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Association of Plateletcrit Value with Gestational Diabetes Mellitus: A Case Control Study in A Tertiary Care Setting

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ABSTRACT

Gestational Diabetes Mellitus (GDM) is linked to numerous maternal and fetal complications. Typically diagnosed in the third trimester with OGTT, early detection could prevent many adverse outcomes. Emerging evidence highlights the role of platelets in GDM pathogenesis, suggesting platelet indices, including plateletcrit, might aid early diagnosis. **Objective:** To determine the association between GDM and elevated plateletcrit levels. **Methods:** A case-control study was conducted over six months (May - October 2024) in Gynaecology and Obstetrics Unit 1, Sir Ganga Ram Hospital, Lahore. After ethical approval, 100 pregnant women meeting the inclusion criteria were enrolled. At 24-28 weeks' gestation, all participants underwent a 75g OGTT. Based on results, they were divided into Group A (controls with normal OGTT) and Group B (cases with deranged OGTT). Plateletcrit values from the second trimester were obtained retrospectively from medical records. Quantitative variables were analyzed using Student's t-test ($P \leq 0.05$ deemed significant), while qualitative variables were expressed as percentages. **Results:** Group B (GDM patients) had significantly higher mean plateletcrit values (0.24 ± 0.08) compared to Group A (non-GDM) (0.14 ± 0.03) ($P = 0.000$). Conversely, the mean platelet count was higher in Group A than Group B ($P = 0.000$). **Conclusion:** Pregnant women with GDM exhibited higher plateletcrit values and lower platelet counts compared to non-GDM women, suggesting plateletcrit as a potential marker for early GDM diagnosis.

INTRODUCTION

Gestational Diabetes Mellitus (GDM) refers to glucose intolerance that develops or is recognized for the first time during pregnancy [1]. It affects approximately 4-8% of pregnant women, out of which 10-15% women develop complications. GDM increases the risk of feto-maternal and neonatal complications such as macrosomia, polyhydramnios, intrauterine death, instrumental delivery, caesarean section, respiratory distress syndrome, perinatal morbidity and mortality. Complications related to GDM can be prevented by early diagnosis [2]. In pregnant women having genetic predisposition, placental hormones like growth hormone, placental lactogen, progesterone and cortisol cause increase in insulin resistance which ultimately leads to GDM. Under the effect of placental lactogen lipolysis increases which increase the free fatty acid level in the body. These free fatty acids further enhance insulin resistance, which causes acceleration of sub-chronic inflammation. The pro-inflammatory cytokines in sub-chronic inflammation contribute to causation of GDM [3]. It is suggested that in gestational diabetes mellitus, there is platelet activation as a result vascular endothelial injury caused by sub-chronic inflammation and insulin resistance [4]. Due to increased platelet turnover and platelet production in bone marrow, younger platelets are released into circulation. These younger platelets are larger and more reactive than smaller ones due to enhanced enzymatic and metabolic activity. This leads to increase in platelet count and platelet indices

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(including Platelet distribution width (PDW), plateletcrit (PCT), and Mean platelet volume (MPV) [5]. Plateletcrit (PCT), a parameter calculated as the product of mean platelet volume (MPV) and platelet count, indicates the total platelet mass in volume of blood circulation. It is suggested to be one of the best indicators reflecting platelet function [3]. The normal plateletcrit during pregnancy is reduced due to expanded plasma volume (0.17) [6]. These pathophysiological changes are suggested to occur weeks or months before GDM is diagnosed. GDM is screened at 28 weeks' gestation using screening tools like glucose challenge test (GCT) or Oral glucose tolerance test (OGTT). Literature have documented the potential role of platelets in pathological mechanism of development of diabetes. High plateletcrit value has the potential of being used as a screening test to know the risk of GDM. Fashami *et al.*, found statistically significant association of platelet indices with gestational diabetes mellitus ($p<0.001$). The sensitivity and specificity of plateletcrit has been found to be higher than other platelet indices [2]. Chandra and Shetty, reported significantly higher platelet count ($p <0.01$) and lesser mean platelet volume (MPV) ($p <0.001$), but he found no difference in plateletcrit value ($p= 0.75$) in women with GDM. Khan and Ashraf, observed mean platelet volume (MPV) ($p=0.002$*) and platelet distribution width (PDW) ($p=0.010$*) to be significantly increased in pregnant women with GDM compared to the apparent healthy pregnant women ($p<0.05$). they did not study the plateletcrit value [7, 8]. Many other researches also studied MPV and PWD in GDM, although plateletcrit is a better parameter reflecting platelet function. Current study was conducted with the objective of determining the association of higher plateletcrit with GDM. Association of high plateletcrit value with GDM, if proven may help us detecting probable risk of the disease long before the clinical diagnosis or abnormality of OGTT or GCT. An additional benefit is the low cost and wide availability of CBC, making it a convenient screening option. Early positive screening with CBC may warn the patient and help initiate lifestyle modifications and planning a diagnostic test for GDM for better pregnancy outcome [9].

Considering the conflicting findings in the literature, this study was carefully planned and designed.

METHODS

This was a case control study conducted over 6 months' period (May 2024 to October 2024) at Obstetrics and gynaecology Unit 1, Sir Ganga Ram Hospital, Lahore. Blood samples were collected from all participants at the time of the screening (24–28 weeks of gestation). The blood was processed as follows: after collection, samples were immediately analyzed for Complete Blood Count (CBC) and plateletcrit, with the results being retrospectively reviewed from the patients' medical records. The sample size was calculated using mean Platelet count in controls group (193.0 ± 55.06) and cases is (144.5 ± 61.9) by taking 80% power of test, 5% margin of Error and 20% drop out rate was 100 (50%) in each group [10]. After permission of Institutional Ethical Committee (No.81-Gynea/Synopsis/ERC), 100 pregnant patients, 20-35 year-old, with singleton pregnancy (confirmed on ultrasound), gestational age 24 -28 weeks (calculated from LMP OR first trimester ultrasound) were enrolled in the study. The age range was kept as 20-35 years as after age of 35 many of patients diagnosed as GDM are known diabetics. The gestation was chosen to be around 28 weeks as OGTT is done at this gestation, and patients were grouped based on OGTT results. To enroll the patients, non-probability consecutive sampling technique was used. Potential confounders, including medical history and concurrent conditions, were minimized by applying strict exclusion criteria. Patients with prior history of GDM, overt diabetes mellitus, alcohol intake, smoking, and any medical or obstetrical disorders (e.g., hypertension, thyroid disease, autoimmune disorders, or polycystic ovarian syndrome) were excluded from the study. Before enrollment written informed consent from was taken from the patients to fulfill the selection criteria. The patients were screened at 24-28 weeks' gestation with 75gm Oral Glucose Tolerance Test (OGTT). To diagnose GDM International Diabetes Federation guidelines was used stating fasting blood glucose level >92mg/dl, glucose level 1-hour post glucose administration>180mg/dl or glucose level 2 hour after glucose administration >153mg/dl [11]. On the basis of result of OGTT the patients were divided into two groups, each group comprising of 50 participants. Women having normal OGTT were enrolled in Group A (Controls) whereas, those having deranged OGTT, diagnosed as GDM were included in Group B (Cases). Patient's CBC with plateletcrit during second trimester was retrospectively taken from her medical record. Data including patients age, parity, gestational age, platelet count and plateletcrit were recorded on preformed proforma. Confounding variables were controlled by exclusion criteria. SPSS version 26.0 was used to analyzed the research data. The quantitative variables i.e. age, gestational age, platelet count and plateletcrit were presented as mean ± SD. The qualitative variables i.e. parity was presented as frequency and percentage. Data were stratified for age to address the confounding variable. After stratification Independent sample t-test was applied. P-value was considered as statistically significant if ≤0.05.

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RESULTS

In Group A (controls) the mean age of patients was 29.4 ± 3.55 years, (range 22-35 years) in comparison to 26.43 ± 4.82 years (range 20-35 years) in group B (cases with GDM). The mean gestational age in Group A was 25.8 ± 1.41 weeks (range 24-28 weeks) as compared to 25.82 ± 1.52 weeks (range 24-28 weeks) in group B. Group A included 56% primigravida and 44% multigravida compared to 60% primigravida and 40% multigravida in Group B. The mean platelet count was found to be 359.31 ± 27.12 (range of 398-313 × 109/L) in Group A, which was higher than mean platelet count in Group B 225.09 ± 49.51 (range 308-146 × 109/L) (P value 0.000). The mean plateletcrit value was significantly higher in Group B pregnant women with GDM (mean ± SD: 0.24 ± 0.08) as compared to Group A non-GDM pregnant women (mean ± SD: 0.14 ± 0.03) (P value=0.000) (Table 1).

Table 1: Comparison of Clinical Factors, Platelet Count and Plateletcrit % Among Groups (n=50)

Variables	Categories	Group A (Controls) Frequency (%) /Mean ± SD	Group B (Cases with GDM) Frequency (%) /Mean ± SD	p-Value
Age (Years)	Mean ± SD	29.4 ± 3.55	26.43 ± 4.82	0.002
	Max	35	35	
	Min	22	20	
Parity	Primigravida	28 (56%)	30 (60%)	0.039
	Multigravida	22 (44%)	20 (40%)	
Gestational Age (Weeks)	Mean ± SD	25.8 ± 1.41	25.82 ± 1.52	0.916
	Max	28	28	
	Min	24	24	
Plateletcrit (%)	Mean ± SD	0.14 ± 0.03	0.24 ± 0.08	0.000
	Max	0.21	0.6	
	Min	0.06	0.18	
Platelet Count × 109/L	Mean ± SD	359.31 ± 27.12	225.09 ± 49.51	0.000
	Max	398	308	
	Min	313	146	

In figure 1, plateletcrit (%) was compared among groups to assess variations in platelet mass, reflecting potential differences in hematological responses. Statistical analysis determined significant differences across study groups.

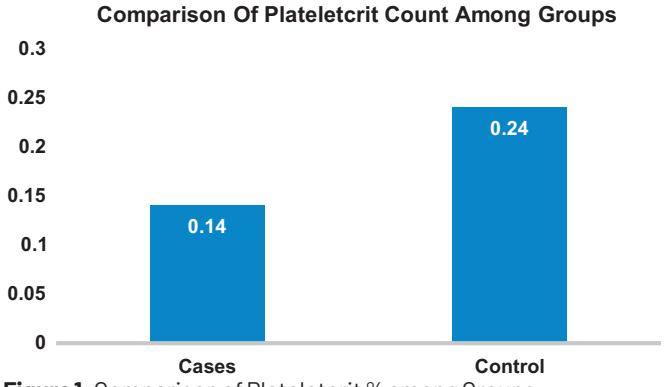

Figure 1: Comparison of Plateletcrit % among Groups

Figure 2 illustrated the comparison of platelet count among groups, highlighted variations in platelet levels. Statistical analysis was performed to assess significant differences across the study groups.

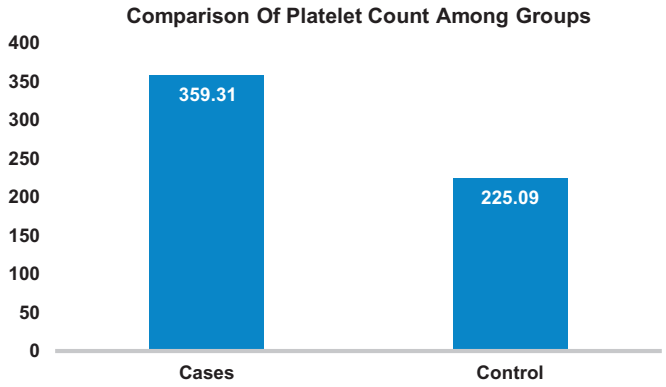

Figure 2: Comparison of Platelet Count Among Groups

The mean plateletcrit value for the <30 group is 0.199 ± 0.062, while for the >30 group, it is 0.217 ± 0.108. Similarly, the mean platelet count is 274.095 ± 68.865 for the <30 group and 257.902 ± 84.624 for the >30 group. In both cases, the p-values 0.266 for plateletcrit and 0.287 for platelet count indicated no statistically significant differences between the two age groups (Table 2).

Table 2: Effect of Age on Plateletcrit and Platelet count

Variables	Age	Mean ± S.D	p-Value
Plateletcrit	<30	0.199 ± 0.062	0.266
	>30	0.217 ± 0.108	
Platelet Count	<30	274.095 ± 68.865	0.287
	>30	257.902 ± 84.624	

DISCUSSION

The prevalence of Gestational Diabetes Mellitus (GDM), a common obstetrical complication, is reported to be 9.3% to 25.5% of pregnancies, depending on the used diagnostic criteria and ethnicity of population [12]. In Pakistan the reported prevalence of GDM in a metanalysis ranged between 8.42% and 35.80%, with overall pooled estimate of 16.7% [13]. Gestational Diabetes Mellitus (GDM) is a chronic inflammatory condition. Numerous factors including hyperlipidemia, insulin resistance, hyperglycemia, inflammatory process, endothelial dysfunction and oxidative stress lead to platelet activation. Platelet interaction with endothelium by producing Reactive Oxygen Species (ROS) causes initiation of endothelial dysfunction in GDM [14]. Endothelial dysfunction characterized by altered balance of vasodilators, such as nitric oxide and vasoconstrictors lead to impaired vascular response. Endothelial dysfunction causes pro-inflammatory and pro-coagulatory vascular environment favoring thrombus formation [15]. Platelet

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reactivity and aggregation is caused by Reactive Oxygen Species (ROS). Due to this there is accumulation of Advanced Glycation End products (AGEs) in plasma that interact with specific receptors on the endothelium to cause endothelial dysfunction. This leads to reduction in the production of Nitric Oxide (NO) and PGI2. All these mechanisms are suggested to contribute to complications of GDM [16]. Activated platelets have larger MPV and higher plateletcrit, which may cause hypercoagulability in the placental bed leading to vascular events. These events can be responsible for feto-maternal complications in GDM patients. Platelet activation may be detected by platelet parameters like MPV, platelet count, plateletcrit and PDW [17]. Amongst these, MPV and plateletcrit are emerging as a potential predictor or a cheap, easily available, screening tool for early detection of GDM in numerous studies. Contradictory results have been reported on the positive association and potential screening role of platelet indices. In current study the association of plateletcrit was determined with GDM. Current GDM pregnant patients were younger than healthy pregnant patients. There was also significant difference in parity between both groups. This is a limitation of study. As ideally both groups should have similar age and parity. Gestational age was similar in both groups. Platelet count was found to be significantly lower in GDM patients as compared to healthy pregnant controls (p value=0.000). This finding is consistent with results of Khan and Ashraf, [8]. Fashami MA *et al.*, reported higher count in GDM pregnant patients 233.0 ± 62.6 compared to 193.3 ± 49.5 [2]. Similarly, Xiang LL *et al.*, also reported higher platelet count in GDM group 221.27 ± 43.04 vs 218.95 ± 45.27 [18]. Simsek and Altekin, and Baldane S *et al.*, found no difference in platelet count between patients with GDM and healthy pregnant women [19, 20]. Main outcome of current study parameter was plateletcrit, which was significantly higher in patients with GDM as compared to healthy controls. However, in another study reported results were consistent with current study, reporting lower platelet count and higher plateletcrit and MPV in women with GDM [20]. Liu G *et al.*, observed contradictory findings reporting no difference in platelet count, MPV and plateletcrit between GDM and normal pregnancy groups [21]. The size of platelet is related to platelet activity and is reflected by MPV, plateletcrit and PWD. Chronic low-grade inflammation in GDM leads to platelet activation and ultimately change in platelet indices. GDM develops when pancreatic function changes due to placental hormones during the second trimester may surpass the body's coping mechanisms. A study on platelet indices in healthy pregnancies and GDM found no difference in inflammatory markers during the first trimester. However, there was an inverse relationship between increasing MPV values and the likelihood of GDM. The analysis showed that while MPV has association with GDM, it is not sufficient as a standalone diagnostic marker [21].

CONCLUSIONS

Pregnant patients with GDM had higher mean plateletcrit values and lower platelet count as compared to non GDM pregnant patients indicating their potential role in its pathophysiology and prediction. Future prospective cohort studies of changes in inflammatory factors and platelet indices during first trimester followed-up to the end of pregnancy are suggested to establish its role as an early screening test to diagnose gestational diabetes mellitus. This study's single-center design, small sample size and retrospective nature may limit the generalizability of its findings.

Authors Contribution

Conceptualization: SC
Methodology: SC, RK, EF
Formal analysis: SC, ZK
Writing, review and editing: SC, ZK

All authors have read and agreed to the published version of the manuscript

Conflicts of Interest

The authors declare no conflict of interest.

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