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Original Research Article

Study on significance of platelet indices, neutrophil to lymphocyte ratio and platelet to lymphocyte ratio as early parameters in prediction of preeclampsia

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Abstract

Background: Preeclampsia is a pregnancy-specific hypertensive disorder and a major contributor to maternal and perinatal morbidity worldwide. Its etiology is closely associated with abnormal placentation, oxidative stress, and widespread endothelial dysfunction. Recent studies have emphasized the potential of platelet activation markers and systemic inflammatory indices in predicting its onset. This study investigates the diagnostic utility of platelet indices and leukocyte-derived parameters during early pregnancy for identifying women at risk of preeclampsia and fetal growth restriction (FGR), offering a potentially cost-effective and timely screening approach.

Aims & Objective: To evaluate the significance of Platelet indices, Neutrophil to lymphocyte ratio (NLR) and Platelet to Lymphocyte ratio (PLR) as early pregnancy parameters to predict occurrence of Preeclampsia.

Materials and Methods: A prospective study carried out on 135 pregnant women in 8-14 weeks of pregnancy either visiting to OBG OPD for regular checkups or admitted under OBG department of JSS Hospital, Mysuru, Karnataka. A complete blood count was performed. “Hemoglobin, platelet count, mean platelet volume (MPV), plateletcrit, platelet distribution width (PDW), lymphocyte count, and neutrophil count were measured. Neutrophil to lymphocyte ratio (NLR) and Platelet to lymphocyte ratio (PLR) were calculated.

Results: In this study, 73 (54.1%) subjects were in age group of < 25 years. Majority of women (50.4%) were primigravidae. During follow-up, 35 (25.9%) women found to have preeclampsia. Mean platelet count, plateletcrit (PCT) and PDW were lower among women who developed preeclampsia and even more in severe PE compared to normal subjects. Mean Neutrophil and NLR were higher among women who developed preeclampsia and more so in severe PE. Mean lymphocyte count and PLR were lower in subjects who developed preeclampsia compared to normal subjects. Among 135, 20 had foetal distress of which 45% of babies were born to mothers with preeclampsia. A total of 23 (17.0%) women had foetal growth restriction. Mean platelet count, plateletcrit, PLR were lower among women who had foetal growth restriction compared to normal subjects.

Conclusion: Present study may conclude that there is a significant relationship between platelet indices and leucocyte parameters with preeclampsia. The estimation of these parameters may be considered as reliable, economic and rapid method for prediction of preeclampsia.

Keywords: Preeclampsia, NLR, PLR, Platelet indices, PDW.

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1. Introduction

Preeclampsia (PE), pregnancy specific hypertensive disorder (Blood pressure ≥140/90 mmHg on two occasions, at least six hours apart) associated with or without proteinuria (0.3g/day or a dipstick of 1+) after 20 weeks of gestation.[1] Preeclampsia affects 2-8% of pregnancies worldwide. Whereas in India, incidence of pregnancy induced hypertension is 10.3%.[2] Preeclampsia is the leading cause of maternal, perinatal morbidity and mortality. Major risk factors of preeclampsia include chronic hypertension, prior preeclampsia, cardiovascular disease, multiple gestations, advanced maternal age (>40 years) and obesity.[3]

The exact etiology of PE remains unclear. However, abnormal placentation plays a key role. Maternal uterine spiral arteries undergo vascular remodelling throughout the first trimester of a normal pregnancy. Trophoblast cells penetrate spiral arterioles and transform muscular arteries into large-bore, low-resistance vessels with high capacity. In first-trimester (10-12 weeks) trophoblasts invade up to decidual segments. The second wave invasion happens during 16-18 weeks and invade up to myometrial segments. By 20 weeks of pregnancy, this procedure is usually completed. In PE, this placentation process is disturbed and spiral arteries remain in a high resistance, vasoconstricted state.[4] This leads to impaired blood supply to foeto-placental unit and results in placental ischemia-reperfusion damage. Consequently, toxic substances such as free radicals are formed, causing placental oxidative stress (OS). The ischemic placenta produces inflammatory, anti-angiogenic, and oxidative chemicals, leading in systemic inflammation, oxidative stress, and activation of endothelium. The clinical presentation of PE occurs when endothelial activation and dysfunction develops in peripheral organs such as the liver, kidney, or brain. Further, which causes disturbance in the haemodynamic changes necessary for maternal adaptation to pregnancy.[5]

PE is a proven risk factor for developing cardiovascular disease later in life.[6] However, the question of whether inflammatory system activation occurs with PE and whether the present evidence is sufficient to justify extensive anti-immune system treatment techniques is currently being debated.[7]

Platelets play a key role in inflammatory process, wound healing, remodelling, angiogenesis, and microbial host defence, in addition to their critical role in hemostasis and thrombosis.8 Platelet indices, which include ‘mean platelet volume (MPV) and platelet distribution width (PDW), as well as platelet large cell ratio (PLCR), are a series of platelet characteristics collected as part of complete blood count.[9,10] Platelets indices have been recently used in the prediction, diagnosis, and prognosis of many diseases, including preeclampsia.[11,12] Platelet indices are platelet activation biomarkers. Platelet distribution width (PDW), Mean platelet volume (MPV), and platelet large cell ratio (PLCR), calculated collectively in CBC profiles.[8,13]

The rate of platelet formation and activation is indicated by MPV.[14] MPV considered as an inflammatory marker in some illnesses, such as preeclampsia.[15] In women at risk for preeclampsia, platelet activation begins as early as first month of pregnancy. Studies have revealed that when platelets are stimulated, their size increases, causing increase in MPV, PDW, and PLCR.[12,16]

Even though the exact cause of elevated blood pressure in preeclampsia is obscure, platelets get activated and attach to endothelium at the site of injury. In the early stages of hypertension in pregnancy, platelet aggregation is increased. As a result, the bone marrow produces large, young platelets, resulting in an increase in mean platelet volume (MPV).[17]

Early prediction of development of Preeclampsia is an important preventive strategy to reduce maternal as well as foetal morbidity and mortality. Hence, the present study was carried out: (a) To assess the significance of Platelet indices, NLR and PLR as early pregnancy parameters to predict occurrence of Preeclampsia; (b) To analyse the significance of Platelet indices, NLR and PLR as early pregnancy parameters to predict occurrence of Foetal Growth Restriction (FGR).

2. Materials and Methods

2.1. Study design

This is a prospective study was conducted in Department of Obstetrics and Gynaecology, JSS Hospital, Mysuru, Karnataka, India, after obtaining the approval from the Institutional Ethics Committee and written informed consent from all study subjects. The study duration was from November 2019 to April 2021. Sample size was calculated with 90% power and 95% confidence interval by using the formula n=2Sp2[Z1-α/2 + Z1-β]2/μd2, Sp2 = S12 + S22/2.

Sample size was found to be 125.6 which is rounded off to 135 considering compensation of dropout rates. The data was collected from pregnant women in 8-14 weeks of pregnancy, either visiting to Department of Obstetrics and Gynaecology - OPD for regular check-ups or admitted under OBG department of JSS Hospital, Mysuru, Karnataka, India. A total of 135 women pregnant women in 8-14 weeks of pregnancy were included in this study.

2.2. Diagnosis of preeclampsia

Preeclampsia was diagnosed with blood pressure of ≥140/90 mmHg noted for the first-time during pregnancy on two occasions at least four hours apart, after 20 weeks of gestation with proteinuria of ≥300 mg/24 hours or +1 by dipstick method in a random urine sample. Mild preeclampsia was considered when blood pressure of ≥140/90 mmHg or more on two occasions at least 4 hours apart after 20 weeks of gestation and with proteinuria (dipstick reading of +1).

Severe preeclampsia was defined as the presence of any of the following criteria: SBP ≥160 mmHg or DBP ≥110 mmHg on two separate measurements, performed at six-hour intervals, elevated serum creatinine concentrations >1.1 mg/dL or doubling of the serum creatinine concentrations in the absence of other renal diseases, elevated liver transaminases to twice normal concentration, platelet count less than 100,000/microliter, headache, visual impairment, epigastric pain or pain in the right upper quadrant.[18] FGR diagnosis is made when estimated foetal weight (EFW) in 3rd trimester scan is less than 10th percentile along with doppler changes. One thirty-five pregnant women in 8-14 weeks of pregnancy either visiting to OBG OPD for regular check-ups or admitted under OBG department of JSS Hospital, Mysuru, were considered for the study.

2.3. Inclusion criteria

Singleton pregnancies, period of gestation: Between 8 to 14 weeks, maternal age 20-35 years.

2.4. Exclusion criteria

Poor obstetric history such as previous occurrence of Preeclampsia, recurrent pregnancy loss, preterm labour, history of FGR or IUFD (Intra Uterine Foetal Demise), women with systemic diseases such as Diabetes mellitus, hypertension, cardiac disorders, renal and hepatic diseases, pregnancies with foetal chromosomal anomalies and congenital defects, family history of Preeclampsia, Obese women with BMI>30 Kg/m^2.

Under aseptic conditions, 3ml of venous blood sample was collected from study subjects in EDTA vacutainers for Complete Hemogram. Samples were analysed in JSS Hospital Haematology laboratory. NLR and PLR were calculated. Five mL urine sample was collected for urinary protein analysis by dipstick method. Blood pressure was measured. Patients were followed up with regular ANC checkups until termination of pregnancy. Those who develop Preeclampsia during the course were watched for development of the following - Threatened abortion (bleeding PV <28 weeks), Preterm labour, Eclampsia, Antepartum haemorrhage, DIC, HELLP Syndrome, Post-partum haemorrhage, Shock, Intra uterine growth restriction, Intra uterine foetal demise. Socio demographic and clinical details were obtained from all the study subjects. A general, physical and obstetric examination were carried out.

During the follow-up, 35 pregnant women developed preeclampsia and 100 remained normotensives. Out of 35 preeclamptics, 23 had mild preeclampsia and 12 had severe preeclampsia. Therefore, based on the development of signs and symptoms subjects were divided into normal (n=100) and preeclampsia (n=35) groups and blood indices of these two groups obtained at the beginning in the form of complete hemogram were compared to see if women who develop Preeclampsia will have deranged blood indices in early pregnancy or not.

2.5. Statistical analysis

Results were expressed as mean ± SD. Categorical variables were expressed in percentages. Inferential statistics like Chi-square test, one-sample t test was used. P value ($p<0.05$) considered as significant. Analysis was performed using SPSS, version 22.0.

3. Results

The baseline characteristics of study subjects were presented in **Table 1**. In this study, 73 (54.1%) subjects were < 25 years of age, 49 (36.3%) were in 26-30 years of age and 13 (9.6%) were 31 years and above. Majority of them, 68 (50.4%) were primigravida and 67 (49.6%) were multigravida. Preeclampsia was observed in 35 (25.9%) pregnant women.

Table 1: Demographic details of the study subjects

Demographic details	No. (percentage)
Age (years) group	
Less than 25	73 (54.1%)
26-30	49 (36.3%)
31 and above	13 (9.6%)
Parity	
Primigravida (n, %)	68 (50.4%)
Multigravida (n, %)	67 (49.6%)
Preeclampsia	
Absent	100 (74.1%)
Present	35 (25.9%)

In this, SBP (154.91 ± 9.88 mmHg), DBP (98.51 ± 11.04 mmHg), Neutrophil (82.84 ± 6.71 %), and NLR (8.81 ± 5.77) were significantly increased and Platelet count (1.98 ± 0.81), Lymphocyte (12.78 ± 6.35%) and Birth weight (2.58 ± 0.58kg) were significantly decreased in preeclampsia subjects compared to women who did not developed preeclampsia (**Table 2**).

In this, SBP (170.80 ± 9.01 mmHg), DBP (120.92 ± 14.14 mmHg) were significantly increased and Platelet count (1.59 ± 0.61) was significantly decreased in severe preeclampsia compared to mild preeclampsia. However, NLR and PLR were increased in severe preeclampsia, did not reached statistical significance (**Table 3**).

In this study, 73.9% of children born to women with preeclampsia were found to have foetal growth restriction, which was found to be statistically significant (**Table 4**).

Platelet count (2.13 ± 0.83), plateletcrit (0.22 ± 0.06) were lower among women who had foetal growth restriction compared to those who did not. Only platelet count was found to be significant. PDW, MPV were slightly on higher side among women who had foetal growth restriction compared to those who did not (**Table 5**).

In this study, significantly increased neutrophil count (82.53 ± 6.50%) and NLR (7.78 ± 5.00) were observed

among women who had foetal growth restriction compared to the ones who did not. Lymphocyte (13.73 ± 5.60) and PLR (26.31 ± 7.48) were lower among women who had foetal growth restriction compared to the ones who did not. Lymphocyte count was found to be significant (**Table 6**).

Table 2: Comparison of study parameters between women with and without preeclampsia

Parameters	Preeclampsia		p-value
	Present (n=35)	Absent (n=100)	
Systolic blood pressure (SBP) (mmHg)	154.91 ±9.88	115.30 ±16.81	0.001
Diastolic blood pressure (DBP) (mmHg)	98.51 ± 11.04	74.84 ±6.45	0.001
Hb (%)	11.58 ± 1.77	11.99 ± 1.14	0.111
PCV (%)	34.40 ±4.4	35.28 ± 3.9	0.295
Platelets, x (10^9/L)	1.98 ± 0.81	2.70 ± 0.89	0.001
Platelet crit (%)	0.22 ±0.08	0.25 ±0.07	0.116
PDW (%)	15.56 ±1.58	15.63 ±1.81	0.841
MPV (fl)	10.28 ± 0.89	10.05 ± 1.41	0.373
Neutrophil (%)	82.84 ± 6.71	76.06 ± 7.89	0.001
Lymphocyte (%)	12.78 ±6.35	18.64 ±6.94	0.001
NLR	8.81 ± 5.77	5.12 ± 3.38	0.001
PLR	26.75 ± 6.63	27.37 ± 9.08	0.712
Birth weight (kg)	2.58±0.58	2.99 ±0.38	0.001

Hb: Hemoglobin; PCV: Packed cell volume; PDW: Platelet distribution width; MPV: Mean platelet volume; NLR: Neutrophil to lymphocyte ratio; PLR: Platelet to lymphocyte ratio

Table 3: Comparison of study parameters between mild preeclampsia and severe preeclampsia

Parameters	Mild PE (n=23)	Severe PE (n=12)	p-value
Systolic blood pressure (SBP) (mmHg)	150.91 ±8.12	170.80 ±9.01	0.001
Diastolic blood pressure (DBP) (mmHg)	99.771 ± 10.21	120.92 ± 14.14	0.001
Hb (%)	11.43 ± 1.50	11.85 ± 2.24	0.523
PCV (%)	33.02 ±4.2	34.08 ± 2.5	0.280
Platelets, x (10^9/L)	2.18 ± 0.83	1.59 ± 0.61	0.042
Platelet crit (%)	0.24 ± 0.07	0.19 ± 0.05	0.136
PDW (%)	15.44 ±1.81	15.80 ±1.05	0.526
MPV (fl)	10.38 ± 0.94	10.09 ± 0.78	0.371
Neutrophil (%)	81.77 ± 6.39	84.89 ± 7.11	0.197
Lymphocyte (%)	13.44 ± 6.67	11.52 ± 5.74	0.403
NLR	8.35 ± 5.89	9.6 ± 5.68	0.528
PLR	26.25 ± 7.46	27.70 ± 4.79	0.548

Table 4: Relationship between foetal growth restriction and preeclampsia

Foetal growth restriction	Preeclampsia		Total	Chi square	p-value
	Absent	Present			
Absent	94 (83.9)	18 (16.1)	112 (83.0)	33.24	0.001
Present	6 (26.1)	17 (73.9)	23 (17.0)		
Total	100	35	135		

Table 5: Difference in platelet parameters between the women with and without foetal growth restriction

Parameters	Foetal growth restriction		T	p-value
	Absent	Present		
Platelets, x (10^9/L)	2.59 ± 0.93	2.13 ± 0.83	2.187	0.031
Plateletcrit	0.24 ± 0.08	0.22 ± 0.06	1.258	0.211
PDW	15.61 ± 1.83	15.65 ±1.34	0.096	0.924
MPV	10.08 ± 1.34	10.25 ± 1.10	0.576	0.566

Table 6: Difference in leukocyte related parameters between the women with and without foetal growth restriction

Parameters	Foetal growth restriction		T	p-value
	Absent	Present		
Neutrophil (%)	77.33 ± 8.08	82.53 ± 6.50	2.89	0.004
Lymphocyte (%)	17.82 ± 7.36	13.73 ± 5.60	2.51	0.013
NLR	5.75 ± 4.42	7.78 ± 5.00	1.922	0.050
PLR	27.39 ± 8.71	26.31 ± 7.48	0.554	0.580

Table 7: Relationship between foetal distress and preeclampsia

Foetal Distress	Preeclampsia		Total	Chi square	p-value
	Absent	Present			
Absent	89 (77.4)	26 (22.6)	115 (85.2)	4.448	0.035
Present	11 (55.0)	9 (45.0)	20 (14.8)		
Total	100	35	135		

Table 8: Relationship between blood group and preeclampsia

Blood Group	Preeclampsia		Total	Chi square	p-value
	Absent	Present			
A-	1 (50.0)	1 (50.0)	2 (1.5)	2.061	0.914
A+	24 (77.4)	7 (22.6)	31 (23.0)		
AB+	8 (72.7)	3 (27.3)	11 (8.1)		
B-	1	0	1		
B+	21 (77.8)	6 (22.2)	27 (20.0)		
O-	1 (50.0)	1 (50.0)	2 (1.5)		
O+	44 (72.1)	17 (27.9)	61 (45.2)		
Total	100	35	135		

Out of 135 subjects, foetal distress was present in 20 (14.8%) women. In this study, it was observed that 45% of children born to mothers with preeclampsia were having foetal distress and this association was found to be statistically significant (**Table 7**).

It was observed that, A - negative and O - negative blood groups had 50% incidence of preeclampsia followed by O - positive with 27.9% and AB - positive with 27.3% incidence of preeclampsia there was no statistically significant association between blood groups and preeclampsia (**Table 8**).

In the current study, it was observed that, 46.8% of women with preeclampsia underwent emergency LSCS and 5.6% underwent elective LSCS while 9.1% delivered vaginally. This association was found to be statistically significant.

4. Discussion

Preeclampsia is a life-threatening pregnancy-specific disorder with no current treatment. In the pathophysiology of preeclampsia, role of placenta is crucial and is considered as a source of inflammation and release of vasoconstrictor molecules to initiate endothelial cell injury, endothelial dysfunction and vasoconstriction.[19]

A total of 135 women in 8-14 weeks of gestation were enrolled in this study, majority i.e., 73 (54.1%) were in age group of <25 years followed by 49 (36.3%) between 26 to 30 years and least 13 (9.6%) were above 31 years. These findings were supported by Gogoi et al, reported mean age of women 28.15 ± 5.55.[20] Another study by Mannaerts et al, reported maternal age of preeclampsia subjects was 28.01 ± 4.91 years.[21]

In this study, 35 (25.9%) subjects developed preeclampsia. Among them, 23 (65.7%) had mild -PE and 12 (34.3%) had severe PE. In contrast, Gogoi et al, study reported that 38 women had mild PE and 29 had severe-PE.[20]

In this study, preeclampsia women had lower platelet counts, plateletcrit (Pct), PDW and PLR compared to women without preeclampsia. The mean platelet count and PLR were significantly reduced in the study group compared to control group in Gogoi et al, study, which is consistent with our findings.[20] Compared to MPV, PDW is a more accurate indicator of activation of platelets. PDW and plateletcrit levels have been reported to rise significantly in preeclampsia and have been linked to disease severity in several studies.[22,23]

In our study, mean Neutrophil, NLR were significantly higher among women who developed preeclampsia compared to the ones who did not. Mean Neutrophil and NLR were higher among women who had foetal growth restriction

compared to the ones who did not. Among these, mean neutrophil levels were found to be statistically significant. These findings were supported by Gogoi et al, reported increased NLR in preeclampsia group.[20] These markers have been examined in diabetes, ulcerative colitis, coronary artery disease, and cancer, among other illnesses.[24,25]

Increased activity of inflammatory cells and immunologic responses in preeclampsia have been linked to the release of inflammatory cytokines and autoantibodies, as well as increased superoxide generation, culminating in endothelial dysfunction.[26] Dysregulation of helper T cells (TH1 & TH2) immunological response is linked to preeclampsia. When comparing severe preeclampsia women to healthy nonpregnant women, Yavuzcan et al, reported significant rise in NLR.[27] Preeclamptic women and those with severe PE on the other hand, have considerably higher NLR than normal pregnant females. A study by Kurtoglu et al, reported significantly increased NLR in preeclampsia.[28]

In our study mean lymphocyte count and PLR were lower among subjects who developed preeclampsia compared to the ones who did not. However, mean lymphocyte count was significant. Placental hypoxia, diminished antioxidants and increased oxidative stress as well as an increase in proinflammatory markers were well documented in pathophysiology of preeclampsia.[29,30] However, PLR values were not statistically significant across groups in our study.

Concerned with severity of preeclampsia, mean platelet count, PLR, plateletcrit, MPV were lower among women who developed severe preeclampsia. The difference in mean platelet count and PLR among women with stages of preeclampsia was found to be significant. Mean MPV and PDW were slightly on higher side among women who had severe preeclampsia. These findings were supported by Gogoi et al, study.[20]

Platelet activation is a key element in pathophysiology of preeclampsia. An increased platelet activation phase occurs before onset of preeclampsia conditions with thrombocytopenia. Increased MPV is a sign of high megakaryocyte synthesis in the bone marrow as a result to thrombocytopenia stress, and it varies according to the degree of the inflammatory response. When compared to controls, platelet count was reduced and MPV was increased in subjects with preeclampsia in our study. Vilchez et al, and Kashanian et al, reported higher MPV levels in preeclampsia.[31,32]

Women with severe preeclampsia exhibited greater mean neutrophil count, NLR and lymphocyte count. Women with severe preeclampsia, on the other hand, had slightly lower lymphocyte levels. These findings are consistent with those of Gogoi et al.[20]

Concerned with foetal growth restriction, preterm birth and birth of small-for-gestational-age (SGA) children, preeclampsia is strongly linked to low birth weight.[33] In this study, preeclampsia was more common in gestational age before 37 weeks.

In this, 62 (45.9%) women were delivered by emergency LSCS and 53 (39.3%) were delivered by FTVD. 46.8% of women with preeclampsia underwent emergency LSCS and only 5.6% underwent elective LSCS and 9.1% underwent FTVD.

In a large cohort study of babies at >23 weeks (average being 37 weeks), there was an elevated risk of RDS where there was a hypertension issue in the mother, as well as differing neonatal outcomes in cases of moderate chronic hypertension, maternal prenatal hypertension and mild preeclampsia.[34] In the retrospective cohort study by Wen et al, found that 4788 (60.3%) of the women had LSCS section in PE group which is higher compared to control group without PE.[35]

Babies born to mothers who had preeclampsia had significantly lower birth weights than their healthy peers. These findings are consistent with Gunnarsdottir et al, study.[36] In this, Foetal distress was present in 20 (14.8%) subjects. Among 135 (100%), 45% of babies born to mothers with preeclampsia were having foetal distress. This is in accordance with the study by Nagraj et al, reported more cases of foetal distress in preeclampsia compared to control subjects.[37]

In present study, out of 135 (100%), 23 (17.0%) women had foetal growth restriction. 73.9% of babies born to women with preeclampsia were found to have foetal growth restriction. The mean platelet count, plateletcrit, PLR was lower among women who had foetal growth restriction compared to those who did not. The difference in mean platelet count among women with and without foetal growth restriction was found to be statistically significant. Mean lymphocyte, Neutrophil and NLR were higher among women who had foetal growth restriction compared to ones who did not. There was statistical significance in mean neutrophil levels among these differences.

When comparing women who ended up with preeclampsia to those who did not, the mean haemoglobin level was lower. Subjects with severe preeclampsia had a slightly increased mean haemoglobin level. Haemoglobin level was slightly lower among women with foetal growth restriction compared to the ones who did not. It is similar to the study of Gogoi et al.[20]

PCV was slightly lower among subjects with preeclampsia. In contrast, Heilmann et al, reported that PCV was considerably higher in women with PE compared to normal women.[38] However, few studies have found no link

between the MCHC parameter and Hb level changes in preeclampsia.[39,40]

A-negative and O-negative blood groups had 50% incidence of preeclampsia followed by O-positive with 27.9% and AB-positive with 27.3% incidence of preeclampsia there was no statistical significance between blood groups and preeclampsia.

5. Conclusion

In conclusion, platelet count, neutrophil and lymphocyte count, MPV, NLR and PLR were useful to detect the risk of PE, due to increased platelet destruction, inflammatory process and platelet turnover in patient with preeclampsia. Increasing MPV, PDW and PCT along with decreasing platelet count, Lymphocyte count and PLR may play a role in predicting preeclampsia. Furthermore, measurement of platelet indices and leucocyte counts may be regarded as a simple, reliable, cost-effective, and timelier method for detecting preeclampsia and determining its severity. Further studies with large sample size are recommended.

6. Ethical Approval

JSS/MC/PG/5189/2019-20.

7. Authors' Contributions

Dr. Meghadeepa S, Dr. Harshadeepa S, Dr. Sahana Kashyap, Pooja Shekar were involved in the study conception and design, data collection, analysis and interpretation of results, Dr. Yashas Ullas L and Dr. Suma K were involved in the critical review of manuscript along with all authors. All authors approved the final manuscript.

8. Source of Funding

None.

9. Conflict of Interest

None.

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