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Original Article

Assessment of Renal and Hematological Parameters in Patients with Type 2 Diabetes Mellitus: A Cross-Sectional Observational Study

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ABSTRACT

Background: Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder associated with progressive renal dysfunction and hematological abnormalities that contribute to increased morbidity and mortality. This study aimed to evaluate renal and hematological parameters among patients with T2DM.

Methods: A hospital-based cross-sectional observational study was conducted at A.C.S. Medical College and Hospital, Chennai, involving 30 participants, including 15 patients with T2DM and 15 healthy controls. Serum urea and creatinine were analyzed using the Beckman Coulter AU480 analyzer, while hematological parameters were measured using the Sysmex XN-1000 automated hematology analyzer. Statistical analysis was performed using the independent *t*-test, with $p<0.05$ considered statistically significant.

Results: Diabetic patients showed higher mean serum urea (37.80 ± 23.34 mg/dL) and serum creatinine (1.63 ± 1.91 mg/dL) than controls, although the differences were not statistically significant ($p>0.05$). Hemoglobin (11.00 ± 1.61 g/dL; $p=0.003$), packed cell volume ($p=0.021$), and mean corpuscular hemoglobin concentration ($p=0.034$) were significantly reduced among diabetic patients.

Conclusion: Patients with T2DM exhibited early renal alterations and significant anemia-related hematological changes. Routine assessment of renal function and complete blood count may facilitate early detection of diabetic complications and improve clinical management.

Keywords: Type 2 diabetes mellitus; renal function; hemoglobin; serum creatinine; serum urea; anemia.

Introduction:

Diabetes mellitus (DM) is one of the most prevalent chronic non-communicable diseases worldwide and represents a major public health challenge owing to its rapidly increasing incidence and associated long-term complications. Type 2 diabetes mellitus (T2DM), characterized by insulin resistance and progressive pancreatic β-cell dysfunction, accounts for approximately 90–95% of all diabetes cases. The disease is associated with persistent hyperglycemia, which progressively damages multiple organ systems, including the kidneys, cardiovascular system, retina, peripheral nerves, and hematopoietic system. The growing prevalence of T2DM has imposed a substantial clinical and economic burden on healthcare systems globally, particularly in low- and middle-income countries where access to early diagnosis and comprehensive disease management remains limited [1,2].

According to the International Diabetes Federation (IDF), more than 537 million adults were living with diabetes in 2021, and this number is expected to increase to 643 million by 2030 and approximately 783 million by 2045 if current trends continue [3]. India has emerged as one of the countries with the highest burden of diabetes, earning the designation of the "diabetes capital" in many epidemiological reports. Urbanization, sedentary lifestyle, obesity, unhealthy dietary habits, and genetic susceptibility have contributed significantly to the increasing prevalence of T2DM among the Indian population [4]. Consequently, the burden of diabetes-related complications, including diabetic nephropathy and anemia, has also risen considerably. Persistent hyperglycemia induces a series of metabolic and biochemical alterations involving oxidative stress, chronic inflammation, activation of the polyol pathway, formation of advanced glycation end products (AGEs), and activation of protein kinase C. These mechanisms collectively promote endothelial dysfunction, glomerular basement membrane thickening, mesangial expansion, and progressive renal fibrosis, ultimately leading to diabetic kidney disease (DKD) [5,6]. Diabetic nephropathy remains one of the leading causes of chronic kidney disease (CKD) and end-stage renal disease worldwide, accounting for a substantial proportion of patients requiring renal replacement therapy [7]. Assessment of renal function is therefore an essential component of diabetes management.

Serum creatinine and blood urea remain among the most commonly used biochemical markers for evaluating renal function in routine clinical practice. Elevated serum creatinine reflects a reduction in glomerular filtration rate, whereas increased blood urea nitrogen indicates impaired renal clearance and altered protein metabolism. Although these conventional biomarkers are relatively inexpensive and widely available, they often become abnormal only after considerable nephron loss has occurred. Therefore, regular monitoring of renal parameters is crucial for the early detection and management of diabetic nephropathy [8,9].

In addition to renal dysfunction, diabetes mellitus is increasingly recognized as a disorder that significantly affects hematological homeostasis. Chronic hyperglycemia alters the structural and functional integrity of blood cells through several mechanisms, including oxidative damage, non-enzymatic glycation of membrane proteins, chronic inflammation, and microvascular injury [10]. These pathological processes reduce erythrocyte deformability, shorten red blood cell survival, impair oxygen transport, and contribute to abnormalities in platelet and leukocyte function [11]. Consequently, hematological abnormalities have been proposed as useful indicators of disease severity and progression in patients with diabetes. Anemia is one of the most frequently encountered hematological abnormalities among patients with diabetes, particularly those with diabetic nephropathy. The development of anemia in diabetes is multifactorial and involves inadequate erythropoietin production by damaged kidneys, chronic systemic inflammation, functional iron deficiency, nutritional deficiencies, and reduced erythrocyte lifespan. Previous studies have demonstrated that anemia may develop earlier in diabetic patients than in individuals with other causes of chronic kidney disease, suggesting that diabetes itself contributes independently to impaired erythropoiesis [12,13]. Reduced hemoglobin concentration adversely affects tissue oxygenation, accelerates renal deterioration, and increases the risk of cardiovascular complications and mortality among diabetic patients. Several hematological indices, including hemoglobin (Hb), packed cell volume (PCV), mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), and mean corpuscular hemoglobin concentration (MCHC), have been investigated as potential markers of disease progression in T2DM. Declining hemoglobin concentration and PCV are commonly observed in diabetic patients with renal impairment, whereas alterations in red blood cell indices may reflect underlying inflammation, nutritional deficiencies, or impaired erythropoiesis [14,15].

Furthermore, changes in platelet indices, particularly mean platelet volume (MPV), have been associated with increased platelet activation and enhanced thrombotic risk in patients with diabetes [16]. Although platelet count may remain within normal limits, functional abnormalities contribute significantly to the development of both microvascular and macrovascular complications. Numerous studies have reported significant associations between renal dysfunction and hematological abnormalities in patients with T2DM. Abebe et al. observed significantly lower hemoglobin levels and altered red blood cell indices among diabetic patients compared with healthy individuals [17]. Similarly, Chutani and Pande reported increased serum creatinine and urea concentrations accompanied by reductions in hemoglobin and hematocrit among patients with diabetes mellitus [18]. Kifle et al. further demonstrated significant alterations in complete blood count parameters in T2DM patients, highlighting the potential value of routine hematological assessment during diabetes follow-up [19]. Despite these observations, variations in demographic characteristics, glycemic control, duration of disease, nutritional status, and ethnicity have resulted in inconsistent findings across different populations.

In India, relatively few studies have simultaneously evaluated renal biochemical markers and hematological parameters among patients with T2DM attending tertiary care hospitals. Most available studies have focused either on renal function or hematological alterations independently, limiting comprehensive understanding of the interrelationship between these two systems. Simultaneous evaluation of renal and hematological parameters may provide valuable insights into the early identification of diabetic complications and facilitate more effective patient management. The present study was therefore undertaken to assess renal function parameters, including serum urea and serum creatinine, together with selected hematological parameters such as hemoglobin, packed cell volume, and mean corpuscular hemoglobin concentration among patients with Type 2 diabetes mellitus attending a tertiary care teaching hospital. The study also aimed to compare these parameters with healthy controls and determine whether significant hematological alterations accompany changes in renal function. Early recognition of these abnormalities may assist clinicians in identifying patients at increased risk of diabetic nephropathy and anemia, thereby supporting timely therapeutic interventions and improving long-term clinical outcomes [20–30].

MATERIALS AND METHODS

This hospital-based cross-sectional observational study was conducted over a period of three months in the Department of Biochemistry at A.C.S. Medical College and Hospital, Chennai, Tamil Nadu, India, to evaluate renal and hematological parameters among patients with Type 2 diabetes mellitus (T2DM). A total of 30 participants were enrolled, comprising 15 clinically diagnosed T2DM patients (cases) and 15 apparently healthy age- and sex-matched individuals (controls). Adult participants aged between 20 and 80 years with a confirmed diagnosis of Type 2 diabetes mellitus based on the American Diabetes Association (ADA) criteria were included in the study. Healthy volunteers without a history of diabetes served as the control group. Pregnant women, chronic smokers, alcoholics, patients with non-diabetic chronic kidney disease, those undergoing dialysis, individuals with hematological disorders, acute infections, malignancies, recent blood transfusion, or erythropoietin therapy were excluded to minimize potential confounding factors. Prior to participation, written informed consent was obtained from all subjects, and the study protocol was approved by the Institutional Ethics Committee of A.C.S. Medical College and Hospital in accordance with the ethical principles of the Declaration of Helsinki [31,32].

Approximately 5 mL of venous blood was collected from each participant under aseptic precautions. Blood samples were collected into plain vacutainer tubes for biochemical investigations and EDTA vacutainer tubes for hematological analysis. Serum was separated by centrifugation at 3000 rpm for 10 minutes and analyzed immediately. Renal function was assessed by estimating serum urea and serum creatinine using the Beckman Coulter AU480 Fully Automated Clinical Chemistry Analyzer (Beckman Coulter Inc., Brea, CA, USA). Serum urea was measured using the enzymatic urease method, while serum creatinine was determined by the modified Jaffe kinetic method. Hematological parameters, including hemoglobin (Hb), red blood cell (RBC) count, packed cell volume (PCV), mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentration (MCHC), total leukocyte count (TLC), and platelet count, were analyzed using the Sysmex XN-1000 Automated Hematology Analyzer (Sysmex Corporation, Kobe, Japan).

Internal quality control procedures were performed daily according to the manufacturers' recommendations to ensure the reliability and accuracy of the analytical measurements [33,34]. The collected data were entered into Microsoft Excel and analyzed using IBM Statistical Package for the Social Sciences (SPSS) version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD), whereas categorical variables were summarized as frequencies and percentages. The normality of data distribution was assessed using the Shapiro–Wilk test. Comparisons between diabetic patients and healthy controls were performed using the independent samples Student's t-test. A two-tailed p-value of <0.05 was considered statistically significant, with $p<0.01$ interpreted as highly significant. The findings were presented using tables and graphical illustrations to facilitate comparison between the study groups [35].

RESULTS

A total of 30 participants were included in the present study, comprising 15 patients with Type 2 diabetes mellitus (cases) and 15 apparently healthy individuals (controls). Renal function and hematological parameters were evaluated and compared between the two groups. The comparison of renal function parameters is presented in Table 1. The mean serum urea concentration was higher in diabetic patients (37.80 ± 23.34 mg/dL) than in controls (26.60 ± 7.52 mg/dL). Similarly, the mean serum creatinine concentration was elevated in the diabetic group (1.63 ± 1.91 mg/dL) compared with the control group (0.90 ± 0.44 mg/dL). However, the differences in serum urea ($p = 0.506$) and serum creatinine ($p = 0.237$) were not statistically significant.

Parameter	Control (n = 15) Mean ± SD	Cases (n = 15) Mean ± SD	*p*-value
Serum Urea (mg/dL)	26.60 ± 7.52	37.80 ± 23.34	0.506
Serum Creatinine (mg/dL)	0.90 ± 0.44	1.63 ± 1.91	0.237

Table 1. Comparison of Renal Function Parameters Between Controls and Patients with Type 2 Diabetes Mellitus

The comparison of renal function parameters between diabetic patients and healthy controls is illustrated in Figure 1. Although higher mean serum urea and creatinine concentrations were observed among diabetic patients, the differences between the two groups were not statistically significant. The hematological findings are summarized in Table 2. The mean hemoglobin concentration was significantly lower in diabetic patients (11.00 ± 1.61 g/dL) than in healthy controls (13.17 ± 2.12 g/dL), and this difference was highly significant (p = 0.003). Packed cell volume (PCV) was also significantly reduced in diabetic patients (33.7 ± 4.2%) compared with controls (41.0 ± 5.2%) (p = 0.021). Likewise, mean corpuscular hemoglobin concentration (MCHC) was significantly lower among diabetic patients (31.3 ± 1.8 g/dL) than among controls (33.5 ± 1.3 g/dL) (p = 0.034).

Parameter	Control (n = 15) Mean ± SD	Cases (n = 15) Mean ± SD	p-value
Hemoglobin (g/dL)	13.17 ± 2.12	11.00 ± 1.61	0.003
Packed Cell Volume (%)	41.0 ± 5.2	33.7 ± 4.2	0.021
MCHC (g/dL)	33.5 ± 1.3	31.3 ± 1.8	0.034

Table 2. Comparison of Hematological Parameters Between Controls and Patients with Type 2 Diabetes Mellitus

The comparison of hematological parameters between diabetic patients and healthy controls is presented in Figure 2. Diabetic patients demonstrated lower mean values of hemoglobin, packed cell volume, and MCHC compared with the control group, with all three parameters showing statistically significant differences. No statistically significant differences were observed in total leukocyte count or platelet count between diabetic patients and healthy controls. The mean values of these parameters remained within the normal physiological range in both groups.

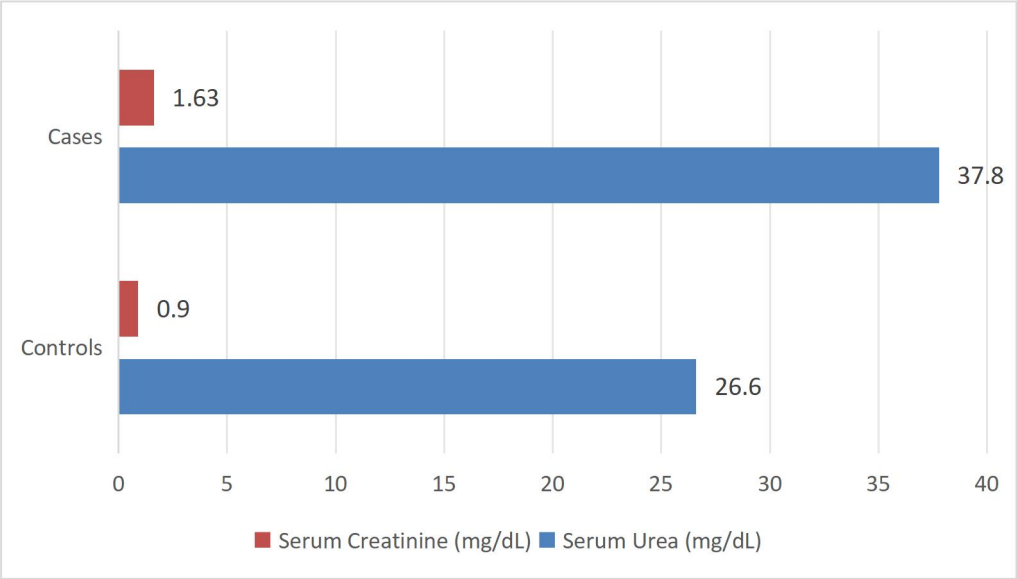

Figure 1. Comparison of Serum Urea and Serum Creatinine Between Controls and Patients with Type 2 Diabetes Mellitus

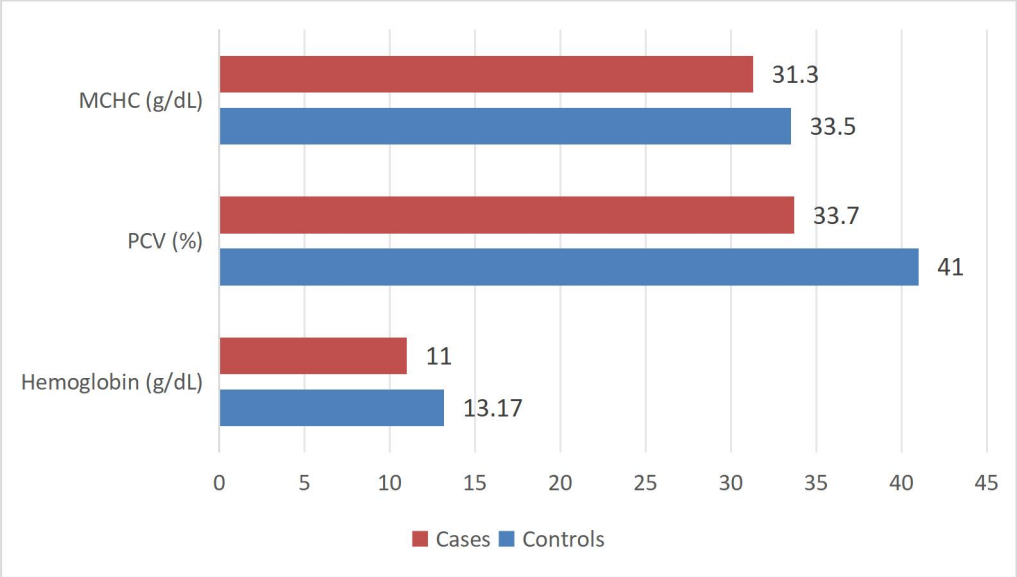

Figure 2. Comparison of Hematological Parameters Between Controls and Patients with Type 2 Diabetes Mellitus

DISCUSSION

The present cross-sectional study evaluated renal function and hematological parameters among patients with Type 2 diabetes mellitus (T2DM) and compared the findings with those of healthy controls. The study demonstrated higher serum urea and creatinine concentrations in diabetic patients, although these differences were not statistically significant. In contrast, significant reductions were observed in hemoglobin, packed cell volume (PCV), and mean corpuscular hemoglobin concentration (MCHC), indicating the presence of anemia-related hematological alterations among individuals with T2DM. These findings emphasize that diabetes affects multiple physiological systems simultaneously and highlight the importance of routine monitoring of both renal and hematological parameters for early identification of diabetic complications.

Diabetic nephropathy is one of the most common microvascular complications of T2DM and remains a leading cause of chronic kidney disease and end-stage renal disease worldwide [2]. Persistent hyperglycemia induces oxidative stress, endothelial dysfunction, activation of the renin-angiotensin-aldosterone system, and accumulation of advanced glycation end products, resulting in progressive glomerular injury and declining renal function [5,7]. In the present study, diabetic patients exhibited higher mean serum urea and serum creatinine concentrations compared with healthy controls. Although these differences did not reach statistical significance, the observed trend suggests early impairment of renal function among diabetic patients. The lack of statistical significance may be attributed to the relatively small sample size and the inclusion of patients with varying durations and severity of diabetes. The present findings are consistent with those reported by Biri et al., who observed elevated serum urea and creatinine levels among diabetic patients compared with healthy individuals, suggesting early renal involvement in diabetes mellitus [10]. Similarly, Mishra et al. demonstrated a positive association between fasting blood glucose, serum urea, serum creatinine, and the duration of diabetes, indicating progressive deterioration of renal function with prolonged hyperglycemia [30]. Karki et al. also reported significantly higher renal function markers among patients with Type 2 diabetes mellitus, emphasizing the importance of routine biochemical monitoring for early detection of diabetic nephropathy [24].

Although serum creatinine and urea remain widely used indicators of renal function, these conventional biomarkers often increase only after substantial nephron loss has occurred, highlighting the need for more sensitive markers such as estimated glomerular filtration rate (eGFR), urinary albumin excretion, and cystatin C in future studies [16,20,28]. One of the most important findings of the present study was the significant reduction in hemoglobin concentration among diabetic patients compared with healthy controls. The mean hemoglobin level was significantly lower in the diabetic group ($p = 0.003$), indicating a high prevalence of anemia. Anemia is increasingly recognized as an important complication of diabetes mellitus and frequently develops before advanced chronic kidney disease becomes clinically evident. Reduced erythropoietin synthesis due to diabetic renal injury, chronic inflammation, oxidative stress, functional iron deficiency, and reduced erythrocyte survival collectively contribute to the development of anemia in diabetic individuals [13,21].

Our findings are in agreement with those reported by Abebe et al., who demonstrated significantly lower hemoglobin concentrations among patients with Type 2 diabetes mellitus compared with healthy controls [1]. Similarly, Chutani and Pande observed significant reductions in hemoglobin and hematocrit in diabetic patients, attributing these changes to impaired erythropoiesis and chronic renal dysfunction [6,11]. Kifle et al. also reported decreased hemoglobin levels and altered erythrocyte indices among diabetic patients, suggesting that routine complete blood count evaluation may facilitate early identification of hematological complications [25]. Collectively, these studies support the findings of the present investigation and reinforce the close relationship between diabetes mellitus and anemia. Packed cell volume (PCV) was significantly reduced among diabetic patients in the present study. PCV reflects the proportion of circulating blood volume occupied by erythrocytes and is considered an important indicator of oxygen-carrying capacity. Reduced PCV among diabetic patients may result from decreased erythropoietin production, chronic inflammation, nutritional deficiencies, or reduced red blood cell survival. Previous investigations have demonstrated similar reductions in hematocrit among diabetic individuals, particularly in those with diabetic nephropathy [21,29]. Reduced PCV contributes to impaired tissue oxygenation and may accelerate the progression of diabetic complications, including cardiovascular disease and chronic kidney disease.

The present study also demonstrated a significant reduction in mean corpuscular hemoglobin concentration (MCHC) among diabetic patients. MCHC reflects the average concentration of hemoglobin within erythrocytes and provides valuable information regarding red blood cell morphology. Reduced MCHC may indicate impaired hemoglobin synthesis, functional iron deficiency, or chronic inflammatory changes associated with diabetes mellitus. Similar observations have been reported by Aghaei et al. and Kifle et al., who identified significant alterations in erythrocyte indices among diabetic patients compared with healthy controls [3,25]. These findings suggest that diabetes mellitus influences not only the quantity of erythrocytes but also their structural and functional characteristics. Although alterations in erythrocyte-related parameters were evident, the present study did not demonstrate statistically significant differences in total leukocyte count or platelet count between diabetic patients and controls.

Similar findings have been reported by several investigators, who observed that while platelet numbers often remain within the normal range, platelet function and activation are significantly altered in diabetes mellitus [9,22,26]. Increased platelet activation, elevated mean platelet volume, and enhanced platelet aggregation have been implicated in the pathogenesis of diabetic vascular complications, even in the absence of changes in platelet count. Therefore, assessment of platelet indices and functional markers may provide additional prognostic information in future investigations. The coexistence of altered renal function markers and anemia-related hematological abnormalities observed in the present study supports the concept of a close interaction between diabetic nephropathy and impaired erythropoiesis. Even mild renal dysfunction may reduce erythropoietin synthesis, leading to declining hemoglobin levels before overt renal failure develops. Early recognition of these changes is clinically important because untreated anemia accelerates cardiovascular complications, reduces quality of life, and contributes to faster progression of chronic kidney disease [2,13,21]. The findings of the present study have important clinical implications. Routine assessment of serum urea, serum creatinine, and complete blood count should be incorporated into the regular follow-up of patients with Type 2 diabetes mellitus.

Early identification of declining renal function and anemia may facilitate timely therapeutic interventions, including optimization of glycemic control, correction of nutritional deficiencies, management of chronic kidney disease, and prevention of further complications. Such comprehensive evaluation may improve long-term clinical outcomes and reduce diabetes-related morbidity. The present study has certain limitations. First, the sample size was relatively small, limiting the statistical power and generalizability of the findings. Second, the study was conducted at a single tertiary care center, and therefore the results may not represent the broader diabetic population. Third, important clinical variables such as glycated hemoglobin (HbA1c), duration of diabetes, estimated glomerular filtration rate (eGFR), urinary albumin excretion, iron profile, vitamin B12 status, and inflammatory biomarkers were not evaluated. Finally, the cross-sectional design precludes establishing causal relationships between diabetes, renal dysfunction, and hematological alterations. Future multicenter studies with larger sample sizes and comprehensive biomarker assessment are required to validate these findings and further elucidate the relationship between renal impairment and hematological abnormalities in patients with Type 2 diabetes mellitus. Overall, the present study demonstrates that patients with Type 2 diabetes mellitus exhibit early alterations in renal biochemical markers together with significant reductions in hemoglobin, packed cell volume, and mean corpuscular hemoglobin concentration. These findings underscore the importance of integrated renal and hematological evaluation as part of routine diabetes management, enabling earlier detection of complications and supporting timely interventions to improve patient outcomes [1,2,6,10,17,24,25].

Conclusion:

The present study demonstrated that patients with Type 2 diabetes mellitus exhibit alterations in both renal and hematological parameters when compared with healthy individuals. Although serum urea and serum creatinine levels were higher among diabetic patients, the differences were not statistically significant, suggesting the presence of early renal impairment. In contrast, significant reductions in hemoglobin, packed cell volume, and mean corpuscular hemoglobin concentration indicated a high prevalence of anemia-related hematological abnormalities in the diabetic population. These findings highlight the close association between impaired renal function and altered erythropoiesis in diabetes mellitus.

Routine assessment of renal function tests together with complete blood count parameters may facilitate the early detection of diabetic nephropathy and anemia, allowing timely therapeutic intervention and improved clinical management. Further multicenter studies with larger sample sizes and inclusion of advanced renal biomarkers such as estimated glomerular filtration rate (eGFR), urinary albumin excretion, and cystatin C are recommended to validate the present findings and better characterize the progression of renal and hematological complications in patients with Type 2 diabetes mellitus.

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