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Association Between Insulin Resistance and Hematopoietic Stem Cell Functions in Patients with Type 2 Diabetes Mellitus

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ABSTRACT

Background: Intestinal obstruction is a common surgical emergency with significant morbidity and mortality, but postoperative adhesions are its most frequent cause. Patients with a virgin abdomen, those without any prior abdominal surgery, pose diagnostic challenges; this study explored the etiological spectrum, clinical features, and outcomes of such cases in a tertiary care hospital in Pakistan.

Methods: A six-month prospective observational study at Lady Reading Hospital, Peshawar, enrolled 73 eligible adult patients, categorizing them into Group 1 (surgical management for mechanical causes such as hernias, tumours, or volvulus) and Group 2 (conservative management for functional or self-resolving causes). Data collected encompassed demographics, presenting symptoms, vital signs, symptom duration, and etiological factors, supplemented by relevant laboratory investigations and imaging studies. Statistical analysis was performed using SPSS version 26, with continuous variables summarized as mean ± standard deviation and compared using the independent t-test, categorical variables presented as frequencies and percentages, and analysed using the Chi-square or Fisher's exact test, and a p-value of <0.05 was considered statistically significant.

Results: The mean age of Group 1 was significantly higher (55.73 ± 4.86 years) than that of Group 2 (44.13 ± 6.32 years, $p < 0.001$). Tumours were the most common cause (24.7%), followed by hernias (20.5%) and volvulus (13.7%). Group 1 had longer hospital stays (12.45 ± 2.94 days vs. 7.76 ± 2.03 days, $p < 0.001$) and higher clinical severity. Complications and mortality rates were low and comparable between groups, with effective management in both cohorts.

Conclusion: The cause of intestinal obstruction differs in patients with a virgin abdomen, which has a vast clinical impact. It demands early diagnosis and proper treatment plans to enhance the quality of life and reduce mortality. Further investigations of patient follow-up with a focus on discharge and ways of minimizing the number of days patients spend in the hospital are important.

Keywords: Intestinal Obstruction, Abdomen, Postoperative Complications, Hernia, Intestinal Neoplasms, Volvulus, Paralytic Ileus, Prospective Studies, Treatment Outcome, Pakistan.

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INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder characterized by insulin resistance and lower secretion, affecting millions worldwide. As T2DM progresses, hyperglycemia results, leading to systemic complications like cardiovascular disease, nephropathy, retinopathy, and neuropathy[1,2]. Disruptions in biological processes like inflammation, oxidative stress, and endothelial dysfunction contribute to the disease. Recent studies suggest that impaired bone marrow stem cells (HSCs) may be linked to insulin resistance and contribute to T2DM complications[3,4]. HSCs are essential for blood and immune cell production, but metabolic disorders like insulin resistance can impair their functionality, leading to attenuated immune responses, delayed wound healing, and increased susceptibility to infections and inflammation[5].

Chronic inflammation from metabolic disturbances affects the bone marrow microenvironment, which is crucial for hematopoietic stem cells (HSCs). Insulin resistance can disrupt the organization of the bone marrow niche, affecting HSCs' self-renewal and differentiation capabilities[6]. This leads to hematopoiesis and immune responses in T2DM patients. Inflammatory signaling pathways, such as NF-κB activation, result in pro-inflammatory cytokines like TNF-α and IL-6, leading to HSC exhaustion and skewed differentiation towards myeloid lineages. Recent research has focused on the relationship between metabolic disorders and stem cell behavior, particularly the impact of insulin resistance on HSCs[7,8]. Studies indicate that reduced insulin signaling disrupts the quiescent state of HSCs and hinders their self-renewal capabilities, leading to increased cellular stress and diminished regenerative potential. Alterations in insulin signaling within HSCs can lead to a preference for myeloid differentiation over lymphoid differentiation, resulting in an imbalance in immune cell production [9].

Insulin resistance affects human stem cells in type 2 diabetes, but its cause is unclear. Further research is needed to understand its long-term impact on HSC function and potential therapeutic interventions. The study investigates insulin resistance's impact on hematopoietic stem cell function in type 2 diabetes patients, examining changes in bone marrow microenvironment and inflammatory signaling pathways. It seeks interventions to restore HSC function and improve patient outcomes.

METHODS

This six-month prospective observational study at Lady Reading Hospital, Peshawar, with 230 patients diagnosed with Type 2 Diabetes Mellitus (T2DM) who were enrolled to evaluate the relationship of IR with HSC function. The study was conducted after approval from institute's ERC under reference #321/KSC/2024. The demographic details of the patients as age, gender, weight, height, BMI, and ethnicity on admission were elicited. Clinical parameters like HbA1c, fasting glucose, insulin levels, and the index of insulin resistance (HOMA-IR) were noted. These also included the analysis of blood pressure, lipid profile (cholesterol, LDL, HDL), triglycerides, and complete blood count. Significance was defined as $p<0.05$. All participants provided their informed, written consent, and this study was approved by the Institutional Review Board.

The data was entered and analyzed using SPSS v27.0 to assess the relationship between insulin resistance and hematopoietic stem cell functions in individuals with type 2 diabetes mellitus. The study utilized both descriptive and inferential statistics to explore this association. Descriptive statistics offered valuable information regarding demographic factors such as age, gender, and BMI, while the Insulin Resistance Index was utilized to quantify insulin resistance. Nonparametric tests, including Chi-Square and Independent-Samples Median Tests, were applied to evaluate categorical variables such as comorbidities and medications. A significance threshold of $p < 0.05$ was established to determine statistical differences among the groups.

RESULTS

The objective of the present study was to find the link of insulin resistance and the function of HSCs in patients suffering from T2DM. Several physiological and biochemical parameters of interest were age, weight, BMI, serum insulin, index of insulin resistance, and several others, including hematological markers. Relationships between some of these parameters are presented in the following figures as discussed below.

Table 1. Demographic Data and Clinical Characteristics of The Study Population.

Demographic and Clinical Characteristics	Mean (± SD)
Age (years)	54.38 (± 14.54)
Weight (kg)	85.26 (± 21.21)
BMI (kg/m²)	28.69 (± 6.28)
Duration of T2DM (years)	9.30 (± 5.40)
HbA1c (%)	8.76 (± 1.89)
Fasting Glucose Level (mg/dL)	125.09 (± 31.57)

Table 1 Summarizes the demographic data and clinical characteristics of the study population. The mean age of the patients was 54.38 years, with a standard deviation of 14.543. The mean weight and BMI were 85.26 kg and 28.69 kg/m², respectively. Most participants belonged to the Balochi ethnic group (22.2%), followed by Punjabi (21.7%) and Sindhi (20.4%) groups. Samples were spread evenly between persons of different educational levels, though 31.3% of them had higher education and 35.2% had primary-level education.

Table. 2. Average Insulin Levels, Insulin Resistance, and Blood Pressure Levels of The Patients.

Metabolic Parameters	Mean (± SD)
Insulin Level (µU/mL)	20.55 (± 10.87)
Insulin Resistance Index	8.03 (± 4.09)
Blood Pressure (mmHg)	229.07 (± 44.20)
Cholesterol (mg/dL)	229.07 (± 44.20)
LDL (mg/dL)	121.57 (± 42.36)
HDL (mg/dL)	55.72 (± 14.10)

Table 2. Data regarding insulin levels, insulin resistance assessed by the index of insulin resistance, and blood pressure levels in relation to the study population. The mean level of insulin was 20.55 µU/mL, and the mean level of the insulin resistance index was 8.03. Regarding the results for blood pressure, hypertensive values were demonstrated by 33.0% of participants, while normotensive values were determined for 38.3%.

Table. 3. Hematological Parameters.

Hematological Parameters	Mean (± SD)
White Blood Cell Count (10^3/µL)	7.56 (± 2.01)
Red Blood Cell Count (10^6/µL)	4.95 (± 0.58)
Platelet Count (10^3/µL)	270.76 (± 69.83)

Table 3 shows the hematological parameters assessed in the study group. Some of these hematological parameters are represented in. All the WBC, RBC, and platelet counts seemed to lie in the normal range. Mean

count of WBC and RBC was documented at 7.56 and 4.95, respectively. The mean count of platelet was documented at 270.76 x 10^3 cells/µL.

Table 4. Association Between Insulin Resistance and Hematological Parameters.

Association with HSC Function	p-value
Insulin Resistance & WBC Count	0.362
Insulin Resistance & RBC Count	0.509
Insulin Resistance & Platelet Count	0.159

Non-parametric tests were conducted to determine the association of insulin resistance with HSC function. For this purpose, **Table 4** was considered, which shows that the value of p is greater than 0.05 for hematological indices like WBC, RBC, and PLT. A trend has been observed without being significant; in other words, an inverse relationship has been found, which doesn't hold significance for PLT and insulin resistance.

Table 5. Comorbidities and Medications Recorded.

Comorbidities	N (%)
Hypertension	50 (21.7%)
Cardiovascular Disease	42 (18.3%)
Kidney Disease	49 (21.3%)
Obesity	44 (19.1%)
Medications	**N (%)**
Sulfonylureas	75 (32.6%)

Insulin	54 (23.5%)
Metformin	43 (18.7%)
SGLT2 Inhibitors	58 (25.2%)

Table 5 shows comorbidities and medications reported by the participants. The most common comorbidity was hypertension, with a prevalence of 21.7% in the study population. Cardiovascular diseases and kidney disease prevailed in 18.3% and 21.3%, respectively. For pharmacotherapy, the most represented group was on sulfonylureas at 32.6%, followed by 23.5% on insulin and 18.7% on metformin.

Table 6. The Relationship Between The Duration of T2dm and The Type of Treatment Used.

Treatment History	N (%)
Diet Control	66 (28.7%)
Insulin Therapy	94 (40.9%)
Oral Medications	70 (30.4%)
Previous Insulin Treatment	105 (45.7%)

Table 6 shows the relationship between the duration of T2DM and the type of treatment used. The duration of diabetes varied significantly among participants, with a mean duration of 9.30 years. Interestingly, 40.9% of the participants had a history of insulin therapy, while 28.7% managed their condition through diet control alone. Additionally, 54.3% of the participants had no prior insulin treatment, indicating a variation in treatment history depending on the disease progression.

Analysis of the association between insulin resistance and hematopoietic parameters was carried out through conducting the nonparametric tests. Table 4 reports that there is no significant difference across samples along the Type 2 Diabetes Duration ($p > 0.05$). Independent samples' median test also reported having no such significant association between insulin resistance and WBC or RBC counts. However, there is a developing trend towards lower platelet count with higher insulin resistance. Besides, there was no significant correlation of insulin resistance with the values of BMI, HbA1c, fasting glucose levels, or lipid profiles ($p > 0.05$). Results bring into light that although insulin resistance is a prime trait of the metabolic regulation process in T2DM

patients, the impact on HSC function may not be direct, at least in the sample and parameters measured under this research. Given the fact that BMI, HbA1c, and fasting glucose levels or lipid profiles are used as diagnostic factors for Diabetes Mellitus Type-2.

DISCUSSION

This study aimed to explore the relationship between insulin resistance (IR) and hematopoietic stem cell (HSC) function in patients diagnosed with Type 2 Diabetes Mellitus (T2DM). The findings of this research provide a detailed examination of the biochemical, physiological, and hematological parameters of 230 individuals, analyzing how these factors interact with insulin resistance and what implications they may have for HSC function. While several studies have previously examined insulin resistance in the context of metabolic and cardiovascular diseases, fewer have focused on the direct impact of insulin resistance on HSCs, which are crucial for hematopoiesis and immune function.

Insulin resistance is a central feature of T2DM, characterized by the body's reduced ability to respond to insulin, leading to elevated blood glucose levels. The metabolic implications of this include hyperinsulinemia, dyslipidemia, and increased cardiovascular risk. In this study, we observed that the average insulin resistance index (HOMA-IR) was 8.03, a value that indicates significant insulin resistance within the study population. This is consistent with the established literature, which positions insulin resistance as a hallmark of T2DM and an important factor in its progression. Insulin resistance impacts various metabolic pathways, including lipid metabolism and inflammation, both of which are known to contribute to the dysfunction of HSCs. Previous studies have suggested that the chronic low-grade inflammation characteristic of T2DM can influence HSC activity, leading to impaired hematopoiesis and altered immune responses. However, the results of this study did not indicate a significant association between insulin resistance and major hematological parameters, such as white blood cell (WBC), red blood cell (RBC), or platelet counts. Hematopoietic stem cells play a critical role in the maintenance of the blood and immune systems. Dysfunctional HSCs can lead to inadequate immune responses, anemia, or thrombocytopenia. In the context of diabetes, emerging evidence suggests that hyperglycemia and associated metabolic derangements may influence HSC function. Several mechanisms have been proposed for this interaction, including oxidative stress, inflammation, and altered growth factor signaling, which can impair HSC proliferation and differentiation.

Despite these theoretical underpinnings, the current study found no significant associations between insulin resistance and traditional hematological markers, such as WBC, RBC, or platelet counts. The lack of statistical significance ($p > 0.05$) in these associations could be due to the complex regulatory mechanisms governing HSC function, which may not be directly influenced by insulin resistance in a linear manner. However, a non-significant trend towards an inverse relationship between insulin resistance and platelet count was observed [10]. Although this observation did not meet the threshold for statistical significance, it suggests that further investigation into the relationship between insulin resistance and thrombopoiesis may be warranted.

One potential explanation for the absence of a significant relationship between insulin resistance and HSC function in this study is the heterogeneity of the diabetic population. Patients with T2DM often present with varying degrees of disease severity, comorbidities, and treatment histories, all of which can influence insulin resistance and its metabolic effects[11]. Furthermore, the duration of diabetes in this study population averaged 9.3 years, with a range of treatment strategies employed, including diet control, insulin therapy, and oral hypoglycemic agents. It is possible that these variations in disease duration and treatment could have masked more subtle associations between insulin resistance and HSC dysfunction [12,13,14]

Additionally, the study population presented with a range of HbA1c levels (mean: 8.76%), fasting glucose levels (mean: 125.09 mg/dL), and BMI values (mean: 28.69 kg/m²), indicating that these individuals were at varying stages of metabolic control . The lack of significant correlations between insulin resistance and hematological parameters could therefore be reflective of the fact that insulin resistance, while a key player in the pathophysiology of T2DM, may not exert a direct influence on HSC function in the absence of other contributing factors, such as chronic inflammation or oxidative stress, which were not directly measured in this study[15].

The findings of this study have important implications for the clinical management of patients with T2DM [16,17] Although insulin resistance is clearly linked to many adverse metabolic outcomes, its role in directly impairing hematopoietic stem cell function may be less pronounced than initially hypothesized [18,19] This suggests that while managing insulin resistance remains critical for controlling hyperglycemia and reducing cardiovascular risk in patients with T2DM, additional factors may need to be considered when addressing hematopoietic and immune system dysfunctions in these patients [16,18].

In clinical practice, this may mean that interventions aimed at improving HSC function, such as anti-inflammatory therapies or treatments targeting oxidative stress, could be more effective than focusing solely on insulin resistance [19,20] Moreover, the observed trend between insulin resistance and platelet count, although not statistically significant, raises the possibility that platelet function and production could be a more sensitive marker of HSC dysfunction in the context of T2DM [21]. Further research into this area could help to clarify the potential role of thrombopoiesis as a biomarker for stem cell dysfunction in insulin-resistant patients [22,23,24]

It is important to acknowledge the limitations of this study. First, the cross-sectional design limits the ability to infer causality between insulin resistance and HSC function. Longitudinal studies are needed to better understand the temporal relationship between these factors and to determine whether changes in insulin resistance over time lead to measurable changes in HSC function. Second, the study population was drawn from a single geographic region, limiting the generalizability of the findings to other populations with different ethnic and genetic backgrounds. Additionally, while this study focused on traditional hematological markers, more specific assays of HSC function, such as colony-forming unit assays or flow cytometric analysis of HSC subsets, were not performed. These assays may provide a more nuanced understanding of how insulin resistance affects HSC activity at a cellular level.

CONCLUSION

The study assessed the relation of IR with HSC function among the patients with T2DM. While conducting extensive clinical parameters and looking into multiple differences in patients regarding their insulin resistance, hematological markers, and demographic factors, it failed to find any significant correlation of IR with HSC function. In summary, the present findings suggest that insulin resistance, a critical feature of metabolic dysregulation in T2DM, would play little or even no role in the function of HSCs. Concerning insulin resistance, only a very weak inverse, non-significant trend was observed between insulin resistance and platelet count, representing an interesting direction for further study. Generally, these results mirror the complexity of T2DM pathology and indicate a need for conducting research in larger and more heterogenous populations to determine if and how insulin resistance might functionally be related to hematopoiesis. These findings justify further studying the long-term impact of insulin resistance on hematopoietic health in diabetics.

LIST OF ABBREVIATIONS

None

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None

CONFLICT OF INTEREST

None

ETHICAL APPROVAL

This study was approved by the Institutional Review Board (IRB)

AUTHORS' CONTRIBUTION

None

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