

Science

INTERNATIONAL JOURNAL OF RESEARCH – GRANTHAALAYAH A knowledge Repository



PREVALENCE OF MICROALBUMINURIA IN TYPE - 2 DIABETES MELLITUS: A HOSPITAL BASED STUDY

Dharamveer Yadav¹, Bhawani Kochar¹, Ankur Mathur¹, Arun Chougle²

¹ Department of Biochemistry, SMS Medical College and Hospital, Rajasthan University of Health Sciences, Jaipur, India

² Department of Radiological Physics, SMS Medical College and Hospital, Rajasthan University of Health Sciences, Jaipur, India

Abstract

Aim: Microalbuminuria (MAU) has been shown to be a risk factor for nephropathy in patients with type 2 diabetes. In this study, we aimed to explore the association between MAU and other risk factors in the development of diabetic nephropathy with type 2 diabetes.

Methods: Five twenty one patients with type 2 diabetes were recruited in this study. Medical records were used to collect data of age, duration of diabetes, Body Mass Index (BMI) and history of hypertension. Blood samples were collected after 12 hours overnight fasting to estimate fasting blood glucose (FBG), Glycosylated haemoglobin (HbA1C), serum creatinine, and serum uric acid. MAU was quantified using the dipstick method in early morning urine samples.

Results: Out of 521 subjects 37.5% (186 subjects) were found to be suffering from MAU. Systolic and diastolic blood pressure (p<0.001), HbA1C (p<0.001) and fasting blood glucose (p<0.001) were significantly higher in subjects with MAU as compared to normoalbuminuria subjects. Further, on multiple logistic regression analysis it was observed that duration of diabetes, systolic and diastolic blood pressure, serum creatinine and FBG are associated with MAU.

Conclusion: The overall prevalence of the MAU was high in the present study, which necessitates for early detection of nephropathy to reduce the burden of diabetes induced kidney disease in the future.

Keywords: Type-2 Diabetes Mellitus; Glycosylated Haemoglobin; Blood Glucose; Diabetic Nephropathies.

Cite This Article: Dharamveer Yadav, Bhawani Kochar, Ankur Mathur, and Arun Chougle. (2017). "PREVALENCE OF MICROALBUMINURIA IN TYPE - 2 DIABETES MELLITUS: A HOSPITAL BASED STUDY." *International Journal of Research - Granthaalayah*, 5(12), 217-222. https://doi.org/10.29121/granthaalayah.v5.i12.2017.496.

1. Introduction

Type 2 diabetes mellitus is one of the major causes of the mortality and morbidity in the world [1]. It has been predicted that world-wide the prevalence of diabetes in adults would increase to 5.4 % by the year 2025 as compared to the prevalence rate of 4.0% reported in 1995. Consequently the number of adults with diabetes in the world would rise from 135 million to 300 million in the year 2025 as compared to numbers of 1995 [2]. It is expected that much of this increase in prevalence rate will occur in developing countries. While 42% increase is expected in developed countries whereas 170% increase is expected in the developing countries. In the latter, most of the diabetic patients are in the age group of 45-64 years while in developed countries, most of them are ≥ 65 years [3]. Therefore, diabetic patients in developing countries are even more vulnerable to develop the microvascular complications of diabetes, including diabetic nephropathy and that too at an early age of life.

Diabetic nephropathy is the leading cause of end stage renal disease worldwide [4,5] and moreover, MAU is considered to be an early stage of diabetic nephropathy [6, 7,8,9]. Further MAU is often the first sign of renal involvement predicting overt nephropathy [10]. For this reason measurement of urine albumin is often used as a sensitive marker and predictor of overt nephropathy in patients with diabetes [11]. Monitoring MAU and other risk factors associated with this condition is important to take measures to prevent or postpone overt nephropathy.

The aim of this study was to evaluate the prevalence of MAU and renal impairment in patients with type 2 diabetes mellitus. The result could be important in increasing awareness of primary physicians towards the importance of regular urinary albumin screening in order to improve medical care of diabetic patients.

2. Methods

The present study group comprised of 521 type-2 diabetic patients attending the Department of Medicine and Department of Endocrinology of our institute during the period from September 2016 to January 2017. Type 2 diabetes was diagnosed according to American Diabetes Association revised criteria 2016[12]. Patients with incomplete record, urinary tract infection, pregnant woman and patients with acute infections and patients having proteinuria > 300 mg/day were excluded from the study as we have separately reported on the prevalence of MAU. We used subject medical records to obtain information regarding the gender, age, duration of diabetes, height and weight (to estimate BMI), history of hypertension and medication used. Patients were categorised as being hypertensive if they were on antihypertensive treatment or if they had a systolic blood pressure >130 mmHg and/or diastolic blood pressure >85 mmHg. A blood sample was collected after an overnight fasting of 12 hours and the blood glucose, HbA_{1C}, serum creatinine, serum uric acid levels were estimated with commercially available kits by fully auto analyser. Quantitative estimation of fasting blood glucose was done by Glucose oxidaseperoxidase end point method[13] andHbA_{1C} estimation was done by Latex agglutination inhibition assay. Estimation of serum uric acid was done by uricase method[14] whereas serum creatinine levels were measured by Jaffe's method[15]. MAU was assessed using the dipstick method by Clinitek status instrument in urine samples collected in the morning.

Data analysis was conducted using the Statistical Package for Social Sciences (SPSS) for windows version 24.0 (Free trial version). In addition, multiple logistic regression analysis was used to determine association between MAU and other risk factors.

3. Results

The study was conducted on 521 patients of type 2 diabetes and it was observed that MAU was present in 186 (35.70%) patients. The results of present study are depicted in Table 1.

Parameters	Normoalbuminuria	Microalbuminuria group	P Value
	group (n=335)	(n=186) [Mean ± SD]	
	[Mean ± SD]		
Age (years)	56.22 ± 11.18	60.05 ± 9.19	< 0.001
BMI (kg/M^2)	24.41 ± 1.21	24.34 ± 1.31	0.537
Systolic Blood pressure	133.29 ± 4.16	135.95 ± 5.00	< 0.001
(mmHg)			
Diastolic Blood pressure	83.34 ± 3.23	86.30 ± 3.69	< 0.001
(mmHg)			
FBG (mg/dl)	176.28 ± 19.97	200.01 ± 28.36	< 0.001
HbA _{1C} (%)	9.05 ± 1.88	9.84 ± 1.90	< 0.001
Creatinine (mg/dl)	1.13 ± 0.32	1.35 ± 0.24	< 0.001
Uric acid (mg/dl)	5.37 ± 1.35	5.93 ± 1.48	< 0.001

Table 1: Clinical and biochemical characteristics of study subjects

Table 1 show the clinical and biochemical characteristics of the study subjects. In the present study, the mean age was observed to be 56.22 ± 11.18 years and 60.05 ± 9.19 years in normoalbuminuric and microalbuminuric group respectively. The microalbuminuric subjects had significantly higher age as compared with the normoalbuminuric subjects (p<0.001). On further analysis, it was observed that the microalbuminuric subjects had significantly increased systolic and diastolic blood pressure as compared to the normoalbuminuric subjects (p < 0.001). It was also observed that fasting blood glucose, HbA_{1C}, serum creatinine and serum uric acid were found to be higher in microalbuminuric group (p < 0.001) whereas BMI was statistically insignificant and slightly lower in subjects with MAU (p = 0.537).

The prevalence of MAU in relation to duration of diabetes is depicted in table 2.

Duration of Diabetes (in years)	Prevalence (%)	Odds Ratio (95% CI)	P Value
≤5	09/42 (21.4%)	-	
06-10	21/78 (26.9%)	1.3509 (0.5543 - 3.2923)	0.5082
11-15	48/125 (38.4%)	2.2859 (1.0062 - 5.1922)	0.0483
16-20	102/259 (39.38%)	2.3822 (1.0941 – 5.1866)	0.0288
>20	06/17 (35.29%)	1.5714 (0.3368 - 7.3324)	0.4843

Table 2: Prevalence of microalbuminuria in relation to duration of diabetes

CI= Confidence interval

In the present study, we observed that an increase in frequency of the MAU is directly related to the duration of diabetes. Taking duration of diabetes <5 years as the reference the odds ratio were 1.3, 2.2, 2.3 and 1.5 for duration of 6-10 years, 11-15 years, 16-20 years and >20 years respectively. The frequency of MAU continued to increase up to 15-20 years of diabetes (Fig. 1). Further, we found that the frequency of MAU decreases beyond 20 years of diabetes. The reason behind this might be less number of subjects in the group having duration of diabetes > 20 years and needs more in depth study.

The results of multiple logistic regression analysis using MAU as dependent variable are shown in Table 3.

Variables	B	Standard	P value	Odds ratio (95% CI)
		Error β		
Age (years)	0.019	0.012	0.130	1.019 (0.994-1.044)
BMI (kg/M^2)	0.005	0.101	0.963	1.005 (0.824-1.224)
Systolic Blood pressure	0.139	0.028	< 0.0001	1.149 (1.087-1.215)
(mmHg)				
Diastolic Blood pressure	0.286	0.040	< 0.0001	1.331 (1.231-1.439)
(mmHg)				
FBG (mg/dl)	0.039	0.006	< 0.0001	1.039 (1.028- 1.051)
$HbA_{1C}(\%)$	0.244	0.063	< 0.0001	1.277 (1.128-1.446)
Creatinine (mg/dl)	2.489	0.436	< 0.0001	12.043 (5.126 - 28.295)
Uric acid (mg/dl)	0.101	0.090	0.260	1.107 (0.928-1.320)

Table 3: Multiple logistic regression analysis using microalbuminuria as dependent variable

The other variables like age, BMI, systolic blood pressure, diastolic blood pressure, fasting blood glucose, HbA_{1C} , serum creatinine and serum uric acid were taken as continuous variables. All the clinical and biochemical parameters included in this study showed a significant association with MAU except age, BMI and serum uric acid.

4. Discussion

In the present study, we investigated the prevalence of MAU and its risk factors in 521 type 2 diabetic subjects. The evidence of nephropathy was observed in 186 subjects (35.70%). Gupta et al.[16] reported a prevalence of 26.2% in 65 type 2 diabetic North Indian non proteinuric patients. While John et al. [17] reported a prevalence of 19.7% from tertiary hospital in Vellore, South India and Vijay et al.[18] reported that 15% had proteinuria among 600 patients of type 2 diabetic in Chennai city. Klein et al. [19] in their study they found that the frequency of MAU was 29.2% of those taking insulin and 22.0% of those not taking insulin. Agarwal R et al.[20]observed that 30.2% had developed nephropathy among 3369 patients of type 2 diabetes. The variation in the prevalence can be attributed to factors such as geographical and other socioeconomic and environmental factors. This could be due to the renal ethnic variation, i.e. genetic or due to poor control of the diabetes, hypertension in the susceptibility to diabetic nephropathy.

The result of our multiple logistic regression suggests that fasting blood glucose, HbA_{1C}, systolic blood pressure, diastolic blood pressure and serum creatinine are significantly (p < 0.001) associated with MAU. In support of this, an earlier study by Gupta et al. [16] reported HbA_{1C} to be associated with MAU. Viswanathan et al.[21] found that the initial HbA_{1C} along with initial systolic blood pressure is an important factor for proteinuria. This study revealed a strong associated with high prevalence of MAU. Our results were in consistence with the finding of Rema et al. [22] and Ramchandra et al. [23] who found positive association of hypertension with diabetic nephropathy. Vijay et al. [18] reported duration of diabetes systolic and diastolic blood pressure, serum creatinine to be associated with diabetic nephropathy.

We could not find any significant association of BMI and serum uric acid with MAU in type 2 diabetic subjects. While a study by Agarwal R et al. [20] found 30% of patients with BMI developed nephropathy. Suryawanshi et al. [1] found positive correlation of serum uric acid and urine microalbumin in their study.

One of the limitations of our study was that it was not population based, which may introduce referral bias affecting the results.

5. Conclusion

In conclusion, the overall prevalence of MAU in type 2 diabetic subjects is 35.70%, Assessment of MAU and other risk factors can, therefore, be helpful in early identification of patients at risk of developing such complications to reduce the burden of diabetic kidney disease in the future.

References

- [1] Suryawanshi KS, Jagtap PE, Belwalkar GJ, Dhonde SP, Nagane NS, Joshi VS.To study serum uric acid and urine microalbumin in type-2 diabetes mellitus. SSRG Int J Med Sci 2015; 2(3):24-9.
- [2] King H, Aubert RE, Herman WH. Global burden of Diabetes, 1995-2025: Prevalence, numerical estimates and projections. Diabetes Care 1998; 21: 1414-31.
- [3] Vishwanathan V. Type 2 diabetes and diabetic nephropathy in India- Magnitude of the problem. Nephrol Dial Transplant 1999; 14:2805-07.
- [4] Cordonnier D, Bayle F, Benhamou PY, Milongo R, Zaoni P, Maynard C et al. Future trends of management of renal failure in diabetics. Kidney Int suppl.1993; 41:S8–13.
- [5] US Renal Data System. 1989 Annual data report. Bethesda, MD: National Institute of Diabetes and Digestive and Kidney Diseases, 1989.
- [6] Mogensen CE, Steffes MW, Deckert T, Christiansen JS. Functional and morphological renal manifestations in diabetes mellitus. Diabetologia 1981; 21:89–93.
- [7] Viberti GC,Keen H.The patterns of proteinuria in diabetes mellitus: relevance of pathogenesis and prevention of diabetic nephropathy. Diabetes 1984; 33: 686-92.
- [8] Alzaid AA. MAU in patients with NIDDM: an overview. Diabetes Care 1996; 19:79–89.
- [9] Parving HH, Gall MA, Skott P, Jorgensen HE, Lokkegaard H, Jorgensen F et al. Prevalence and causes of albuminuria in non-insulin dependent diabetic patients. Kidney Int 1990; 41:758–62.
- [10] Jong PE, Hillege HL, Joan PS, Zeeuw D. Screening for MAU in the general population: A tool to detect subjects at increased risk for progressive renal failure in an early phase.Nephrol Dial Transpl 2003; 18:10-13.

- [11] Hiddo J, Heerspink L, Holtkamp F, Ravid M. Monitoring kidney function and albuminuria in patients with diabetes. Diabetes Care 2011; 34: 325-9.
- [12] American Diabetes Association. Standards of medical care in diabetes—2016. Diabetes Care. 2016; 39 (suppl 1):S1-106.
- [13] Trinder, P. Determination of blood glucose using an oxidase peroxidase system with a noncarcinogenic chromogen. JClin Pathol 1969; 22: 158-61.
- [14] Praetorius E, Poulson H. Enzymatic determination of uric acid; with detailed directions. Scand J Clin lab Inv 1953; 5(3): 273-80.
- [15] Bonsnes RW, Taussky HH. On the colorimetric determination of creatinine by the Jaffe reaction. J Biol Chem 1945, 158: 581-91.
- [16] Gupta DK, Verma LK, Khosla PK, Dash SC. The prevalence of MAU in diabetes: a study from north India. Diabetes Res Clin Pr 1991; 2: 125–8.
- [17] John L, Rao PS, Kanagasabapathy AS. Prevalence of diabetic nephropathy in non-insulin dependent diabetes. Indian J Med Res 1991; 94: 24–9.
- [18] Vijay V, Snehalatha C, Ramachandran A, Viswanathan M.Prevalence of proteinuria in non-insulin dependent diabetes. J Assoc Physician I 1994; 42:792–4.
- [19] Ronald Klein, Barbara EK, Scot E Moss. Prevalence of MAU in older -onset diabetes. Diabetes Care 1993; 16(10): 1325-30.
- [20] Agrawal R, Phogawat M, Agrawal RP. Prevalence of Nephropathy and Its Risk Factors in Type 2 Diabetes: A Tertiary -Care Hospital Based Study. RUHS J Health Sci 2016; 1(1): 20-3.
- [21] Viswanathan VV, Snehalatha C, Ramchandran A, Viswanathan M. Proteinuria in NIDDM in South India. Analysis of predictive factors. Diab Res Clin Pract 1995; 28: 41-6.
- [22] Rema M, Ponnaiya M, Mohan V. Prevalence of retinopathy in non-insulin dependent diabetes mellitus at a diabetes centre in Southern India. Diab Res Clin Pract 1996; 34: 29-36.
- [23] Ramchandran A, Snehalatha C, Satyavani K, Latha E, Sasikala R, Vijay V. Prevalence of vascular complications and their risk factors in type 2 diabetes. J Assoc Physician I 1999; 47: 1152-6.

*Corresponding author. *E-mail address:* dharam143s@ gmail.com