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STUDY OF MICROALBUMINURIA AND PERIPHERAL NEUROPATHY IN PREDIABETES

Neurology

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ABSTRACT

Background: Prediabetes is an intermediate metabolic state associated with increased risk of progressing to type 2 diabetes mellitus (T2DM). Evidence suggests that complications typically linked to long-standing diabetes, such as microalbuminuria and peripheral neuropathy, may begin developing even during the prediabetic stage. Early recognition of these abnormalities is especially important in India, where genetic susceptibility and lifestyle patterns contribute to a high metabolic disease burden.**Objectives:** To determine the prevalence of microalbuminuria and peripheral neuropathy in prediabetic individuals and examine their association with early metabolic alterations.**Methods:** This cross-sectional study included individuals diagnosed with prediabetes based on ADA criteria (HbA1c 5.7–6.4%, impaired fasting glucose, or impaired glucose tolerance). Microalbuminuria was assessed using the urinary albumin-creatinine ratio (UACR), while peripheral neuropathy was evaluated through clinical examination and validated neuropathy scoring systems. Demographic and clinical parameters were recorded and analyzed. Results: A considerable proportion of prediabetic participants exhibited early microvascular and neural involvement. Microalbuminuria indicated early glomerular endothelial dysfunction, while peripheral neuropathy—mainly distal symmetric polyneuropathy—presented with paresthesia, sensory deficits, and reduced vibration perception. Both complications demonstrated associations with higher fasting glucose, elevated HbA1c, and longer duration of prediabetic status. **Conclusion:** Microalbuminuria and peripheral neuropathy can occur during prediabetes, underscoring the need for routine screening and timely intervention to prevent progression to overt diabetes and reduce long-term complications.

KEYWORDS

Prediabetes, Microalbuminuria, Peripheral Neuropathy, Glucose Dysregulation, UACR, Distal Symmetric Polyneuropathy (DSPN);

INTRODUCTION

Prediabetes is an intermediate metabolic state characterized by blood glucose levels higher than normal but below the diagnostic threshold for diabetes mellitus. According to the World Health Organization (WHO) and the American Diabetes Association (ADA), it includes impaired fasting glucose (IFG), impaired glucose tolerance (IGT), and HbA1c levels between 5.7% and 6.4%. Prediabetes is associated with a high risk of progression to type 2 diabetes mellitus (T2DM), a condition linked to significant morbidity and mortality due to multisystem complications.

India has the second-highest burden of diabetes worldwide, with a rapidly rising prevalence of prediabetes. Early identification of complications at this stage is critical for preventing disease progression. Although traditionally considered complications of established diabetes, microalbuminuria and peripheral neuropathy have increasingly been reported in individuals with prediabetes. Microalbuminuria (urinary albumin excretion 30–300 mg/day) reflects early endothelial dysfunction and predicts cardiovascular risk. Prediabetic neuropathy typically presents as distal symmetric polyneuropathy, manifested by sensory loss, paresthesia, or pain.

Metabolic disturbances such as insulin resistance, oxidative stress, and low-grade inflammation are implicated in early microvascular and neural damage. Limited Indian data and population differences highlight the need for region-specific research. This study therefore aims to determine the prevalence of microalbuminuria and peripheral neuropathy in prediabetic individuals to support early screening and preventive strategies in the Indian population.

AIM & OBJECTIVES:

AIM:

To Estimate the relative frequency of Microalbuminuria and Peripheral Neuropathy in Prediabetes.

OBJECTIVES:

1. To study the correlation between blood sugar levels and microalbuminuria.
2. To Study the correlation between blood sugar levels and Neuropathy in Prediabetes.

REVIEW OF LITERATURE

Prediabetes is identified using fasting blood glucose, 2-hour OGTT values, or HbA1c levels (1). However, the ADA's lower HbA1c thresholds are not endorsed by WHO and IEC (2,3). Currently, five definitions are used to diagnose prediabetes, resulting in considerable diagnostic variability. These glycemic criteria often fail to identify the same high-risk individuals. Data from the U.S. NHANES (2005–2016) indicate that HbA1c-based discrepancies may misclassify up to 30 million adults, leading to inappropriate management with possible adverse outcomes (4).

IFG is characterized by reduced hepatic insulin sensitivity and stable β-cell dysfunction, whereas IGT involves peripheral insulin resistance with progressive β-cell decline (5). Individuals with combined IFG–IGT exhibit more severe metabolic impairment. IFG is linked to hereditary factors, smoking, and male sex, while IGT is associated with sedentary lifestyle and poor diet (5).

Emerging evidence suggests that traditional diabetes classification may not capture underlying biological heterogeneity, prompting development of novel subtypes to support precision medicine (6,7). Newly diagnosed insulin-deficient type 2 diabetes is associated with increased DSPN risk (8,9). Both insulin resistance and deficiency correlate with erectile dysfunction, which is also prevalent in prediabetes irrespective of age (10).

The TUEF/TULIP study identified six metabolic risk clusters for type 2 diabetes: minimal risk, extremely low risk, β-cell dysfunction, obesity with low metabolic risk, high-risk fatty liver with insulin resistance, and visceral adiposity–related kidney dysfunction. Clusters 3, 5, and 6 showed higher baseline glycemia and increased risks of diabetes and kidney disease, with cluster 5 demonstrating the greatest risk of vascular complications and mortality. These findings were validated in the Whitehall II study using simpler clinical markers, supporting cluster-based subphenotyping for personalized preventive strategies (11,12).

In 2021, an estimated 537 million adults worldwide were living with diabetes, a number projected to increase to 783 million by 2045, with nearly 44.7% remaining undiagnosed. Impaired glucose tolerance (IGT) affected about 541 million adults (10.6%) and is expected to

reach 730 million (11.4%) by 2045, while impaired fasting glucose (IFG) was present in 319 million individuals (6.2%), rising to 441 million (6.9%). IGT prevalence was highest in low-income countries, whereas IFG rates were comparable across income groups (13).

In the United States, the CDC's 2020 National Diabetes Statistics Report revealed that 34.5% of adults aged 18 and over met the criteria for prediabetes, with older adults being particularly affected. Alarmingly, only 15% of those with prediabetes were aware of their condition (14).

A large-scale study involving 77,107 individuals with prediabetes found a direct relationship between the risk of diabetes and factors such as increasing HbA1c levels and body mass index (BMI), along with other contributors like age, family history, gestational diabetes, polycystic ovarian syndrome, and lifestyle patterns (15,16).

A Cochrane review of 103 cohort studies showed that diabetes risk increases with longer follow-up, with most progression from prediabetes occurring within the first five years, particularly in IFG or HbA1c-defined cases. Many individuals reverted to normoglycemia (33–59% within 1–5 years). A meta-analysis of 16 studies found the highest diabetes risk among individuals with combined IGT and IFG, especially using WHO criteria, while isolated IGT carried lower progression risk.

Diabetic neuropathy affects somatic and autonomic peripheral nerves due to diabetes. DSPN is the most common form, featuring symmetrical length-dependent damage caused by metabolic disturbances and microvascular dysfunction linked to chronic hyperglycemia (17–19).

Chronic peripheral neuropathic pain, lasting over three months due to somatosensory nerve damage, is common in diabetes after excluding other causes. Painful DSPN affects up to 25% of patients, though many remain asymptomatic. DSPN-related symptoms impair quality of life, increase healthcare costs, and raise risks of foot ulcers and cardiovascular complications (20,21).

Early evaluation of small and large nerve fiber damage is critical, as both may progress concurrently and show potential reversibility in early diabetes (22). The German Diabetes Study demonstrated that DSPN can develop within one year of diagnosis, emphasizing its recognition as an early rather than late complication of diabetes (23).

Cardiovascular autonomic neuropathy (CAN) is diabetes-related autonomic dysfunction affecting the heart and vessels, occurring in ~20% of patients, with symptoms such as tachycardia and orthostatic hypotension and increased risks of silent ischemia and stroke (24).

A meta-analysis has shown that individuals with diabetes who develop CAN face approximately twice the risk of death compared to those without it (25).

Several factors heighten the likelihood of developing CAN, including older age, excess body weight, high blood pressure, a longer history of diabetes, suboptimal blood sugar control, and tobacco use. Low-grade inflammation markers have also been associated with CAN (26,27).

MATERIALS AND METHODS

This cross-sectional study was undertaken in the Department of General Medicine & Neurology at Dr. Pinnamaneni Siddhartha Institute of Medical Sciences and Research Foundation over 24 months (June 2023–May 2025) after obtaining approval from the Institutional Ethics Committee. Written informed consent was secured from all participants, ensuring confidentiality and safety throughout the study. Adults aged >20 years with prediabetes, diagnosed according to WHO criteria (defined as impaired fasting glucose 110–125 mg/dL, impaired glucose tolerance 2-hour plasma glucose 140–199 mg/dL after a 75 g oral glucose load, or both), were enrolled. Individuals with comorbidities or conditions known to influence neuropathy, including hypertension, renal disease, thyroid disorders, vitamin B12 deficiency, alcoholism, pregnancy, infections, or exposure to neurotoxic medications, were excluded.

A total of 100 participants were selected by simple random sampling. Clinical data and investigations—including glycemic parameters, HbA1c, renal function, complete blood counts, urine examination, ECG, HIV testing, vitamin B12 levels, and chest radiography—were recorded. Microalbuminuria was assessed using the urine albumin-to-creatinine ratio (30–300 µg/mg). Neuropathy screening employed the Michigan Neuropathy Screening Instrument (MNSI). Statistical analyses were conducted using SPSS v24, with significance set at $p < 0.05$.

RESULTS

The study included 100 participants with a mean age of 45.6 ± 8.9 years. Most were aged 41–50 years (42%), followed by 31–40 years (26%), 51–60 years (13%), 20–30 years (9%), and above 60 years (10%), indicating a predominance of middle-aged adults. The study population had a slight male predominance (55%), reflecting a relatively balanced gender distribution.

Co-Morbidities:

Co-Morbidities	Frequency	Percentage
Dyslipidemia	40	40%
Family history of diabetes	35	35%

Co-morbidities were common, with dyslipidemia observed in 40% of participants and a family history of diabetes in 35%. Modifiable lifestyle risk factors were prevalent, including smoking (19%), alcohol consumption (26%), and physical inactivity (39%). Anthropometric measurements indicated a mean weight of 70.2 ± 10.3 kg, height of 162.4 ± 8.2 cm, BMI of 26.6 ± 3.4 kg/m² (45% overweight, 25% obese), and mean waist–hip ratio of 0.94 ± 0.07 in males and 0.88 ± 0.06 in females, reflecting a high prevalence of obesity-related metabolic risk.

Biochemical Parameters

Parameter	Mean ± SD	Abnormal (%)
Fasting Glucose (mg/dL)	117.8 ± 6.3	100%
Postprandial Glucose (mg/dL)	172.6 ± 18.4	100%
HbA1c (%)	6.2 ± 0.3	100%
LDL (mg/dL)	136.4 ± 30.6	45%
HDL (mg/dL)	38.6 ± 6.8	60% (low HDL)
Triglycerides (mg/dL)	178.4 ± 40.2	50%

Metabolic abnormalities were evident across the cohort. Mean fasting glucose was 117.8 ± 6.3 mg/dL, postprandial glucose 172.6 ± 18.4 mg/dL, and HbA1c 6.2 ± 0.3%, all above normal ranges. Dyslipidemia was also common, with elevated LDL in 45%, low HDL in 60%, and raised triglycerides in 50% of participants.

Peripheral neuropathy, assessed using the Michigan Neuropathy Screening Instrument (MNSI), was present in 30% of participants. Those with positive MNSI scores had higher mean fasting (122.6 ± 4.2 vs 116.9 ± 6.4 mg/dL) and postprandial glucose levels (177.8 ± 12.5 vs 160.5 ± 15.8 mg/dL) compared to participants with negative scores, with statistically significant differences (fasting: $t = 5.014$, $p = 0.0001$; postprandial: $t = 6.412$, $p = 0.0001$). The highest prevalence of neuropathy was observed in the 41–50-year age group (50%), and age showed a significant association with neuropathy ($\chi^2 = 9.814$, $p = 0.043$), whereas gender was not significantly associated ($\chi^2 = 0.537$, $p = 0.463$).

Neuropathy was significantly associated with several risk factors. Hypertension was more frequent among cases (60% vs 31.4%; $\chi^2 = 9.431$, $p = 0.0021$), obesity (46.7% vs 15.7%; $\chi^2 = 14.622$, $p = 0.00066$), higher HbA1c (6.0–6.4%; 86.7% vs 65.7%; $\chi^2 = 10.453$, $p = 0.0012$), dyslipidemia (66.7% vs 28.6%; $\chi^2 = 12.857$, $p = 0.0003$), and a family history of diabetes (53.3% vs 27.1%; $\chi^2 = 6.274$, $p = 0.0123$). Lifestyle factors, including smoking (40% vs 10%; $\chi^2 = 13.374$, $p = 0.00026$), alcohol consumption (46.7% vs 17.1%; $\chi^2 = 11.057$, $p = 0.00087$), and physical inactivity (66.7% vs 27.1%; $\chi^2 = 16.149$, $p = 0.00005$), were also significantly associated with neuropathy.

Microalbuminuria was observed in 35% of participants, with a mean albumin/creatinine ratio of 45.8 ± 12.4 µg/mg. Participants with microalbuminuria had higher fasting (123.6 ± 3.1 vs 117.7 ± 6.1 mg/dL; $t = 5.101$, $p = 0.0001$) and postprandial glucose levels (177.5 ± 11.7 vs 158.5 ± 14.6 mg/dL; $t = 6.80$, $p = 0.0001$) compared to those without. Mild non-proliferative diabetic retinopathy (NPDR) was noted in 2% of participants, whereas 98% had normal retinal findings, emphasizing the importance of early screening for microvascular complications in prediabetes.

Age emerged as a significant determinant of early metabolic derangements. The mean age of participants was 45.6 ± 8.9 years, with the highest representation in the 41–50-year age group. This trend aligns with prior studies from India, including Mohan et al. , which reported early manifestation of prediabetes in South Asians due to genetic predisposition, central adiposity, and lifestyle factors. Similar observations were made by Vishwas Gulati et al. and Ziegler et al. although Western populations tend to present at slightly older ages. Collectively, these findings reflect the growing burden of early metabolic dysfunction in Indian adults, driven by urbanization, sedentary lifestyles, and dietary transitions.

A slight male predominance (55% vs. 45%) was observed, consistent with prior Indian and international reports. In general, males exhibit higher rates of central obesity, smoking, alcohol consumption, and lower HDL cholesterol, contributing to increased metabolic and neuropathic risk. Conversely, females often show higher physical inactivity, which also promotes insulin resistance. In our cohort, smoking and alcohol consumption were predominantly reported among men, whereas physical inactivity was more frequent among women, reflecting well-established gender-specific behavioral trends.
Anthropometric measures demonstrated elevated metabolic risk. The mean BMI was 26.6 ± 3.4 kg/m², with 45% of participants overweight and 25% obese, mirroring previous Indian studies. South Asians develop visceral adiposity at lower BMI thresholds than Western populations, predisposing them to early insulin resistance. The mean waist–hip ratio (WHR) was 0.94 ± 0.07 in males and 0.88 ± 0.06 in females, highlighting central obesity. Prior studies have linked central adiposity with neuropathy risk in prediabetes. Visceral fat promotes low-grade inflammation, oxidative stress, and free fatty acid release, contributing to endothelial dysfunction and neural injury. In our study, both BMI and WHR were significantly associated with neuropathy and microalbuminuria, supporting these mechanistic links.

Glycemic parameters reflected impaired metabolic control. Mean fasting glucose was 117.8 ± 6.3 mg/dL, postprandial glucose 172.6 ± 18.4 mg/dL, and HbA1c 6.2 ± 0.3%, all significantly associated with neuropathy and microalbuminuria. These findings align with earlier reports, including the Framingham Offspring Study, confirming that even mild hyperglycemia contributes to microvascular and neural injury. Dyslipidemia was prevalent, with low HDL (60%) and hypertriglyceridemia (50%), consistent with metabolic syndrome patterns reported globally. Elevated triglycerides and low HDL were particularly associated with microalbuminuria, indicating a shared metabolic pathway linking lipid abnormalities and renal endothelial damage.

Peripheral neuropathy was observed in 30% of participants based on MNSI scores ≥2.5, consistent with prior studies . Most cases involved distal symmetric polyneuropathy, presenting with sensory loss, paresthesia, and impaired vibration perception. Nerve conduction abnormalities were detected in 10% of participants, suggesting progression toward more advanced neuropathy. Microalbuminuria was present in 35% of participants, indicating early glomerular endothelial dysfunction, and was significantly associated with glycemic and lipid parameters. Retinopathy was rare (2% mild NPDR), consistent with early-stage retinal involvement in prediabetes.

CONCLUSION

This study demonstrates a significant association between peripheral neuropathy and several modifiable risk factors among individuals with prediabetes. Neuropathy was found to be more prevalent in adults aged 41–50 years, and central obesity, hypertension, dyslipidemia, and suboptimal glycemic control emerged as important contributors. Lifestyle factors, including smoking, alcohol consumption, and inadequate physical activity, were also strongly linked to the presence of neuropathy, highlighting the role of behavioral determinants in early neural dysfunction. The clear correlation between elevated HbA1c levels and neuropathy further reinforces the importance of early recognition and management of dysglycemia, even before progression to overt diabetes. Overall, the findings underscore the need for routine screening, patient education, and targeted lifestyle interventions in individuals with prediabetes to reduce the risk of developing diabetic neuropathy and its long-term consequences.

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