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Original research article

Microalbuminuria as a marker of kidney disease

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Abstract

Patients with diabetes mellitus type II may have a pre-diabetic phase known as "period" prior to the onset of the condition. Insulin resistance is a key factor in the pathophysiology of pre-diabetes. This study aims to examine the prevalence of microalbuminuria in a non-diabetic population with elevated metabolic risk and determine if there is any association between microalbuminuria and data related to glucose metabolism.

Keywords: Microalbuminuria, marker, kidney disease, uric acid, creatinine

Introduction

Prior to the onset of type II diabetes mellitus (DM2), individuals may experience a phase known as pre-diabetes. Insulin resistance is the underlying mechanism contributing to the pathophysiology of pre-diabetes [1-3]. The condition is distinguished by a diminished cellular absorption of glucose in response to insulin, necessitating increased secretion of this hormone by the pancreas in order to sustain normal glucose levels, therefore resulting in a state of hyperinsulinemia. The user's text is not clear. Please provide more information or rephrase your question. The homeostasis model assessment of insulin resistance index (HOMA-IR) is a commonly employed test for the evaluation of insulin resistance. This index assesses the levels of endogenous insulin and glucose, and is widely regarded as a reliable and precise tool for such assessment [3-6].

The pre-diabetes state may be associated with an elevated risk of problems even before the transition to type 2 diabetes. According to a research conducted by Rosenbaum *et al*. [7] Microalbuminuria was observed in individuals with pre-diabetes. The study further indicated that microalbuminuria served as an indicator of endothelial dysfunction, rather than just being associated with nephropathy. A separate research demonstrated a statistically significant increase in microalbuminuria among persons diagnosed with pre-diabetes in comparison to a control group with normal glycemic levels [8].

The presence of microalbuminuria in non-diabetic people has been shown to be associated with insulin resistance [9, 10]. The pathophysiology of microalbuminuria in individuals with pre-diabetes has not been definitively established. There is a putative association between hyperglycemia and renal damage, as well as a potential direct impact of insulin resistance on numerous organs, leading to endothelial impairment [11]. The findings of a study conducted on the general population indicate a correlation between microalbuminuria and mortality due to both cardiovascular and non-cardiovascular causes. These results further support the notion that microalbuminuria may serve as an indicator of endothelial damage [12].

In a clinical investigation conducted at our medical facility, it was shown that 68% of a group exhibiting heightened metabolic risk displayed pre-diabetic conditions [13]. Therefore, with the focus on the prevention of Type 2 Diabetes Mellitus (T2DM), it is imperative to evaluate the potential presence of endothelial damage within this specific demographic.

Therefore, this study was conducted in order to demonstrate the prevalence of microalbuminuria in a population without diabetes but with a high metabolic risk. Additionally, the study aimed to assess any potential association between microalbuminuria and data related to glucose metabolism.

Materials and Methods

30 people were selected randomly with microalbuminuria and 30 people were selected randomly without. The participants underwent oral glucose tolerance testing (GTT) two hours after consuming 75 g of glucose. Additionally, their serum glucose and insulin levels were measured after an 8-hour fasting period. These measurements were used to calculate the HOMA-IR index (fasting blood glucose (mmol/L) x fasting insulin (one/L)/22.5), with reference values of ≤ 3.4. The user's text is too short to be rewritten in an academic manner. The blood concentrations of total cholesterol, HDL-cholesterol, LDL-cholesterol, triglycerides, and creatinine were assessed following a 12-hour period of fasting using established protocols. The creatinine clearance was determined using the simplified Modification of Diet

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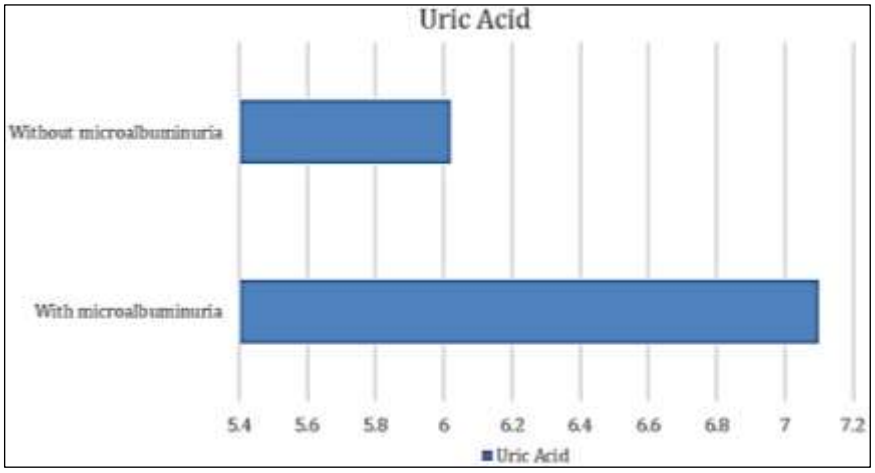
in Renal Disease (MDRD) formula. The examination of microalbuminuria was conducted on a 24-hour urine sample using the chemiluminescence technique, with a normal result of < 20 µg/minute.

Results

Table 1: Comparison with normal vs abnormal microalbuminuria

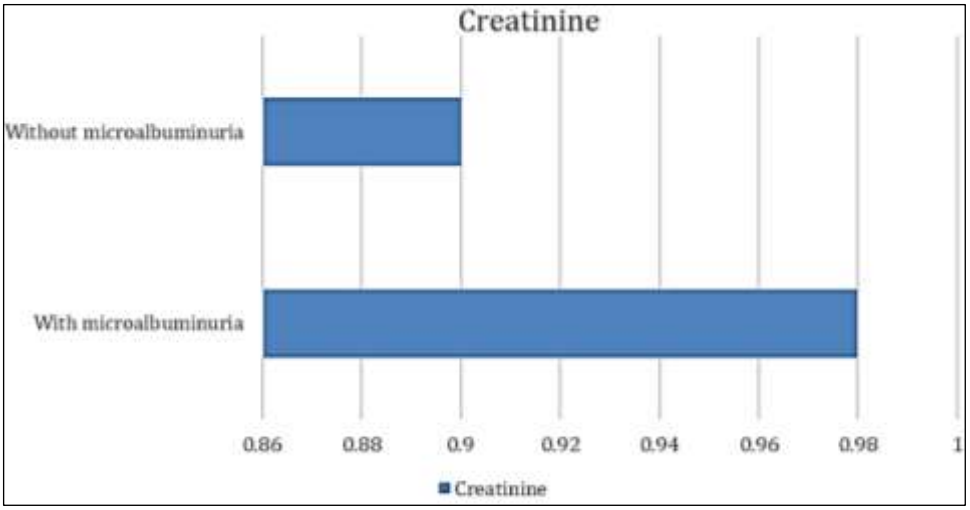
	Without microalbuminuria	With microalbuminuria
Age	51.56 ±6.74	52.96 ± 3.79
BMI	27.93±2.47	29.78± 3.73
HC	102.39±1.38	1.19±2.46
WC	38.37 ±4.49	42.38±4.38
HTN	14	19
Dyslipidemia	21	26
Fasting Glucose	92.39 ± 3.45	104.39±9.34
GTT	136.38±3.55	152.48±9.49
Total cholesterol	188.39±23.40	221.39±3.49
HDL	52.49±3.49	41.34±1.39
LDL	125.39±1.29	139.39±5.30
Triglycerides	132.49±4.4	135.59±4.27
Creatinine	0.9±01	0.98±0.2
Uric acid	6.02±0.1	7.1±0.42
Microalbuminuria levels	4.9±0.02	61.39±3.40



Graph 1: Uric acid levels comparison

Table 1: Uric acid levels comparison

Uric Acid	Without microalbuminuria	With microalbuminuria	r	p-value
	6.02	7.1	0.59	<0.001



Graph 2: Ceatinine comparison

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Table 3: Creatinine comparison

	Without microalbuminuria	With microalbuminuria	r	p-value
Creatinine	0.9	0.98	0.29	<0.001

Discussion

Microalbuminuria can be regarded as a distinct cardiovascular risk factor that is independent of traditional atherogenic variables, including blood pressure, glucose metabolism, dyslipidemia, and smoking. It is associated with a 2.3-fold increase in the likelihood of experiencing cardiovascular events. [18]. The criterion of normalcy in this context is established by considering individuals diagnosed with diabetes mellitus who have microalbuminuria levels that may accurately forecast their likelihood of developing diabetic nephropathy. Within the aforementioned healthy population, a range of 3 to 15% exhibit microalbuminuria levels equal to or exceeding 15 µg/min. This raises inquiries regarding the pathogenic significance of microalbuminuria in individuals without diabetes. Several studies have demonstrated that a higher level of HDL-cholesterol (HDL-C) in individuals aged 18 and 19 is associated with a reduced risk of coronary artery disease (CAD) [14-21]. However, there is a lack of research evaluating whether HDL-C acts as a protective factor against the development of nephropathy. Nonetheless, studies have indicated that individuals without nephropathy tend to have higher levels of HDL-C compared to those with nephropathy. The age range under consideration is between 22 and 27 years.

Based on histopathological, epidemiological, and experimental evidence, it is suggested that dyslipidemia has the potential to initiate glomerular damage and play a role in the course of renal disease. Multiple research have demonstrated a correlation between plasma lipoproteins and renal impairment in individuals with type 2 diabetes and microalbuminuria. These investigations have found a favourable link between microalbuminuria and elevated levels of lipoproteins that include apolipoprotein E. A subsequent analysis conducted on a specific portion of the ARIC (Atherosclerosis Risk in Communities) study aimed to determine the correlation between plasma lipids and decline in renal function. The findings of this analysis indicated that elevated levels of triglycerides and HDL-cholesterol were associated with an increased likelihood of kidney dysfunction, whereas LDL-cholesterol did not demonstrate a predictive relationship. The processes behind dyslipidemia in the course of glomerular disease are comparable to those implicated in atherosclerosis.

Over the past decade, there has been a resurgence of interest in uric acid as a possible mediator of endothelial dysfunction and renal disease. There exists a correlation between serum uric acid levels and unfavourable cardiovascular outcomes within the general population. Animal studies have implicated uric acid as a potential contributor to dysfunction and endothelial inflammation, as well as a factor in the advancement of renal disease. A prospective research was conducted over a period of five years, involving a sample of 1,743 Korean males. The investigation utilised multivariate analysis to determine the association between high blood uric acid levels and the likelihood of developing microalbuminuria. The findings of the study indicated that high serum uric acid levels were identified as an independent risk factor for the development of microalbuminuria.

Currently, there is a lack of pathophysiological elucidation on the association between microalbuminuria and serum uric acid levels, unless it may be attributed to the reduction in renal clearance of creatinine, which therefore affects the clearance of uric acid.

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Conclusion

Based on the findings of this study, it is recommended that the evaluation of microalbuminuria in individuals with pre-diabetes or metabolic risk factors should be considered as a potential indicator of early-stage nephropathy.

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