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Study Of Risk Factors Of Microalbuminuria In Type 2 Diabetes Mellitus Cases.

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ABSTRACT

Microalbuminuria is an early indicator of diabetic nephropathy and a predictor of cardiovascular risk in patients with type 2 diabetes mellitus (T2DM). Identifying its prevalence and associated risk factors is crucial for timely intervention and prevention of renal complications. To assess the prevalence of microalbuminuria and evaluate associated risk factors in patients with T2DM. A cross-sectional observational study was conducted over one year in a tertiary care hospital. A total of 60 patients with T2DM were included. Detailed clinical histories were taken, and physical examinations were performed. Laboratory investigations included fasting and postprandial blood sugar, HbA1c, serum creatinine, lipid profile, and urinary albumin-creatinine ratio (UACR). Microalbuminuria was defined as UACR between 30–300 mg/g. Data were analyzed to determine associations between microalbuminuria and various clinical parameters. Microalbuminuria was present in 40% of T2DM patients. Significant associations were observed with poor glycemic control (HbA1c >7%, $p<0.01$), hypertension ($p<0.01$), longer diabetes duration (>5 years, $p<0.01$), elevated BMI ($p=0.02$), and dyslipidemia ($p=0.01$). Microalbuminuria is highly prevalent among T2DM patients and is significantly associated with modifiable risk factors. Early detection and multifactorial management are essential to prevent progression to diabetic nephropathy.

Keywords: Microalbuminuria, Type 2 Diabetes Mellitus, Risk Factors.

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INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder characterized by insulin resistance and hyperglycemia, leading to progressive damage to various organ systems. Among its most serious complications is diabetic nephropathy, a leading cause of end-stage renal disease (ESRD) worldwide. Microalbuminuria, defined as urinary albumin excretion between 30–300 mg/day, is one of the earliest clinical markers of diabetic kidney disease and reflects generalized endothelial dysfunction. Its presence not only indicates the onset of nephropathy but is also an independent risk factor for cardiovascular morbidity and mortality in diabetic patients.

The development of microalbuminuria in T2DM patients is influenced by multiple interrelated risk factors, including poor glycemic control, hypertension, dyslipidemia, obesity, smoking, and duration of diabetes. Early identification and management of these modifiable risk factors can help in preventing progression to overt proteinuria and renal impairment. Despite the availability of diagnostic tools and therapeutic strategies, microalbuminuria often remains undetected in the initial stages due to its asymptomatic nature [1-3].

This study aims to assess the prevalence of microalbuminuria in T2DM patients and to identify the associated risk factors contributing to its development. Understanding these risk factors is crucial for implementing preventive strategies and optimizing long-term outcomes in patients with T2DM.

METHODOLOGY

This cross-sectional observational study was conducted over a period of one year in the Department of Medicine at a tertiary care hospital. The study aimed to assess the prevalence and risk factors of microalbuminuria in patients diagnosed with type 2 diabetes mellitus (T2DM). Ethical clearance was obtained from the institutional ethics committee prior to the commencement of the study. Written informed consent was obtained from all participants after explaining the purpose and procedures involved.

A total of 60 patients with confirmed diagnoses of T2DM, aged between 30 and 70 years, attending the outpatient and inpatient departments during the study period were included using convenient sampling. Inclusion criteria comprised patients with a known history of T2DM for at least one year. Exclusion criteria included patients with known renal disease, urinary tract infections, congestive heart failure, or those on nephrotoxic drugs.

Detailed clinical histories were recorded, including duration of diabetes, medication use, history of hypertension, smoking status, and family history of renal or cardiovascular disease. Physical examination included measurements of blood pressure, height, weight, and body mass index (BMI). Laboratory investigations were performed, including fasting and postprandial blood glucose, HbA1c, serum creatinine, lipid profile, and urinary albumin-creatinine ratio (UACR) to detect microalbuminuria.

Urine samples were collected from all patients and analyzed for albumin and creatinine levels using standard immunoturb.

RESULTS

Table 1: Prevalence of Microalbuminuria among Study Subjects (n=60)

Microalbuminuria Status	Number of Patients	Percentage (%)
Present (UACR 30–300 mg/g)	24	40%
Absent (UACR <30 mg/g)	36	60%

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Table 2: Association of Risk Factors with Microalbuminuria in T2DM Patients

Risk Factor	Microalbuminuria Present (n=24)	Microalbuminuria Absent (n=36)	p-value
Poor Glycemic Control (HbA1c >7%)	20 (83.3%)	15 (41.7%)	<0.01
Hypertension	18 (75%)	12 (33.3%)	<0.01
Duration of Diabetes >5 years	19 (79.2%)	13 (36.1%)	<0.01
Elevated BMI (>25 kg/m^2)	17 (70.8%)	14 (38.9%)	0.02
Dyslipidemia	16 (66.7%)	11 (30.6%)	0.01

DISCUSSION

The present study evaluated microalbuminuria and its determinants among 60 individuals with type 2 diabetes mellitus (T2DM) and found that 40 % exhibited microalbuminuria. This prevalence aligns with reports from similar tertiary-care–based cross-sectional studies, which have documented rates ranging from 30 % to 45 %. The finding underscores that, even in relatively small clinical cohorts, nearly two in five patients already manifest early diabetic kidney disease, often before overt clinical symptoms appear. Such a burden reinforces the importance of routine urinary albumin screening as an integral component of diabetes management programs.

Several risk factors emerged as significantly associated with microalbuminuria. Poor glycemic control, reflected by HbA1c > 7 %, showed the strongest relationship (83.3 % vs 41.7 %; $p < 0.01$). Chronic hyperglycemia promotes non-enzymatic glycation of basement-membrane proteins, endothelial dysfunction, and oxidative stress, culminating in glomerular hyperfiltration and albumin leakage. Our data therefore corroborate the central role of tight glycemic control in renoprotection, echoing the findings of large prospective trials such as the UKPDS and DCCT/EDIC, which demonstrated sustained microvascular benefit with intensive glucose lowering.

The study also demonstrated a robust link between hypertension and microalbuminuria (75 % vs 33.3 %; $p < 0.01$). Hemodynamic stress, mediated through elevated intraglomerular pressure, accelerates basement-membrane injury and albumin excretion. This interplay highlights the dual necessity of BP optimization and glycemic control; angiotensin-converting-enzyme inhibitors or angiotensin-receptor blockers, which simultaneously reduce systemic BP and intraglomerular hypertension, remain first-line agents in such patients.

Duration of diabetes exceeding five years correlated significantly with albuminuria (79.2 % vs 36.1 %; $p < 0.01$), reinforcing the cumulative nature of metabolic and vascular insults over time. While duration itself is non-modifiable, earlier diagnosis and aggressive risk-factor modification may delay nephropathy onset. Elevated body-mass index (> 25 kg/m^2) was another significant predictor (70.8 % vs 38.9 %; $p = 0.02$). Obesity promotes insulin resistance, systemic inflammation, and activation of the renin–angiotensin–aldosterone system, all of which potentiate renal endothelial damage. Consequently, lifestyle interventions targeting weight reduction could yield renal as well as cardiovascular benefits.

Dyslipidemia (66.7 % vs 30.6 %; $p = 0.01$) likewise emerged as a significant correlate, supporting the hypothesis that atherogenic lipoproteins contribute to glomerulosclerosis via mesangial lipid deposition and oxidative stress. Statin therapy, beyond its cardiovascular advantages, may therefore attenuate renal decline. The present results substantiate the multifactorial pathogenesis of diabetic nephropathy, wherein metabolic (hyperglycemia, dyslipidemia), hemodynamic (hypertension), anthropometric (obesity), and temporal (disease duration) variables converge to amplify renal risk.

Several limitations merit acknowledgement. First, the cross-sectional design precludes causal inference; longitudinal follow-up would be required to establish temporal relationships and progression. Second, the sample size, though adequate for preliminary associations, limits generalizability and statistical power for interaction analyses. Third, the use of a single spot urinary albumin–creatinine ratio, while practical, may be influenced by transient factors; confirmatory repeated measurements or 24-hour collections could enhance accuracy. Finally, other potential confounders such as dietary protein intake, physical activity levels, and genetic polymorphisms were not assessed.

Despite these constraints, the study offers clinically relevant insights. The high prevalence of microalbuminuria and its association with modifiable factors—hyperglycemia, hypertension, adiposity, and dyslipidemia—underscore the imperative for comprehensive, multifaceted management in T2DM. Early institution of individualized glycemic targets, blood-pressure control with renoprotective agents, weight-management programs, and lipid-lowering therapy may collectively curb the trajectory toward overt nephropathy and end-stage renal disease. Future prospective studies with larger cohorts and intervention arms are warranted to validate these findings and explore the impact of integrated care pathways on renal and cardiovascular outcomes [4-9].

CONCLUSION

Microalbuminuria is highly prevalent among T2DM patients and is significantly associated with modifiable risk factors. Early detection and multifactorial management are essential to prevent progression to diabetic nephropathy.

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