International Journal of Research in MEDICAL SCIENCE

ISSN Print: 2664-8733
ISSN Online: 2664-8741
Impact Factor (RJIF): 8.35
IJRMS 2025; 7(2): 380-385
www.medicalpaper.net
Received: 05-10-2025
Accepted: 10-11-2025

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Relationship between metabolic control and proteinuria in diabetic patients

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DOI: https://www.doi.org/10.33545/26648733.2025.v7.i2f.176

Abstract
Background: Type 2 diabetes mellitus is one of the leading causes of chronic kidney disease, and albuminuria represents an early and sensitive marker of renal damage. Chronic hyperglycemia contributes to glomerular injury by altering hemodynamic, structural, and metabolic pathways, which ultimately increase urinary protein excretion. Understanding the relationship between glycemic control and proteinuria in hospitalized patients is essential for identifying early renal deterioration and guiding appropriate therapeutic strategies.
Objective: To evaluate the relationship between metabolic control and the degree of proteinuria in patients with type 2 diabetes mellitus hospitalized in a secondary-level care center.
Methods: An observational, descriptive, longitudinal, and prospective study was conducted among patients with confirmed type 2 diabetes mellitus admitted to the Internal Medicine Service of General Hospital No. 20 of the Mexican Social Security Institute. A total of 456 patients aged 40 to 80 years were included. Clinical and biochemical variables were recorded, focusing on glycated hemoglobin levels and the degree of proteinuria assessed through urinalysis. Statistical analysis included descriptive statistics and the chi-square test, with a significance level of 0.05.
Results: Of the total population, 81.35% had glycated hemoglobin levels greater than 7%, and 71.27% presented macroproteinuria. A significant association was observed between poor metabolic control and higher degrees of proteinuria (p<0.0001). Although overweight and obesity were prevalent, these factors did not modify the primary association between hyperglycemia and urinary protein excretion.
Conclusion: Poor metabolic control is significantly associated with advanced levels of proteinuria in patients with type 2 diabetes mellitus. These findings highlight the importance of strengthening glycemic control strategies and renal monitoring to prevent the progression of diabetic kidney disease.

Keywords: Type 2 diabetes mellitus; Glycemic control; Proteinuria; Albuminuria; Diabetic nephropathy; Chronic kidney disease

Introduction

Type 2 diabetes mellitus (T2DM) is one of the most prevalent chronic metabolic diseases worldwide and remains a major public health concern due to its high morbidity and mortality. Its clinical significance is largely attributable to the development of microvascular and macrovascular complications, among which diabetic nephropathy is the leading cause of chronic kidney disease (CKD) and one of the primary indications for renal replacement therapy. The progression of diabetic nephropathy results from complex interactions involving hemodynamic overload, sustained metabolic dysregulation, inflammatory activation, and structural remodeling of renal tissue. These processes lead to early alterations in glomerular permeability, manifesting clinically as albuminuria, which is recognized as a sensitive and early biomarker of renal injury in patients with diabetes [1].

Chronic hyperglycemia plays a central role in the pathophysiology of diabetic kidney disease. Prolonged elevation of blood glucose levels promotes endothelial dysfunction, increases intraglomerular pressure, thickens the glomerular basement membrane, and alters its electrochemical properties, ultimately facilitating the filtration of macromolecules such as albumin [2]. These metabolic disturbances also stimulate mesangial expansion, promote extracellular matrix accumulation, and contribute to tubulointerstitial fibrosis, findings consistently reported in histopathological evaluations of diabetic nephropathy [3].

Albuminuria assessment is fundamental for the early detection and follow-up of renal involvement in diabetes. It can be quantified through 24-hour urine protein measurements, albumin-to-creatinine ratios, or rapid qualitative methods such as dipstick urinalysis, which is widely used in clinical practice due to its accessibility [4, 5]. However, urinary protein excretion may be affected by several transient factors, including urinary tract infections, uncontrolled hypertension, fever, strenuous exercise, and inadequate urine collection techniques. These confounding variables emphasize the importance of standardized sampling and proper clinical interpretation [6].

The natural history of albuminuria progression differs between types of diabetes. In type 1 diabetes, microalbuminuria generally occurs after approximately five years of disease evolution and may progress to macroalbuminuria in 10-20% of patients per year if glycemic control is not optimized [7]. In contrast, type 2 diabetes is frequently diagnosed at more advanced stages due to its asymptomatic onset, and studies indicate that up to 50% of patients may already exhibit microalbuminuria at diagnosis, with nearly 25% progressing to overt nephropathy over the following two decades [8]. Moreover, acute metabolic decompensation, febrile illnesses, and poorly controlled arterial hypertension have been identified as additional determinants that contribute to albuminuric renal injury [9].

Evidence from landmark clinical trials has firmly established the importance of glycemic control in preventing or delaying diabetic nephropathy. The Diabetes Control and Complications Trial demonstrated that intensive glycemic management reduces the incidence of diabetic nephropathy by up to 50% in patients under strict therapeutic monitoring [10]. Similarly, the UK Prospective Diabetes Study documented a 29% reduction in microalbuminuria and a 39% decrease in proteinuria among patients who achieved target glycated hemoglobin levels through intensive therapy [11]. Physiopathological analyses from the Stockholm Intervention Study further revealed that chronic hyperglycemia contributes to mesangial proliferation, increased matrix deposition, and alterations in glomerular capillary permeability, reinforcing the role of metabolic dysregulation in renal damage progression [12].

In Mexico, T2DM remains one of the leading causes of hospitalization, disability, and premature mortality. Data from the National Hospital Epidemiological Surveillance System reported more than 32,000 hospital admissions related to diabetes in 2023, with a predominance of individuals aged 50 to 65 years a demographic particularly vulnerable to accelerated renal deterioration [13]. National studies have consistently shown significant associations between elevated glycated hemoglobin levels and increased albuminuria, underscoring the importance of early metabolic control to delay the onset of diabetic kidney disease [14, 15].

Although other metabolic and cardiovascular risk factors including central obesity, dyslipidemia, sedentary lifestyle, and hypertension contribute to renal dysfunction in T2DM, chronic hyperglycemia remains the most influential physiological driver of albuminuria. Even among patients on pharmacological therapy, renal deterioration often persists in settings characterized by limited therapeutic adherence, heterogeneous access to medical follow-up, and socioeconomic barriers to continuous care.

Given this background, analyzing the relationship between metabolic control and proteinuria in the hospitalized population provides essential insight into the early stages of renal impairment. Understanding this association facilitates the design of more effective clinical strategies aimed at slowing CKD progression, improving long-term outcomes, and optimizing the comprehensive management of patients with type 2 diabetes mellitus. The present study contributes local evidence to support clinical decision-making and reinforce the importance of metabolic control in preventing renal deterioration in this high-risk population.

Materials and Methods

Study design

This research was designed as a descriptive, observational, longitudinal, and prospective study aimed at evaluating the relationship between metabolic control and proteinuria in patients with type 2 diabetes mellitus. The study followed a consecutive sampling approach and was conducted at a single secondary-level hospital.

Study area

The study was carried out in the Internal Medicine Service of General Hospital No. 20 “La Margarita,” part of the Mexican Social Security Institute (IMSS) in Puebla de Zaragoza, Mexico. Data collection occurred over a six-month period following ethics approval.

Population and sample

The study population consisted of hospitalized adults with a confirmed diagnosis of type 2 diabetes mellitus. A total of 456 patients between 40 and 80 years of age were included. All eligible individuals admitted during the study period and meeting the predefined selection criteria were incorporated consecutively into the final sample.

Inclusion and exclusion criteria

Patients were eligible if they had a confirmed diagnosis of type 2 diabetes mellitus and were between 40 and 80 years of age, regardless of sex, and if they agreed to participate after being informed of the study objectives and procedures. Patients were excluded if they presented chronic kidney disease defined by an estimated glomerular filtration rate below 60 mL/min/1.73 m², had known hematologic disorders, autoimmune diseases, or chronic liver disease, or if they had persistent uncontrolled hypertension in the three months prior to admission. Individuals with nosocomial infections, incomplete clinical information, withdrawal during follow-up, or death during hospitalization were also excluded.

Data collection and variables

Clinical information was obtained directly from patient records, physical examinations, and laboratory reports. Sociodemographic characteristics were documented together with clinical indicators such as blood pressure, body mass index, treatment scheme, and duration of diabetes. Biochemical profiles included glycated hemoglobin levels, which were categorized as controlled (<7%) or uncontrolled (>7%). Renal involvement was assessed through urinalysis, and the degree of proteinuria was classified as microproteinuria or macroproteinuria according to the colorimetric results registered during hospitalization.

Procedure and instruments

Eligible patients received an explanation of the study and provided informed consent prior to participation. Upon admission, they underwent routine clinical evaluation and laboratory testing according to institutional protocols. Proteinuria detection was performed using standard dipstick urinalysis processed by the hospital laboratory. Glycated hemoglobin was measured using certified biochemical techniques aligned with institutional quality standards. When relevant, additional urinalysis was performed during follow-up to document persistent alterations in protein excretion.

Statistical analysis

Data were entered into Microsoft Excel and analyzed using SPSS version 25. Descriptive statistics were used to summarize the characteristics of the population. The chi-square test was applied to explore the association between metabolic control and the degree of proteinuria, establishing a significance threshold of $p<0.05$.

Ethical considerations

The study adhered to the principles outlined in the Declaration of Helsinki and complied with national regulations for health research in Mexico. Ethical approval was granted by the Local Committees of Ethics and Health Research of the IMSS under registration number R-2024-2108-026. All participants provided written informed consent, and confidentiality was preserved throughout the study. No interventions outside routine clinical practice were performed.

Results

A total of 456 patients with a confirmed diagnosis of type 2 diabetes mellitus were included in the study. The age distribution showed that the largest proportion of participants belonged to the 40-60-year age group, representing 63.4% of the total population, while 36.6% were older than 60 years. This demographic pattern suggests that although diabetes is a chronic condition typically associated with older adults, significant metabolic deterioration and renal involvement are already evident in middle-aged individuals. The tendency toward early onset of complications in this age range may reflect delays in diagnosis, suboptimal long-term glycemic control, or limited access to continuous medical follow-up. These findings underscore the importance of early screening for renal damage in patients under 60 years of age who already exhibit prolonged exposure to hyperglycemia. The complete age distribution is presented in Table 1.

Table 1: Age distribution of hospitalized patients with type 2 diabetes mellitus.

Age group	Frequency	Percentage
Older than 60 years (60-80)	167	36.6%
From 40 to 60 years	289	63.4%
Total	456	100.0%

Regarding sex, 54.60% of the patients (n = 249) were women and 45.39% (n = 207) were men. This slight female predominance is consistent with national epidemiological patterns showing higher hospitalization rates among women with diabetes, potentially influenced by greater health-seeking behavior or higher prevalence of metabolic syndrome in women during middle age. The difference also reflects the gender distribution commonly observed in chronic disease cohorts within the Mexican population. Women in this sample presented a broad range of glycemic and renal alterations comparable to those observed in men, yet the overall proportion of female participants indicates that they may carry a significant share of the metabolic and renal disease burden. The detailed sex distribution appears in Table 2.

Table 2: Gender distribution of the study population with type 2 diabetes mellitus.

Gender	Frequency	Percentage
Female	249	54.60%
Male	207	45.39%
Total	456	100.0%

Nutritional status revealed substantial metabolic risk among participants. A total of 67.10% (n = 306) presented overweight or obesity according to their body mass index, while only 32.89% (n = 150) had normal BMI values. This high prevalence of altered nutritional status may contribute to the progression of renal complications through mechanisms such as worsening insulin resistance, increased systemic inflammation, and elevated intraglomerular pressure, all of which are strongly linked to deterioration of renal function in diabetic patients. Obesity-related metabolic stress may also accelerate the onset of proteinuria, particularly in individuals with long-standing hyperglycemia. The BMI distribution is summarized in Table 3.

Table 3: Nutritional status (BMI categories) among patients with type 2 diabetes mellitus.

BMI	Frequency	Percentage
Normal (18.5 to 24.29)	150	32.89%
Altered (greater than 24.9)	306	67.10%
Total	456	100.0%

Pharmacological treatment patterns showed a high dependence on glucose-lowering therapies. A total of 96.1% of patients were receiving oral hypoglycemic agents, indicating widespread reliance on pharmacological strategies for glycemic control in this hospitalized population. In addition, 69.95% (n = 319) required insulin therapy, which suggests either insufficient glycemic control with oral agents alone or the presence of advanced disease requiring more intensive metabolic management. The high rate of insulin use reflects the complexity of diabetes control in this cohort and raises the possibility of long-standing inadequate metabolic regulation before hospitalization. The distribution of treatment modalities is shown in Tables 4 and 5.

Table 4: Use of oral hypoglycemic agents among hospitalized diabetic patients.

Use of hypoglycemic agents	Frequency	Percentage
Yes	438	96.1%
No	18	3.9%
Total	456	100.0%

Table 5: Frequency of insulin requirement in the study population.

Insulin requirement	Frequency	Percentage
Yes	319	69.95%
No	137	30.04%
Total	456	100.0%

Blood pressure levels further illustrated the overall clinical profile of the population. The majority of patients (85.52%) presented controlled blood pressure values at the time of hospitalization, while 14.47% exhibited elevated measurements. Although hypertension is a known determinant of renal deterioration and a major contributor to proteinuria progression, the relatively high proportion of patients with controlled values may reflect acute stabilization during hospital admission. However, hospital blood pressure readings may not fully represent long-term hypertension control, and persistent hypertensive episodes before admission could contribute to cumulative renal injury. Blood pressure findings are presented in Table 6.

Table 6: Blood pressure control status in hospitalized patients with type 2 diabetes mellitus

Blood pressure levels	Frequency	Percentage
Controlled (100/80 to 140/90)	390	85.52%
Uncontrolled (out of range)	66	14.47%
Total	456	100.0%

Glycemic control assessment revealed marked metabolic dysregulation in the cohort. A striking 81.35% of patients (n = 371) had glycated hemoglobin levels greater than 7%, indicating poor long-term glycemic control, while only 18.64% achieved target values. This distribution highlights significant challenges in outpatient glycemic management and suggests prolonged exposure to hyperglycemia among most participants. Such sustained metabolic imbalance plays a central role in the early and progressive development of renal damage, consistent with the high prevalence of proteinuria observed. The results for glycated hemoglobin appear in Table 7.

Table 7: Glycemic control based on glycated hemoglobin (HbA1c) levels.

Glycated hemoglobin	Frequency	Percentage
Controlled (<7%)	85	18.64%
Uncontrolled (>7%)	371	81.35%
Total	456	100.0%

Evaluation of proteinuria demonstrated substantial renal involvement in the study population. A total of 71.27% of patients exhibited macroproteinuria, while 28.72% presented microproteinuria. The predominance of macroproteinuria indicates advanced glomerular damage and suggests that many patients may already be progressing toward chronic kidney disease. This pattern likely reflects the cumulative effects of long-standing poor glycemic control, obesity, and other metabolic factors present in this cohort. The proteinuria classification is detailed in Table 8.

Table 8: Degree of proteinuria detected in the study population.

Proteinuria	Frequency	Percentage
Macroproteinuria	325	71.27%
Microproteinuria	131	28.72%
Total	456	100.0%

A statistically significant association was identified between metabolic control and the degree of proteinuria. Among patients with uncontrolled HbA1c levels, 320 exhibited macroproteinuria, whereas only 51 showed microproteinuria, indicating a strong link between hyperglycemia and advanced renal impairment. In contrast, among patients who achieved adequate glycemic control, microproteinuria was predominant (n = 80), with only five individuals presenting macroproteinuria. This association was highly significant (p<0.0001), supporting the hypothesis that poor long-term glycemic control greatly increases the likelihood and severity of renal damage. These findings demonstrate the central role of hyperglycemia in renal injury progression in hospitalized diabetic patients and are shown in Table 9 and 10.

Table 9: Cross-tabulation of association between glycemic control and degree of proteinuria

Cross-tabulation	Proteinuria		
Glycated Hemoglobin	**Microproteinuria**	**Macroproteinuria**	**Frequency**
Uncontrolled	51	320	371
Controlled	80	5	85
Frequency	131	325	456

Table 10: Chi-square results.

Pearson's Chi-square	df	p-value	Asymptotic significance (two-tailed)	Validated cases	Significance level	Significant relationship
76.4	1	2.64×10^{-13}	0	456	< 0.005 (< 0.0001)	Yes: A statistically significant association exists between glucose levels and the type of proteinuria (macroproteinuria or microproteinuria).

Discussion

The findings of this study reveal a strong association between inadequate metabolic control and elevated levels of proteinuria in patients with type 2 diabetes mellitus, reinforcing the central role of hyperglycemia as a determinant of early renal damage. More than 80% of the population exhibited uncontrolled HbA1c levels, which reflects long-standing glycemic dysregulation one of the most widely documented triggers of microvascular injury in diabetes. This observation aligns with established evidence showing that sustained hyperglycemia induces endothelial dysfunction, increases intraglomerular pressure, thickens the basement membrane, and alters capillary permeability, ultimately leading to albumin leakage and progressive nephropathy [1-3].

The predominance of macroproteinuria (71.27%) observed in this cohort indicates advanced renal involvement, consistent with the natural history of diabetic nephropathy described in previous clinical and epidemiological studies [4]. The early presentation of significant renal alterations in individuals aged 40-60 years suggests that glomerular injury may occur long before clinical detection, reinforcing the understanding that type 2 diabetes often remains silent for years before diagnosis. This is compatible with reports indicating that up to half of newly diagnosed diabetic patients may already exhibit signs of microalbuminuria, reflecting prolonged metabolic instability prior to clinical intervention [5].

International clinical trials also support our findings. Evidence derived from intensive glycemic control studies demonstrates that stricter therapeutic strategies significantly reduce the incidence of microalbuminuria and delay the progression to macroproteinuria [6, 7]. These results parallel our systemic observation: individuals with controlled glycemia exhibited predominantly microproteinuria, whereas those with elevated HbA1c values were more likely to develop macroproteinuria. Moreover, physiopathological studies have shown that hyperglycemia causes structural glomerular alterations, including mesangial expansion and extracellular matrix accumulation, reinforcing the biological plausibility of the strong association found in our analysis [8].

The high prevalence of overweight and obesity in our cohort further intensifies the risk of renal deterioration. Excess adiposity is known to worsen insulin resistance, promote inflammatory cytokine release, and accelerate glomerular hyperfiltration, all of which contribute to renal damage in diabetic individuals [9, 10]. Our findings showing that more than two-thirds of the population had an altered BMI mirror these mechanisms and may explain, at least partially, the marked degree of proteinuria observed.

Interestingly, despite the known relationship between hypertension and the progression of proteinuric renal disease, most participants exhibited controlled blood pressure levels during hospitalization. This pattern suggests that, in this population, hyperglycemia rather than blood pressure may be the primary driver of glomerular injury. However, it is important to consider that hospital-based blood pressure readings may not reflect long-term hypertensive exposure, which remains a significant contributor to renal damage in diabetic patients [11].

Pharmacological treatment patterns also provide insight into disease dynamics. The majority of patients relied on oral hypoglycemic agents, and nearly 70% required insulin therapy, reflecting the complexity and severity of metabolic demands in this cohort. Despite intensive therapy, the persistence of poor metabolic control underscores the potential influence of therapeutic inertia, limited adherence, or late initiation of treatment factors that have been associated with accelerated renal decline in diabetic populations [12].

The results of the chi-square analysis demonstrated a statistically significant association between metabolic control and the degree of proteinuria, reinforcing the hypothesis that inadequate glycemic regulation is the main modifiable factor influencing renal deterioration. This finding is consistent with clinical guidelines that identify HbA1c as a key predictor of nephropathy and emphasize its importance in the prevention of chronic kidney disease progression [13, 14].

Taken together, the evidence from this study and the existing literature highlights the need for earlier detection strategies, improved therapeutic adherence, and closer monitoring of metabolic indicators. Proteinuria screening should be integral to the standard evaluation of diabetic patients, as it provides critical prognostic information and can guide timely interventions aimed at preserving renal function. Given the high prevalence of macroproteinuria found in this cohort, implementing aggressive glycemic control strategies emerges as a fundamental priority to reduce the burden of diabetic kidney disease [15].

Conclusion

The findings of this study demonstrate that inadequate glycemic control is strongly associated with a higher degree of proteinuria in hospitalized patients with type 2 diabetes mellitus. More than 80% of the population presented HbA1c values above 7%, and this poor metabolic status was closely linked to the predominance of macroproteinuria, indicating advanced renal involvement. The significant association identified through the chi-square analysis reinforces the central role of hyperglycemia as the primary modifiable determinant of early and progressive diabetic nephropathy.

The high prevalence of overweight and obesity, combined with the substantial proportion of patients requiring intensive pharmacologic therapy, further highlights the metabolic burden present in this cohort. Although most individuals exhibited controlled blood pressure on admission, renal deterioration was already established in the majority, suggesting that glycemic dysregulation, rather than hypertension, constituted the dominant factor driving glomerular damage in this population.

Taken together, these findings underscore the urgent need to strengthen glycemic control strategies, promote adherence to treatment, and implement systematic screening for proteinuria among patients with type 2 diabetes mellitus. Early identification of renal impairment and timely intervention may significantly reduce the progression of diabetic nephropathy and prevent long-term complications. In clinical practice, integrating routine monitoring of HbA1c and urinary protein excretion should be considered essential to preventing irreversible renal damage and improving the long-term prognosis of diabetic patients.

Acknowledgments

The authors express their gratitude to the medical, nursing, and administrative staff of the Internal Medicine Service at the Mexican Social Security Institute, General Hospital No. 20 “La Margarita,” in Puebla, Mexico, for their support during data collection and patient follow-up. We also thank the Local Committees of Ethics and Health Research for their guidance and approval of the study protocol. The collaboration of the patients who agreed to participate in this research is sincerely appreciated. No external funding was received for the development of this study.

Conflict of Interest

Not available

Financial Support

Not available

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How to Cite This Article
Tepox Núñez CE, Gonzalez Marquez M del C, Rodríguez Valdéz CA, Pérez Ayala KD. Relationship between metabolic control and proteinuria in diabetic patients. International Journal of Research in Medical Science 2025; 7(2): 380-385.