point precludes assessment of the trajectory of platelet indices, which would be necessary to establish genuine predictive rather than concurrent value. MPV is known to increase with time in EDTA-anticoagulated blood; although processing within two hours limits this effect, it does not completely eliminate it, and the exact interval between blood collection and analysis was not recorded for individual samples. Immature platelet fraction, which directly measures reticulated platelets and could further evaluate the proposed interpretation of thrombopoietic strain, was not available on the Sysmex KX-21 platform.

Gestational age at the time of sampling, mean blood pressure, and subgroup-specific proteinuria values were not available in a form that permitted their inclusion in the present analysis. Maternal and perinatal outcomes were not collected as study outcomes. Consequently, the prognostic value of the platelet indices for subsequent maternal or perinatal clinical events, as distinct from their association with disease category, remains untested.

Declarations

Ethics approval: Institutional Ethics Committee, Base Hospital Delhi Cantt (IEC No. 101/10/Oct/BH-16). Written informed consent was obtained from all participants.

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Conflicts of interest: None declared.

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