

Original Article

PREVALENCE OF MICROALBUMINURIA AS A MARKER OF INCIPIENT NEPHROPATHY IN TYPE 2 DIABETES PATIENTS AT SMIMER HOSPITAL, SURAT

Vilas U. Chavan¹, DVSS Ramavataram², Kinjal L. Miyani³, Hasit D. Lad⁴, S.D. Nilakhe⁴

Financial Support: None declared

Conflict of interest: None declared

Copy right: The Journal retains the copyrights of this article. However, reproduction of this article in the part or total in any form is permissible with due acknowledgement of the source.

How to cite this article:

Chavan VU, Ramavataram DVSS, Miyani KL, Lad HD, Nilakhe SD. Prevalence of Microalbuminuria As a Marker of incipient Nephropathy in Type 2 Diabetes Patients At SMIMER Hospital, Surat.. Natl J Community Med 2014; 5(4):468-73.

Author's Affiliation:

¹Associate Professor; ²Professor and Head; ³M.Sc. MLT student; ⁴Assistant Professor, Department of Biochemistry, Surat Municipal Institute of Medical Education & Research (SMIMER), Surat, Gujarat

Correspondence:

Dr. Vilas U. Chavan
E-mail: drvuchavan@yahoo.co.in

Date of Submission: 23-11-14

Date of Acceptance: 30-12-14

Date of Publication: 31-12-14

ABSTRACT

Background: Diabetic nephropathy (DN) is one of the most significant long-term complications in diabetes mellitus (DM). The study was primarily aimed to screen type 2 DM patients for renal damage and to find out the prevalence of MAU as marker of nephropathy in the SMIMER hospital, Surat.

Methods: We studied 82 known cases of type 2 DM and 50 healthy subjects as control. Plasma glucose, serum lipid profile, urea, uric acid, creatinine and urine albumin and creatinine were measured in subjects. Based on urine albumin and albumin:creatinine ratio (ACR) stages of nephropathy were diagnosed. Data was expressed as mean \pm SD and data was compared by using unpaired student 't' tests for independent sample.

Results: We found significantly higher levels of fasting and post prandial glucose ($P < 0.05$), urea ($P < 0.01$), uric acid ($P < 0.03$), urine albumin and ACR ($P < 0.05$) in DM patients compared to controls. We observed increased level of total cholesterol ($P < 0.05$), triacylglycerol ($P < 0.05$) and non significant decrease in HDL level ($P = 0.11$) in DM patients compared to controls. In DM patients the prevalence of normoalbuminuria, microalbuminuria and macroalbuminuria were 21.95%, 62.19% and 15.85% respectively.

Conclusion: Our study concludes that MAU is a reliable marker of DN in type 2 DM patients. MAU and dyslipidemia together may be more potent risk factor for complications in type 2 DM. Therefore regular screening for MAU is recommended for all asymptomatic DM patients for reducing cardiovascular risks and slowing the progression to end-stage renal disease to reduce the socioeconomic burden of DM.

Key words: Type 2 Diabetes mellitus, Microalbuminuria, Nephropathy, Prevalence, Lipid profile.

INTRODUCTION

Diabetes mellitus (DM) is now one of the most common non-communicable diseases globally. It is epidemic in many low and middle-income countries and major cause of blindness, renal failure, amputation, coronary artery, peripheral vascular disease and stroke (1, 2).

It is estimated that there are approximately 285 million people (6.4%) with diabetes worldwide in 2010 and this number is expected to increase to 438 million (7.7%) of the adult population by 2030. The largest increases will take place in the developing countries (1, 3). World Health Organisation (WHO) projects that diabetes will be the

7th leading cause of death in 2030 (2). India has highest number of people with diabetes in the world, according to the International Diabetes Federation (IDF); the disease affects more than 50 million Indians comprising 7.1 % of the nation's adult (20-79 age groups) population and kills about 1 million Indians per year (1). The high incidence in India is attributed to increased life expectancy in India, economic growth, lifestyle changes, intake of high calorie food and combination of genetic susceptibility (4, 5).

Diabetic nephropathy (DN) is one of the most significant long-term complications in terms of morbidity and mortality for individual with diabetes and is leading cause of chronic kidney disease in the United States. Diabetes is responsible for 20-40% of all end-stage renal disease (ESRD) cases in the United States (6- 8). The incidence of diabetes is increasing worldwide with subsequent increase in the incidence of DN.

Microalbuminuria (MAU) is an increased excretion of albumin in urine above physiological levels. MAU has been considered the first indication of renal injury in patients with diabetes (9). MAU is now widely recognized as a sign of abnormal vascular function and increased vascular permeability (10).

MAU is defined as a urinary albumin excretion (UAE) of 30- 300 mg/ day, when measured in a 24 hour urine collection. It is also defined as values between 20- 200 mg/L or 30 -300 mg/g, if measured with the use of the urinary albumin: creatinine ratio (ACR) in a spot or random urine samples (9,11). Level of urine albumin below these limit are considered normal, whereas any albumin excretion above this limits represents macroalbuminuria or clinical proteinuria (Table 1).

Currently, the National Kidney Foundation (NKF) recommends the use of spot urine ACR obtained under standardized conditions to detect MAU (13, 14). MAU is an independent risk factor for cardiovascular events, therefore, a strategy to detect early diabetic kidney disease by screening for albuminuria is an important step in diabetic kidney disease (10). Additionally there is remarkable lack of awareness among diabetic patients regarding DN. Based on present knowledge our study was primarily aimed to screen type 2 DM patients for renal damage and to find out the prevalence of MAU as marker of nephropathy in the SMIMER hospital, Surat. Secondary objectives were to measure the lipid

profile and other biochemical parameter in study subjects.

METHODS

Presence study was carried out in the department of Biochemistry at Surat Municipal Institute of Medical Education and Research (SMIMER), Surat, Gujarat, India, over a period of 6 months from January to June 2013. Study was approved by institutional ethical committee and informed consent was taken from all participants.

Subjects: Total 132 subjects of which 82 known cases of type 2 DM attending the Medicine department were taken for this study and 50 age and gender matched healthy subjects were taken as control. Control group comprised of relatives of patients and from general population. Patients were on oral hypoglycemic agents and attending for follow up treatment in diabetes. Selection of patients was random and no specific sampling criteria were applied. Patients having past history of hospitalisation for kidney diseases, other metabolic, cardiovascular diseases, tuberculosis, and thyroid disorders were excluded from study.

Laboratory analysis: Blood sample was collected after overnight fasting and spot urine sample was collected in morning (between 8.00 am – 9.00 am). Laboratory analysis was done in the Clinical Biochemistry laboratory on the same day by using commercially available kits on fully automated clinical chemistry analyzer Erba-XL 300 (Transasia Bio-Medicals Ltd. Mumbai, India). Plasma glucose was estimated by the Glucose oxidase/Peroxidase (GOD-POD) method (15). Estimation of total cholesterol, triglycerides and HDL cholesterol were measured by enzymatic method (Aspen Laboratories Pvt. Ltd; India) [16]. Other biochemical parameters were estimated by using commercial reagent kits (Pathozone Diagnostics, India), Urea by enzymatic (urease/glutamate dehydrogenase, kinetic) method [17], uric acid by the uricase/PAP (peroxidase coupled with 4-aminophenazone) enzymatic method [18], creatinine by modified Jaffe's fixed time kinetic method [19], sodium (Na⁺), potassium (K⁺) and chlorides (Cl⁻) collectively called as electrolytes were measured by ion selective electrode method [20] using Combisys-II, Eischweiler BGA plus E instrument (Eischweiler automatic analyzing systems, Eschweiler GmbH & Co. Germany). Urinary albumin was determined by immunoturbidimetry method (2) (Tu-

lip Diagnostics (P) Ltd. Goa, India). Urine was diluted as 1:50 with distilled water before urine creatinine analysis.

Statistical analysis: Data was expressed as mean ± SD. Comparisons of diabetic subjects with control was performed using unpaired student 't' tests for independent sample; a level of P < 0.05 was considered as statistical significant. Statistics were computed using GLM unisariate.

RESULTS

The present study comprised of 82 type 2 DM patients and 50 healthy controls (Table 2). The

study subjects were divided in to two groups according to age, group 1(age 20-40 years) and group 2 (age 41-60 years) In case of DM 26 (15 males and 11 females) were of 20-40 years age group and 56 (32 males and 24 females) were 40-60 years age group.

We found significantly higher values of fasting and post prandial glucose (P<0.05), urea (P<0.01), uric acid (P<0.03), urine albumin and ACR (P<0.05) in DM patients compared to controls (Table 3). We observed increased level of total cholesterol (P<0.05), triacylglycerol (P<0.05) and non significant decrease in HDL level ((P=0.11) in DM patients compared to controls.

Table 1. Cut off values used in literature for indicating normal, microalbuminuria (MAU) and macroalbuminuria (9, 11-13).

Terms	24-hour urine Sample	Spot morning/random urine sample		
	UAE (mg/24 hours)	UAC (mg/L)	ACR* (mg/g)	ACR* (mg/mmol)
Normal	<30	<20	<30	<3
Microalbuminuria	30 to 300	20 to 200	30 to 300	3 to 30
Macroalbuminuria	>300	>200	>300	>30

*ACR (mg/g) values are for both males and females (gender independent) (9, 12).

Table 2. Age and gender distribution of study subjects

Age Gender	20-40 year		40-60 year		Total
	Male	Female	Male	Female	
Diabetes mellitus	15	11	32	24	82
Normal subjects	17	5	21	7	50

Table 3: Baseline laboratory data of study subjects.

Parameter	Diabetic subjects (Mean ± SD) (n=82)	Control subjects (Mean ± SD) (n=50)	P value
Plasma FBS (mg/dl)	169.21±64.89	93.38 ±16.55	<0.05*
Plasma PPBS (mg/dl)	261.81±232.94	112±19.20	<0.05*
Serum Creatinine (mg/dl)	0.71±0.31	0.71±0.24	0.99
Serum Urea (mg/dl)	27.65± 11.09	22.20 ± 3.75	0.01*
Uric acid (mg/dl)	5.37±1.64	4.56±1.23	0.03*
Sodium (mmol/L)	137.65±4.72	141.58±20.09	0.09
Potassium (mmol/L)	4.19±0.8	9.51±26.41	0.06
Chloride (mmol/L)	96.31±6.17	101.46±3.29	0.21
Total Cholesterol (mg/dl)	195.5± 50.15	160.94 ± 21.35	<0.05*
Triglycerides (mg/dl)	190.73 ±73.47	132.96±42.90	<0.05*
HDL (mg/dl)	46.72 ± 10.46	60.40 ± 12.71	0.11
Urine albumin (mg/L)	66.39±84.70	9.02±4.39	<0.05*
Urine Creatinine (gm/L)	0.95±0.37	1.05±0.38	0.81
A/C ratio (mg/g)	79.34±109.74	9.75±5.84	<0.05*

Significant *

Table 4. Comparison of various biochemical parameters between DM patients and controls in group-1 and group-2.

	Diabetic sub- jects (Mean ± SD) (n=26)	Controls (Mean ± SD) (n= 22)	p-value	Diabetic sub- jects (Mean ± SD) (n=56)	Controls (Mean ± SD) (n=28)	p-value
FBS (mg/dl)	180.38±75.72	91.13±16.53	<0.05*	164.03±59.36	95.14±16.66	<0.05*
PPBS(mg/dl)	240±91.36	118.04±26.72	<0.05*	271.94±27.25	125.10±9.52	<0.05*
Creatinine (mg/dl)	0.67±0.25	0.7±0.261	0.71	0.73±0.34	0.72±0.23	0.92
Urea (mg/dl)	28.15±14.58	21.04±3.73	0.03*	27.42±9.18	21.32±3.83	<0.05*
Uric acid (mg/dl)	5.28±1.43	4.3±1.10	0.01*	5.41±1.73	4.75±1.31	0.08
Sodium (mmol/L)	137.65±5.20	137.45±29.15	0.97	137.66±4.53	144.82±6.84	0.05
Potassium (mmol/L)	4.19±0.97	4.25±0.72	0.81	4.18±0.77	13.65±35.00	0.31
Chloride (mmol/L)	94.76±5.79	101.22±3.16	0.07	97.03±6.26	101.64±3.43	0.23
Total Cholesterol (mg/dl)	185.96±53.82	163.45±22.28	<0.05*	199.92±48.21	158.96±20.79	<0.05*
Triglycerides (mg/dl)	180.46±66.03	122.81±34.88	<0.05*	195.5±76.77	140.92±47.37	<0.05*
HDL (mg/dl)	44.66±10.06	62.50±12.90	0.23	47.13±10.83	59.42±12.62	0.33
Urine Albumin (mg/L)	48.46±61.11	9.04±3.51	<0.001*	74.11±92.97	9±5.03	<0.001*
Urine creatinine (gm/L)	0.97±0.32	1.04±0.42	0.40	0.96±0.38	1.05±0.35	0.65
A/C ratio (mg/g)	56.66±68.11	10.16±5.96	<0.001*	89.87±118.32	9.41±5.82	<0.001*

Significant *

Table 5. Distribution and prevalence of DN in diabetes patients.

Stage of Nephropathy	Diabetic subjects	
	Number	Percentage
Normal	18	21.95%
Microalbuminuria	51	62.19%
Macroalbuminuria	13	15.85%
Total	82	100%

There was no significant change in electrolytes (sodium, potassium and chlorides) between cases and controls. We compared significant increase in level of total cholesterol (P<0.05), triacylglycerol (P<0.05) and non significant decrease in HDL level ((P=0.23), urine albumin and ACR (P<0.001) in each age group of cases compared to controls (Table 4).

Table 6. Distribution of study subjects as per diseases duration of DM.

	Diabetic subjects having duration of diseases < 5 Years (n=60)			Diabetic subjects having duration of dis- eases > 5 Years (n= 22)		
	Normoalbu- minuria	Microal- buminuria	Macroalbu- minuria	Normoalbu- minuria	Microalbu- minuria	Macroalbu- minuria
Number of patients	14 (23.33%)	42 (70%)	4 (6.66%)	4(18.18%)	9(40.99%)	9(40.99%)
Gender (female: male)	8F/6M	15F/27M	2F/2M	2F/2M	5F/4M	3F/6M
Urine albumin (mg/L)	26.64±3.27	54.88±27.23	331.75±26.18	24.25±3.94	152.52±70.89	386±73.88
Mean± SD						

In DM patients the prevalence of normoalbuminuria, microalbuminuria and macroalbuminuria were 21.95%, 62.19% and 15.85% respectively (Table 5). Out of 82 DM patients 60 patients had diseases duration < 5 year, out of which 14, (23.33%) had normoalbuminuria, 42 (70 %) had MAU and 4 (6.66%) had macroalbuminuria. 22 DM patients had diseases duration > 5 year, out of which 4 (18.18%) had normoalbuminuria, 9 (40.99 %) had MAU and 9 (40.99%) had macroalbuminuria (Table 6).

DISCUSSION

Diabetes is major cause of chronic kidney disease and is recognized as the most common causes of

end stage renal disease in the world both in developed and developing countries. (6, 22). It has been reported that 40% of adults with diagnosed or undiagnosed diabetes have some degree of chronic kidney disease (23, 24). Increased excretion of albumin is sensitive marker for chronic kidney disease due to diabetes. This stage of renal involvement was termed MAU or incipient nephropathy (13). DN is defined by increased urinary albumin excretion (UAE) in the absence of other renal diseases and categorized into three stages: microalbuminuria (MAU), macroalbuminuria and overt nephropathy or clinical nephropathy (table 1) (25).The rates of progression of newly diagnosed type 2 diabetics between the stages of normoalbuminuria, MAU,

macroalbuminuria and renal failure were 2-3% per year (26). DN is more prevalent among African Americans, Asians, and Native Americans than Caucasians (27).

Out of 82 diabetic subjects 64 subjects had increased excretion of albumin in urine which includes 51 patients (62.19%) had microalbuminuria 13 patients (15.85%) had macroalbuminuria and remaining 18 had normoalbuminuria. Our observation is supported by other studies (28-31). Kedam et al (28) observed 50% prevalence of MAU in cross sectional hospital study (28), and Thakkar et al (29) observed 54.09% of MAU prevalence in newly diagnosed type 2 DM in India. Similar observations were found in general population in China (30), UAE (31) and hospital based study in Pakistan (32) supports our observations.

We observed similar prevalence of macroalbuminuria (15.85%) in our study like Kanakamani et al. study (33) from hospital based study from north India, but they have screened patient by deep stick method and not quantitatively measured urine albumin unlike us.

In our study the prevalence of MAU in diabetic subjects is found higher compared to other studies in India (34-36). Higher prevalence in the present study may be due to the fact that most of the patients were with poor glycaemic control. A significant correlation was found between the prevalence of MAU and the duration of diabetes in our study supported by other studies (36-37)

We observed significant increase in total cholesterol, triacylglycerol and non significant decrease in HDL in DM patients. Dyslipidemia is common in diabetic patients (38) and further predisposes the diabetic patients to cardiovascular disease. Significant alterations in the lipid levels were found to be influenced by a glycaemic control. The MAU is independent risk factor for cardiovascular diseases in type 2 diabetic patients. (28). Al-Shaikh et al. study (32) observed cardiovascular diseases and retinopathy were significantly more in patients with MAU than those with normal microalbuminuria. Hence micro- or macroalbuminuria patients should undergo evaluation for assessment of renal function and the presence of other co-morbid conditions (25). Persistent hyperglycemia known to cause the impairment of renal functions and this is assessed by measuring using the serum urea, creatinine and urine albumin and ACR in our study.

Our study concludes that MAU is a reliable marker of DN in type 2 DM patients. We recommend that all patients with type 2 DM should be screened at the time of diagnosis and yearly thereafter. MAU and dyslipidemia together may be more potent risk factor for complications in type 2 DM. Patients with micro- and macroalbuminuria should undergo an evaluation regarding the presence of co-morbid conditions. Therefore regular screening for MAU is recommended for all asymptomatic DM patients for reducing cardiovascular risks and slowing the progression to end-stage renal disease to reduce the socio-economic burden of DM.

REFERENCES

1. Sicree R, Shaw J, Zimmet P. The global burden: Diabetes and impaired glucose tolerance. In: Gan D, editor. Diabetes atlas. 4th ed. International Diabetes Federation. Belgium: International Diabetes Federation; 2009. pp. 1-105.
2. Global status report on noncommunicable diseases 2010. Geneva, World Health Organization, 2011.
3. Danaei G, Finucane MM, Lu Y, Singh GM, Cowan MJ, Paciorek CJ et al. National, regional, and global trends in fasting plasma glucose and diabetes prevalence since 1980: systematic analysis of health examination surveys and epidemiological studies with 370 country-years and 2.7 million participants. *Lancet*, 2011, 378(9785):31-40.
4. Deepa R, Sandeep S, Mohan V. Abdominal obesity, visceral fat and type 2 diabetes- "Asian Indian Phenotype. In: Mohan V, Rao GHR (ed). Type 2 diabetes in South Asians: Epidemiology, Risk factors and Prevention. Jaypee Brothers Medical Publishers (P) Ltd, New Delhi 2006: 138-152.
5. Ramachandran A, Snehalatha C, Latha E, Manoharan M, Vijay V. Impacts of urbanisation on the lifestyle and on the prevalence of diabetes in native Asian Indian population. *Diabetes Res Clin Pract* 1999;44(3):207-13.
6. Reutens AT, Prentice L, Atkins R. The epidemiology of diabetic kidney disease. In: The epidemiology of diabetes mellitus, 2nd Edition, Ekoé J, et al., Editors. 2008, John Wiley & Sons Ltd: Chichester. p. 499-518.
7. National Institutes of Health. National Institute of Diabetes and Digestive and Kidney Diseases. International comparisons in 2007 Annual Data Report: Atlas of Chronic Kidney Disease and End-Stage Renal Disease in the United States. 2007, National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases. : Bethesda. p. 239-54.
8. R Atkins, P Zimmet. Diabetic kidney disease: Act now or pay later. *The Internet Journal of Nephrology*. 2009 ; 6(1):1-3.
9. Molitch ME, DeFronzo, Franz MJ, Keane WF, Mogensen CE, Parving HH, Steffes MW, American Diabetes Association: Nephropathy in diabetes. *Diabetes Care* 2004; 27(supp 1): S79-S83.
10. Khosla N, Sarafidis PA, Bakris GL: Microalbuminuria. *Clin Lab Med* 2006; 26; 635-653.

11. Levey AS, Eckardt KU, Tsukamoto Y, Levin A, Coresh J, et al. Definition and classification of chronic kidney disease: A position statement from Kidney Disease: Improving Global Outcomes (KIDGO). *Kidney Int* 2005; 67(6); 2089-2100.
12. Lembers Heerspink HJ, Brantsma AH, de Zeeuw D, Bakker SJL, de Jong PE, Gansevoort RT; for the PREVEND study Group. Albuminuria assessed from first-morning-void urine samples versus 24-hour urine collections as a predictor of cardiovascular morbidity and mortality. *Am J Epidemiol* 2008;168(8):897-905.
13. Levey AS, Coresh J, Balk E, Kausz AT, Levin A, Steffes MW, et al; National Kidney Foundation. National Kidney Foundation practice guidelines for chronic kidney disease: Evaluation, and stratification. *Ann Intern Med*. 2003;139(2):137-147.
14. Keane WF, Eknoyan G, Proteinuria, albuminuria, risk, assessment, detection, elimination (PARADE): a position paper of the National Kidney Foundation. *Am J Kidney Dis*. 1999;33:1004-10. [PMID: 10213663]
15. Raabo E, Terkildsen TC. On the enzymatic determination of blood glucose. *Scand J Clin Lab Invest* 1960; 12: 402-407 [PMID: 13738785 DOI: 10.3109/00365516009065404].
16. Rifai N, Bachorik PS, Albers JJ. Lipids, lipoproteins and apolipoproteins. In: Burtis CA, Ashwood ER, editors. *Tietz fundamentals of clinical chemistry*, 5th edn, Philadelphia: Saunders (an imprint of Elsevier); 2001, p. 462-493.
17. Newman DJ, Price CP. Nonprotein nitrogen metabolites. In: Burtis CA, Ashwood ER, editors. *Tietz fundamentals of clinical chemistry*, 5th edn, Philadelphia: Saunders (an imprint of Elsevier); 2001, p. 414-426.
18. Fossati P, Prencipe L, Berti G. Use of 3,5-dichloro-2-hydroxybenzenesulfonic acid/4-aminophenazone chromogenic system in direct enzymic assay of uric acid in serum and urine. *Clin Chem* 1980; 26(2): 227-31.
19. Bowers LD, Wong ET. Kinetic serum creatinine assays. II. A critical evaluation and review. *Clin Chem* 1980; 26(5): 555-61.
20. Scott MG, Heusel JW, Ligrys VA, Siggaard-Anderson O. Electrolytes and blood gases. In: Burtis CA, Ashwood ER, editors. *Tietz fundamentals of clinical chemistry*, 5th edn, Philadelphia: Saunders (an imprint of Elsevier); 2001, p. 495-517.
21. Gerbaut L. Immunoturbidimetry of albumin in serum, cerebrospinal fluid, and urine with a unique calibration curve. *Clin Chem*. 1987;33(7):1260-1. [PMID: 3594872].
22. National Kidney Foundation. Kidney Disease Outcomes Quality Improvement (K/DOQI™) clinical practice guidelines and clinical practice recommendations for diabetes and chronic kidney disease. *Am J Kidney Dis* 2007;49(Suppl 2):S1-S180.
23. U.S. Renal Data System, USRDS 2009 Annual Data Report: Atlas of Chronic Kidney Disease and End-Stage Renal Disease in the United States, National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases, Bethesda, MD, 2009. <http://www.usrds.org/atlas09.aspx>
24. Plantinga LC, Crews DC, Coresh J, Miller ER, Saran R, Yee J et al. for CDC CKD. Surveillance Team. Prevalence of chronic kidney disease in US adults with undiagnosed diabetes or prediabetes. *Clin J Am Soc Nephrol*. Apr 2010; 5(4): 673-682.
25. Gross JL, De-Azevedo MJ, Silveiro SP, Canani LH, Caramori M, Zelmanovitz T. Diabetic Nephropathy: Diagnosis, Prevention, and Treatment. *Diabetes Care* 2005; 28:176-188.
26. Adler A, et al., Development and progression of nephropathy in type 2 diabetes: the United Kingdom Prospective Diabetes Study (UKPDS 64). *Kidney Int* 2003. 63: p. 225-32.
27. Young BA, Maynard C, Boyko EJ. Racial differences in diabetic nephropathy, cardiovascular disease, and mortality in a national population of veterans. *Diabetes Care* 26:2392-2399, 2003.
28. Kedam DP, Rajaseker P. Study of microalbuminuria as a cardiovascular risk factor in type 2 diabetes mellitus. *Asian J Pharm Clin Res* 2012; 5(2): 42-43.
29. Thakkar B, Arora K, Vekariya R, Lulania M, Agnihotri AS. Prevalence of microalbuminuria in newly diagnosed type 2 diabetes mellitus. *NJIRM* 2011; 2(4) : 22-25.
30. Lu B, Wen J, Song XY, Dong XH, Yang YH, Zhang ZY, et al. High prevalence of albuminuria in population-based patients diagnosed with type 2 diabetes in the Shanghai downtown. *Diabetes Res Clin Pract*. 2007 Feb;75(2):184-92. Epub 2006 Aug 8.
31. Al-Maskari F, El-Sadig M, Obineche E. Prevalence and determinants of microalbuminuria among diabetic patients in the United Arab Emirates. *BMC Nephrology* 2008; 9:1. doi:10.1186/1471-2369-9-1
32. Al-Shaikh A. Prevalence of microalbuminuria in type 2 diabetes mellitus at a diabetic clinic in king abdulaziz university hospital. *Pak J Med Sci* 2007; 23(2): 223-226.
33. Kanakamani J, Ammini AC, Gupta N, Dwivedi SN. Prevalence of microalbuminuria among patients with type 2 diabetes mellitus--a hospital-based study from north India. *Diabetes Technol Ther*. 2010 Feb;12(2):161-6. doi: 10.1089/dia.2009.0133.
34. Chowta NK, Pant P, Chowta MN. Microalbuminuria in diabetes mellitus: Association with age, sex, weight, and creatinine clearance. *Ind J Nephrology* 2009;19(2):53-57. DOI: 10.4103/0971-4065.53322
35. Varghese A, Deepa R, Rema M, Mohan V. Prevalence of microalbuminuria in type 2 diabetes mellitus at a diabetes centre in southern India. *Postgrad Med J* 2001;77:399-402
36. Gupta DK, Verma LK, Khosla PK, Dash SC. The prevalence of microalbuminuria in diabetes: a study from north India. *Diabetes Res Clin Pract*. 1991 May;12(2):125-8.
37. Parving H, et al., Prevalence and risk factors for microalbuminuria in a referred cohort of type II diabetic patients: a global perspective. *Kidney Int*, 2006. 69(11): p. 2057-63.
38. Nakhjavani M, Esteghamati AR, Esfahanian F, Heshmat AR. Dyslipidemia in type 2 diabetes mellitus: more atherogenic lipid profile in women. *Acta Medica Iranica* 2006; 44(2): 111-118.