

Is there a role for platelet indices in predicting poor glucoregulation in type 2 diabetes mellitus?

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Abstract

In this editorial, we discuss the recent article by Regassa *et al*, published in the *World Journal of Diabetes*, which highlights the potential role of platelet indices (PI) in predicting poor glucoregulation in patients with type 2 diabetes mellitus (T2DM). Given the high morbidity and mortality associated with T2DM, there is a constant need to find new and accessible methods for predicting and treating individuals with this condition. The pathophysiology of T2DM involves systemic inflammation, metabolic dysfunction, and an increased risk of vascular injury, which are commonly associated with the development of microvascular and macrovascular complications, such as cardiovascular diseases and neuropathies. The link between these complications and T2DM requires further elucidation but may be explained by prolonged exposure to high glycemic levels and increased advanced glycation end products. PI might play an important role in determining whether some individuals are prone to poor glucoregulation. Recent evidence encourages the scientific efforts to demonstrate the consistency of this role and its applicability in monitoring glucoregulation, underscoring the importance of the study by Regassa *et al*.

Key Words: Platelet indices; Platelet count; Type 2 diabetes mellitus; Adult; Glycemic Control; Diabetes complications; Inflammation mediators

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Core Tip: This editorial discusses the role of platelet indices (PI) such as platelet count, mean platelet volume, plateletcrit, platelet large cell ratio, and platelet distribution width as potential predictors of poor gluoregulation in type 2 diabetes mellitus (T2DM). Emerging evidence shows that elevated PI levels correlate with inadequate gluoregulation and microvascular complications. Incorporating PI into routine assessments may offer a cost-effective tool for predicting glycemic outcomes in patients with T2DM, especially in resource-limited settings.

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INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a chronic metabolic condition that affects millions of people globally and is associated with several long-term complications, including cardiovascular diseases. Inadequate gluoregulation is a critical factor contributing to these complications and is associated with high morbidity and mortality[1]. Attention has turned to lower-cost and technically easier alternatives that can predict the effectiveness of gluoregulation in people living with T2DM. Among these, platelet indices (PI) have emerged as potential indicators due to their relationship with inflammatory and metabolic processes[2].

The applicability of this concept was assessed in the study published by Regassa *et al*[3] in the *World Journal of Diabetes*, which examined the use of PI as prognostic predictors of suboptimal gluoregulation among adult subjects with T2DM. It revealed that increased PI are significantly elevated in individuals with poor gluoregulation in T2DM, indicated by fasting blood glucose (FBG) levels exceeding 130 mg/dL, when comparing those with adequate gluoregulation and healthy controls.

Moreover, it was highlighted that these indices could also correlate positively with anthropometric variables, including body mass index (BMI), waist circumference (WC), and waist-to-hip ratio. Additional correlated variables include systolic blood pressure, diastolic blood pressure, FBG, duration of T2DM, and the occurrence of microvascular complications. This editorial briefly discusses the role of PI in predicting poor gluoregulation and the prognosis of subjects with T2DM [3].

PIS AND GLUCOREGULATION

PI, such as platelet count, mean platelet volume (MPV), platelet large cell ratio, plateletcrit (PCT), and amplitude of platelet distribution, also known as platelet distribution width (PDW), have been investigated as predictors of gluoregulation in T2DM. Studies suggest that high PI levels are associated with poorer gluoregulation, indicated by high glycated hemoglobin values (HbA1c), and microvascular and macrovascular complications. For example, Citirik *et al*[4] evaluated the potential of MPV, PDW, and PCT as parameters for detecting subclinical platelet activation in diabetic retinopathy. Demirtas *et al*[5] demonstrated that increased MPV is significantly associated with elevated HbA1c levels, reinforcing the hypothesis that chronic inflammation, commonly found in T2DM, can alter platelet function and quantity.

BIOLOGICAL PATHWAYS

Increasing evidence suggests that chronic inflammation and insulin resistance play a crucial role in modulating platelet function in T2DM[6,7]. Elevated levels of pro-inflammatory cytokines, such as TNF- α and IL-6, contribute to platelet activation by enhancing NF- κ B signaling and upregulating adhesion molecules like P-selectin, thereby fostering a pro-thrombotic state. Additionally, insulin resistance disrupts the PI3K/Akt signaling pathway, which is essential for both glucose metabolism and platelet regulation. Under normal conditions, insulin promotes nitric oxide production through this pathway, reducing platelet reactivity; however, in the presence of insulin resistance, PI3K/Akt dysfunction shifts signaling toward the MAPK pathway, thereby exacerbating platelet hyperactivity and damaging the glycocalyx, which leads to endothelial dysfunction[8]. This process is further aggravated by the production of reactive oxygen species (ROS) and by impaired IRS-1/2 signaling. ROS directly degrades the endothelial glycocalyx, promoting vascular inflammation and endothelial dysfunction[8]. These effects indirectly perpetuate an inflammatory and thrombotic cycle, potentially contributing to platelet activation and exacerbating vascular injury[8,9]. As a result, PI, such as MPV and PDW, are increasingly recognized as potential biomarkers of metabolic imbalance in T2DM, reflecting the heightened thrombotic risk associated with this disease[6,7].

Novel studies also associate the gut microbiome with T2DM, and PI might serve as a marker of this interaction by reflecting metabolic dysregulation and systemic inflammation. Changes in gut microbial composition can influence platelet activation through microbial metabolites, such as short-chain fatty acids and lipopolysaccharides, which

modulate immune responses and insulin sensitivity. These mechanisms may explain the pro-thrombotic state observed in T2DM-linking gut dysbiosis, PI, and disease progression[10]; however, much remains to be explored.

PI are simple, low-cost, and widely available in routine blood tests. Growing evidence suggests that increased MPV and changes in PDW are associated with an exacerbated inflammatory response, which may negatively influence gluco-regulation in patients with T2DM. MPV is an independent risk factor for macrovascular complications such as acute myocardial infarction, coronary artery disease[11,12], peripheral artery disease[13], and cerebral ischemia[14,15]. It has also been widely studied as an independent marker of endothelial dysfunction and altered microcirculation, which are central mechanisms in the development of microvascular complications, including erectile dysfunction[16,17].

FINDINGS FROM RECENT STUDIES

Studies across different populations have provided valuable data on how PI can predict gluco-regulation in T2DM, with the most recent evidence emerging from India. Chawla *et al*[18] evaluated MPV, PDW, and PCT as predictive biomarkers of microvascular complications in Indian patients with T2DM. The results indicated that HbA1c levels were significantly correlated with elevated MPV, PDW, and PCT levels. Thus, PI may serve as predictive biomarkers of microvascular complications in T2DM, such as neuropathy and retinopathy. Khanna *et al*[19] reported similar findings, stating that MPV, PDW, and P-LCR were significantly higher in patients with T2DM who had microvascular complications when compared with patients without complications in India. Nimmala *et al*[20] observed that MPV was significantly higher in subjects with T2DM compared to controls. MPV was associated with microvascular complications and positively correlated with age and T2DM duration.

The use of HbA1c as a predictive biomarker of poor gluco-regulation is widely accepted[21,22]. Schoos *et al*[23] investigated the impact of HbA1c levels on residual platelet reactivity and outcomes following the insertion of coronary drug-eluting stents in subjects with T2DM. They found that those with poor gluco-regulation, indicated by HbA1c > 8.5%, had a higher risk of stent thrombosis and cardiac death after percutaneous coronary intervention. A positive association was also found between HbA1c and high platelet reactivity, suggesting a potential area of research regarding pro-inflammatory interventions and PI in subjects with T2DM and poor gluco-regulation.

Despite its widespread use, HbA1c has several limitations, including cost and the requirement for specialized laboratory equipment capable of performing high-performance liquid chromatography or immunoassay-based methods for accurate measurement[4]. In contrast, PI are derived from routine blood tests through an automated blood count, making them a more accessible and cost-effective option for monitoring gluco-regulation in these settings[4]. This advantage highlights the potential of PI as a complementary tool for monitoring gluco-regulation, especially in contexts where HbA1c testing is not readily accessible.

Therefore, PI are emerging as promising additional tools for monitoring and managing T2DM, and as preliminary screening measures to identify individuals at risk for diabetes-related complications, thereby prompting early intervention, especially in resource-limited settings[24].

CONSIDERING THE LIMITATIONS OF THE STUDY

The study by Regassa *et al*[3] exhibits significant heterogeneity due to variability in population characteristics, clinical parameters, laboratory measurements, and treatment regimens. Differences in disease duration, BMI, WC, and the presence of microvascular complications contribute to inconsistencies, while variations in laboratory techniques and the use of different analyzers (CobasC311 and Sysmex XN550) may affect PI results. Furthermore, the inclusion of patients on different antidiabetic treatments, without controlling for antiplatelet medication use, further complicates the interpretation of the findings. The cross-sectional design limits causal inferences, and the relatively small sample size reduces generalizability beyond the Ethiopian population. This heterogeneity compromises reliability by introducing potential confounders that influence platelet function without adjustment, making it difficult to determine whether altered PI are a cause or a consequence of poor gluco-regulation. To minimize heterogeneity in future research, studies should stratify patients by disease severity and treatment, adopt prospective designs to establish causality, control for confounding metabolic and inflammatory factors, and employ standardized laboratory techniques to ensure consistency. Expanding sample sizes, conducting multicenter studies across diverse populations, and incorporating more advanced platelet function tests may further enhance the validity and applicability of these findings.

CLINICAL IMPLICATIONS AND FUTURE DIRECTIONS

Integrating the assessment of PI into clinical practice may offer a practical and cost-effective method for identifying individuals with poor gluco-regulation. However, it is essential that further studies, particularly those of a long-term, longitudinal, and multicenter nature, should be conducted to confirm these findings and validate the use of PI as reliable predictors of gluco-regulation in diverse T2DM populations. Exploring their application is strongly recommended to enhance clinical diagnostic accuracy and prognostic assessment across various pathological states, particularly in inflammatory and thrombotic conditions.

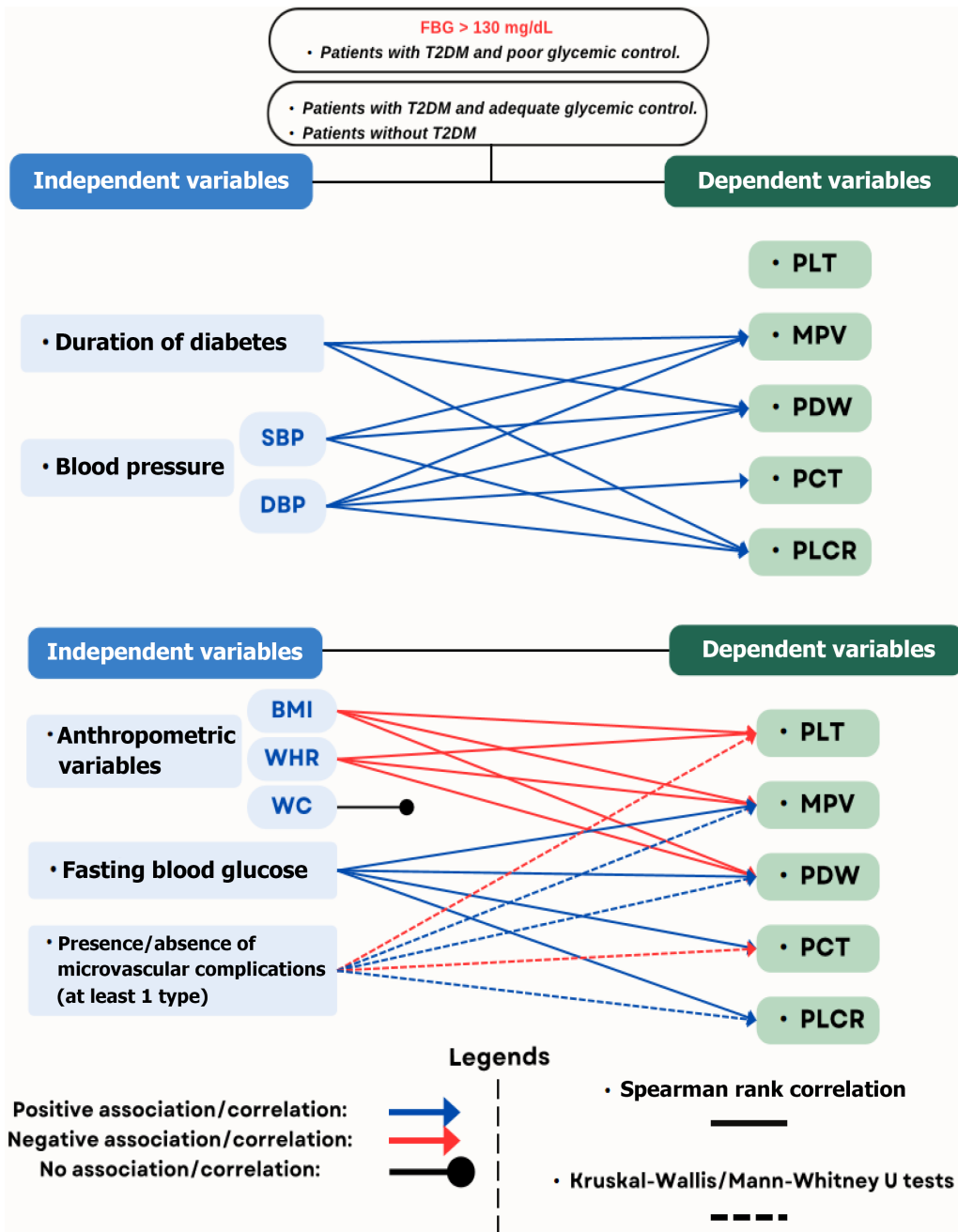


Figure 1 Relationship between dependent and independent variables is included in the study by Regassa *et al*[3]. Solid blue line represents a positive correlation between variables; Solid red line represents a negative correlation between variables; Blue dashed line indicates a positive association between variables; Red dashed line indicates a negative association between variables. BMI: Body mass index; DBP: Diastolic blood pressure; FBG: Fasting blood glucose; MPV: Mean platelet volume; PCT: Plateletcrit; PDW: Platelet distribution width; PLCR: Platelet large cell ratio; PLT: Platelet count; SBP: Systolic blood pressure; T2DM: Type 2 diabetes mellitus; WC: Waist circumference; WHR: Waist-hip ratio.

Relevant research questions, such as “What is the longitudinal relationship between PIs and glucoregulation in T2DM?” and “How do PIs compare with traditional biomarkers of glucoregulation in terms of sensitivity and specificity?” could be addressed through cross-sectional and case-control studies. Meanwhile, the question “Is there a causal relationship between PIs and poor glucoregulation?” could be investigated using experimental research designs.

For example, metabolomic studies, such as the one conducted by Jin and Ma[25], offered new insights into the mechanisms underlying glucoregulation in kidney and cardiovascular disease among individuals with T2DM. Similar studies could complement the interpretation of PI in T2DM, including comparisons with HbA1c levels.

The flowchart in Figure 1 illustrates the interaction between the dependent and independent variables of the study by Regassa *et al*[3].

CONCLUSION

In the study published in this issue, Regassa *et al*[3] found an association between PI measurements and gluco-regulation in T2DM, suggesting the potential utility of these markers in T2DM follow-up. However, the association between PI and more accurate indicators of gluco-regulation, such as HbA1c, remains limited. Although promising, these indices should be interpreted with caution, and their association with other markers and clinical outcomes should be clarified. Advances in the understanding of PI in T2DM may yield new tools to improve disease management and potentially reduce both costs and the risk of diabetes-related complications.

FOOTNOTES

Author contributions: Medeiros GA and de Santana JBF equally conceptualized and drafted the first draft of the editorial; Medeiros GA, de Santana JBF, Negrato CA and Zajdenverg L contributed to the discussion and conception of the manuscript; Medeiros GA and de Santana JBF equally contributed to writing and editing the final draft of the manuscript; All the authors critically revised and approved the final version of the manuscript.

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