Section:

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ORIGINAL ARTICLE

Role of Different Platelet Indices as a Marker of Presence and Severity of Preeclampsia in Pregnant Primigravidas

Abd El-Moneim Mohamed Zakaria, Yasser Mohamed Diab, Mohammed Asaad El-Gharabily*

Department of Obstetrics and Gynecology, Faculty of Medicine, Al-Azhar University, Cairo, Egypt

Abstract

Background: Preeclampsia, which affects 5–8% of pregnancies worldwide, is a major cause of maternal and foetal mortality. Its prevalence differs among various populations and ethnic groupings.

Aim: To investigate the role of different platelet indices in detection of presence and severity of preeclampsia in pregnant primigravidas.

Patients and methods: The Sayed Galal and Al-Hussein University Hospitals’ Obstetrics and Gynecology Departments carried out this case–control study, Al-Azhar University’s Faculty of Medicine between the months of December 2021 and June 2022. This study was conducted on 120 consenting Primigravidas, within 24 h before delivery.

Result: The mean of platelet count (PLT) in Primigravidas with severe preeclampsia group was 180 with SD of 39.48, mean of PLT of patients in Primigravidas with nonsevere preeclampsia group was 210.85 with SD of 47.43, mean of PLT of patients in control group was 354.85 with SD of 79.19. Primigravidas with severe preeclampsia group, Primigravidas preeclampsia group and control group was highly significantly different regarding PLT (P = <0.001)

Conclusion: More importantly, because they may be easily obtained from a standard complete blood count (CBC) test, the determination of PLT parameters such as prenatal care (PC), mean platelet volume (MPV), and platelet distribution width (PDW) can be seen as a straightforward, quick, and affordable method in the diagnosis of preeclampsia (PE). As a result, we counsel doctors to identify PE development in expectant women using PLT characteristics.

Keywords: Marker of presence, Platelet indices, Preeclampsia, Primigravidas, Severity

1. Introduction

Preeclampsia is a leading cause of maternal and foetal mortality that affects 5–8% of pregnancies globally. Its prevalence differs among various populations and ethnic groupings.1

One of the reasons of preeclampsia has been attributed to inadequate trophoblastic invasion of the maternal vascular bed, which results in a reduction in placental blood flow, despite the fact that its specific pathophysiology is not fully known.2

Increased oxidative stress, aberrant placentation, cardiovascular maladaptation to pregnancy, dysfunctional genetic, immunological, nutritional, hormonal, and angiogenic mechanisms, as well as inflammation, are just a few of the many mechanisms and causes that have long been known. Preeclampsia’s precise pathogenesis has proven difficult to comprehend.3

Maternal endothelial failure, increased vascular permeability, and extensive systemic dysfunction are brought on by placental under perfusion.4

Women with preeclampsia have evidence of even greater platelet activation, which results in the development of platelet-derived microthrombi in narrower arteries.5

When platelets come into touch with damaged endothelium, the coagulation system is

* Corresponding author at: Obstetrics and Gynecology Department, Faculty of Medicine, Al-Azhar University, Cairo, Egypt.
E-mail address: drmohamedasaad@gmail.com (M.A. El-Gharabily).

https://doi.org/10.58675/2682-339X.1733
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stimulated, increasing both platelet production and consumption.6

The platelet is a 3 to 4 m-diameter, minimally synthesised a nuclear fragment of a bone marrow megakaryocyte that actively participates in the preservation of vascular integrity and primary hemostasis.7

The interaction of platelets with the arterial wall and their consequent role in the development of atheroma and thrombosis in the aetiology and pathogenesis of peripheral, coronary, cerebrovascular, and other vascular illnesses are of vital importance.8

The aim of this work was to investigate the role of different platelet indices in detection of presence, and severity of preeclampsia in pregnant primigravidas.

2. Patients and methods

As part of a case–control study, the Al-Azhar University Faculty of Medicine conducted this investigation in the obstetrics and gynaecology departments of Sayed Galal and Al-Hussein University Hospitals between December 2021 and June 2022. A total of 120 consenting Primigravidas, within 24 h before delivery, will be included in the study and categorized as: group (A): 40 healthy Primigravidas as controls. Group (B): 40 primigravidas with nonsevere preeclampsia. Group (C): 40 primigravidas with severe preeclampsia.

Inclusion criteria: Pregnant primigravidas, aging 18–35 years and Singleton pregnancy greater than 20 weeks of gestation.

Exclusion criteria: Primigravidas with other co-morbidities (chronic hypertension, diabetes mellitus, hematological disorders, chronic liver or kidney disease), multiple pregnancies and diagnosed fetal anomalies.

2.1. Methods

Patient’s evaluation: Detailed history including the onset of the maternal syndrome and detailed clinical examination.

2.2. Laboratory investigations

Blood: serum concentrations of lactate dehydrogenase, alanine aminotransferase, and aspartate aminotransferase, together with the levels of fibrinogen, Complete blood count (CBC), prothrombin time (PT), activated partial thromboplastin time (aPTT), LDH, and plasma thrombomodulin (TM) will be measured with Enzyme Linked Immunosorben Assay (ELISA).

Urine: 24 h urine collection for protein and creatinine as well as urinary albumin to creatinine ratio (uACr).

Study procedures: A 2 ml blood sample will be collected aseptically within 24 h of delivery and inserted EDTA bottles (ethylene diamine tetra-acetic acid). Platelet volume, mean platelet volume, and platelet distribution width are examples of platelet indicators, will be measured in the samples (platelet count (PLT), MPV, and platelet distribution width (PDW)), using the Automated Counter Sysmex 800i. The three study groups will be compared using the aforementioned platelet indicators.

Ethical considerations: The AL Azhar University Faculty of Medicine’s Obstetrics and Gynecology Department’s ethical committee submitted the study protocol for approval. Each participant who shared in the study gave informed verbal and written agreement after being informed of its goals and methods. At every stage of the investigation, confidentiality and personal privacy were respected.

2.3. Statistical analysis

Utilising version 23.0 of the statistical programme SPSS (Statistical Package for Social Sciences), the acquired data was tabulated and statistically evaluated. While descriptive statistics will be performed for categorical data in the form of number and percentage, they were carried out for both numerical parametric data in the form of median and first and third interquartile ranges as well as numerical nonparametric data in the form of mean SD (standard deviation), minimum and maximum of the range.

3. Results

Table 1 highlighted the research population’s demographic traits. Primigravidas with severe preeclampsia had a range in age from 18 to 32, individuals in the control group ranged in age from 18 to 32, with a mean and standard deviation of 24.42 3.48, while those with nonsevere preeclampsia had ages ranging from 18 to 33, with a mean and standard deviation of 24.02 3.42 Table 2.

PLT was extremely substantially different between the Primigravidas with severe preeclampsia group, the Primigravidas with nonsevere preeclampsia group, and the Control group (P = 0.001). The MPV between the groups of primigravidas with severe preeclampsia, primigravidas with nonsevere preeclampsia, and controls was extremely significant (P = 0.001). Groups of primigravidas with
Table 1. Demographic characteristics of the study population.

<table>
<thead>
<tr>
<th></th>
<th>Primigravidas with severe preeclampsia (n = 40)</th>
<th>Primigravidas with nonsevere preeclampsia (n = 40)</th>
<th>Control group (n = 40)</th>
<th>Test of Significance P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>Mean ± SD. 24.42 ± 3.48</td>
<td>24.02 ± 3.42</td>
<td>24 ± 3.44</td>
<td>F = 0.191</td>
</tr>
<tr>
<td></td>
<td>Median (IQR) 24 (22–27.25)</td>
<td>24 (21–26)</td>
<td>24 (21.75–26.25)</td>
<td>0.826</td>
</tr>
<tr>
<td></td>
<td>Range (Min–Max) 14 (18–32)</td>
<td>15 (18–33)</td>
<td>14 (18–32)</td>
<td></td>
</tr>
<tr>
<td>Gestation age (weeks)</td>
<td>Mean ± SD. 36.58 ± 2.02</td>
<td>36.98 ± 1.03</td>
<td>37.02 ± 2.48</td>
<td>F = 0.647</td>
</tr>
<tr>
<td></td>
<td>Median (IQR) 37 (35–38)</td>
<td>37 (37–37)</td>
<td>37 (35–38)</td>
<td>0.525</td>
</tr>
<tr>
<td></td>
<td>Range (Min–Max) 8 (32–40)</td>
<td>5 (34–39)</td>
<td>13 (32–45)</td>
<td></td>
</tr>
</tbody>
</table>

F, ANOVA test; IQR, interquartile range; SD, standard deviation.
P: P value for comparing between the studied groups.
P-value >0.05: Non significant; P-value <0.05: Significant; P-value <0.001: Highly significant.
P1: Group 1 vs Group 2.
P2: Group 2 vs Group 3.
P3: Group 1 vs Group 3.

Table 2. Platelet indices features in the study population.

<table>
<thead>
<tr>
<th></th>
<th>Primigravidas with severe preeclampsia (n = 40)</th>
<th>Primigravidas with nonsevere preeclampsia (n = 40)</th>
<th>Control group (n = 40)</th>
<th>Test of Significance P</th>
</tr>
</thead>
<tbody>
<tr>
<td>PLT (× 103/μl)</td>
<td>Mean ± SD. 180 ± 39.48</td>
<td>210.85 ± 47.43</td>
<td>354.85 ± 79.19</td>
<td>F = 103.699</td>
</tr>
<tr>
<td></td>
<td>Median (IQR) 187 (149.5–203.5)</td>
<td>208 (170.5–249.25)</td>
<td>353 (315.25–402)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>Range (Min–Max) 143 (98–241)</td>
<td>177 (117–294)</td>
<td>299 (204–503)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mean ± SD. 11.51 ± 1.33</td>
<td>10.14 ± 1.01</td>
<td>8.07 ± 0.81</td>
<td>F = 103.53</td>
</tr>
<tr>
<td></td>
<td>Median (IQR) 11.35 (10.88–12.3)</td>
<td>10.1 (9.57–10.82)</td>
<td>8.1 (7.3–8.6)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>Range (Min–Max) 5.8 (9–14.8)</td>
<td>4.2 (8–12.2)</td>
<td>3.1 (6.6–9.7)</td>
<td></td>
</tr>
<tr>
<td></td>
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<td></td>
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<td></td>
</tr>
<tr>
<td></td>
<td>Mean ± SD. 17.88 ± 2.28</td>
<td>15.48 ± 2.96</td>
<td>11.16 ± 2.79</td>
<td>F = 64.181</td>
</tr>
<tr>
<td></td>
<td>Median (IQR) 18.25 (16.1–19.3)</td>
<td>15.2 (13.85–18.23)</td>
<td>11.65 (9.23–12.62)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>Range (Min–Max) 11.1 (12.2–23.3)</td>
<td>14 (6.1–20.1)</td>
<td>13.6 (5–18.6)</td>
<td></td>
</tr>
</tbody>
</table>

F, ANOVA test; IQR, interquartile range; SD, standard deviation.
P: P value for comparing between the studied groups.
P-value >0.05: Non significant; P-value <0.05: Significant; P-value <0.001: Highly significant.
P1: Group 1 vs Group 2.
P2: Group 2 vs Group 3.
P3: Group 1 vs Group 3.

Table 3. Correlation between mean arterial blood pressure (MAP) and platelet indices.

<table>
<thead>
<tr>
<th>Mean arterial blood pressure (MAP)</th>
<th>Pearson’s correlation coefficients (r) P</th>
</tr>
</thead>
<tbody>
<tr>
<td>PLT</td>
<td>−0.73</td>
</tr>
<tr>
<td>MPV</td>
<td>0.706</td>
</tr>
<tr>
<td>PDW</td>
<td>0.645</td>
</tr>
</tbody>
</table>

P: P value for comparing between the studied groups.
P-value >0.05: Non significant; P-value <0.05: Significant; P-value <0.001: Highly significant.

severe preeclampsia and those without severe preeclampsia and control group was highly significantly different regarding PDW (P<0.001).

Table 3 Platelet markers and mean arterial blood pressure are shown to be correlated (MAP). Between MAP and PLT, Pearson’s Correlation Coefficient (r) was −0.73. Between MAP and MPV, The correlation coefficient (r) for Pearson was 0.706. Pearson’s Correlation Coefficient (r) between MAP and PDW was 0.645.
Table 4: Diagnostic parameters of the platelet indices.

<table>
<thead>
<tr>
<th>Diagnostic parameters</th>
<th>AUC</th>
<th>Cutoff value</th>
<th>Sensitivity</th>
<th>Specificity</th>
</tr>
</thead>
<tbody>
<tr>
<td>MPV</td>
<td>0.967</td>
<td>9.3 (fl)</td>
<td>89%</td>
<td>93%</td>
</tr>
<tr>
<td>PDW</td>
<td>0.920</td>
<td>13.5 (fl)</td>
<td>89%</td>
<td>83%</td>
</tr>
</tbody>
</table>

Table 4; Figs. 1 and 2 showed diagnostic parameters of the platelet indices. Regarding MPV, Cutoff value was 9.3, Sensitivity was 88.75%, Specificity was 92.50%, and area under the curve (AUC) was 0.966. PDW had an AUC of 0.919, a cutoff of 13.5, a sensitivity of 88.75%, and a specificity of 82.50%.

Table 5 showed Regression analysis of the platelet indices. R-squared between MAP and MPV was 0.499 with highly statistical significant difference ($P < 0.001$) between the two variables. R-squared between MAP and PDW was 0.415 with highly statistical significant difference ($P = <0.001$) between the two variables.

4. Discussion

Preeclampsia is a syndrome that often manifests after 20 weeks of pregnancy that affects both the mother and the foetus Nirupama and colleagues.

The Sayed Galal and Al-Hussein University Hospitals’ Obstetrics and Gynecology Departments carried out this case–control study, between December 2021 and June 2022, in the Faculty of Medicine, Al-Azhar University. Within 24 h of delivery, this study involved 120 primigravidas who gave their consent. All patients were split into 3 groups, with group (A) containing 40 primigravidas who were in good health as controls, group (B) included 40 primigravidas with nonsevere preeclampsia and group (C) included 40 primigravidas with severe preeclampsia.

Regarding the demographics of the study population, the current study revealed that the Age in Primigravidas with severe preeclampsia group ranged from 18 to 32 with mean SD = 24.42 3.48, while the Age in Primigravidas with nonsevere preeclampsia group ranged from 18 to 33 with mean SD = 24.02 3.42, and the Age in Control group ranged from 18 to 32 with mean.

Similar to the current work, Bawore and colleagues sought to identify the platelet indices pattern in preeclamptic women. The study included 120 pregnant women with normal blood pressure and 60 pregnant women with preeclampsia. In 60 cases, preeclampsia was discovered, 30 of which were moderate and the other 30 were severe. Ages 25, 20, and 36, 28, and 50, respectively, were the median ages in the full-year normotensive, non-severe, and severe preeclampsia groups. Age, gravidity, parity, gestational age, and BMI did not statistically differ between the three groups in this study. Also, Abd El-Rahman and colleagues, aimed to evaluate the significance of Platelet (PLT) count and platelet indices in the diagnosis of patients with preeclampsia (PE) and eclampsia. Women with PE had mean ages of 27.4 6.67 years and gestational ages of 33.6 3.9 weeks, whereas women with eclampsia had mean ages of 26.85 5.25 years and gestational ages of 32.1 3.21 weeks ($P = 0.16$). Age differences were not statistically significant, parity, gestational age, history of hypertension, PE, or BMI across the research groups.

Duan and colleagues, nevertheless, the investigation of biochemical indicators for evaluating illness severity and PE clinical assessment, and to provide further information on this topic. The patient
groups with severe PE \((n = 78)\), mild PE \((n = 85)\), were developed, along with healthy normotensive (control, \(n = 93)\). Contrary to our findings, the patients in the sPE group have an average age of 32 : 22 : 71 years. The average ages of participants in the control group and the mPE group, respectively, are 28 : 06 : 2 : 84 and 29 : 38 : 5 : 14 years, respectively. Women who acquired sPE are older than the control and mPE groups \((P \leq 0.05)\). Regarding gravidity, there is no discernible difference between the three groups \((P > 0.05)\). Additionally, the sPE group’s delivery timing is much sooner than that of the control and mPE groups \((P \leq 0.05)\).

The disagreement with our results may be due to the differences in inclusion criteria.

The current investigation revealed that there was a highly significant difference \((P = 0.001)\) regarding caesarean section between the three analysed groups. This is consistent with the findings of Duan and colleagues\(^{12}\) who discovered that the sPE and mPE groups had a lower rate of vaginal deliveries and a greater proportion of deliveries by CS than in the control group \((P \leq 0.001)\). Reddy and Prasad’s 13 findings indicated that nulliparous preeclampsia sufferers were more likely to have severe symptoms. Furthered this and necessitated surgical delivery rather than mild PE or control groups. In this study we found that the mean of MAP in Primigravidas with severe preeclampsia group was 125.95 with SD of 7.7, mean of MAP of subjects in Primigravidas with non-severe pre-eclampsia group was 113.78 with SD of 10.76, mean of MAP of subjects in Primigravidas with severe preeclampsia group was 125.95 with SD of 7.7, mean of MAP of subjects in Primigravidas with nonsevere preeclampsia group and control group was 81.92 with SD of 9.19. Primigravidas with severe preeclampsia group, Primigravidas with nonsevere preeclampsia group and control group was highly significantly different regarding MAP \((P = <0.001)\).

In agreement with our results Bawore and colleagues,\(^{10}\) reported that there were notable variations between the three groups in MPV and PDW. As the disease severity increased, the numbers were far higher \((P = 0.001)\). As the condition advanced from the mild to the severe stage in this investigation, the PLT count progressively fell, reaching values of 251 for normotensive, mild, and severe preeclamptic pregnant women \((139–445)\), 196.50 \((110–352)\), and 155 \((97–230)\), respectively \((P = 0.001)\). Also, Abd El-Rahman and colleagues,\(^{11}\) PLT counts were substantially lower in the PE group \((207 \ 42)\) and the eclampsia group \((142 \ 77)\) than in the control group \((214 \ 56)\) \((P = 0.03\) and \(P = 0.04\), respectively). Additionally, the PLT count was significantly lower in the eclampsia group compared with the PE group \((P = 0.01)\). In comparison to the control group \((7.85 \ 0.76)\), MPV was considerably higher in the PE group \((8.60 \ 0.95)\) \((P = 0.02)\).

MPC was significantly lower in the PE group \((P = 0.01)\) and the eclampsia group \((P = 0.03)\) compared with the control group. Additionally, the group with severe preeclampsia had considerably greater mean platelet volume (MPV) and PLT distribution width, according Reddy and Prasad’s 13 findings \((P = 0.001)\). However, Duan and colleagues,\(^{12}\) found that as compared with either healthy controls or mPE patients, the PLT was considerably lower in the sPE group. sPE groups’ only difference from the control groups’ MPV levels is determined to be significantly different. When the PDW of the three groups is compared, no meaningful results are found. Different inclusion criteria could be the cause of the disparity with our findings. Regarding the relationship between platelet indices and mean arterial blood pressure (MAP). Between MAP and PLT, Pearson’s Correlation Coefficient \((r)\) was –0.73. Between MAP and
MPV, Pearson’s correlation had a r value of 0.76. Reddy and Prasad 13 demonstrated that the mean platelet volume (MPV) and PLT distribution width were positively linked with the mean arterial pressure (r = 0.38 and 0.20, respectively). Which is consistent with our findings. Between MAP and PDW, The correlation coefficient (r) for Pearson was 0.645. According to Bawore and colleagues a MAP also demonstrated statistically significant positive relationships with PDW and MPV (rho = 0.731, P = 0.001 and 0.674, P = 0.001, respectively). These findings are consistent with those of the ongoing investigation. Furthermore, there was a significant negative correlation between MAP and PCT and PLT (rho = −0.369 and P = 0.001, respectively).

Bhavana and colleagues16’s study in Asia also reached the same outcome. Additionally, Tesfay and colleagues17 reported that, in line with our results, they reached the same outcome. Additionally, Tesfay and colleagues17 reported that, in line with our results, they reached the same outcome. Furthermore, according to Bawore and colleagues a MAP also demonstrated statistically significant positive relationships with PDW and MPV (rho = 0.731, P = 0.001 and 0.674, P = 0.001, respectively). These findings are consistent with those of the ongoing investigation. Furthermore, there was a significant negative correlation between MAP and PCT and PLT (rho = −0.369 and P = 0.001, respectively).

Consent for publication

I verify that all authors have agreed to submit manuscript.

Availability of data and material

Available.

Funding

No fund.

Conflict of interest

The authors affirm that the publication of this research will not have any bearing on their interests.

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