

## Research Article

# Correlation of glycosylated hemoglobin with microalbuminuria to predict renal damage in diabetic patients

Dipsha Kriplani<sup>1\*</sup>, Akshay Kriplani<sup>3</sup>, Vivek Gupta<sup>1</sup>, N. Samal<sup>1</sup>, Arvind Bhake<sup>1</sup>, Satish Mahajan<sup>2</sup>

<sup>1</sup>Department of Pathology, <sup>2</sup>Department of Medicine, Jawaharlal Nehru Medical College, Datta Meghe Institute of Medical Science, Sawangi (M), Wardha, Maharashtra, India

<sup>3</sup>Department of Surgery, MRM College, Gulbarga, Karnataka, India

**Received:** 10 June 2015

**Revised:** 16 June 2015

**Accepted:** 09 July 2015

### \*Correspondence:

Dr. Dipsha Kriplani,  
E-mail: [dipkrip@gmail.com](mailto:dipkrip@gmail.com)

**Copyright:** © the author(s), publisher and licensee Medip Academy. This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

## ABSTRACT

**Background:** Regular screening of levels of glycosylated hemoglobin and microalbuminuria, diabetic nephropathy can be prevented. The aim was to assess and compare the levels of glycosylated hemoglobin, microalbuminuria and serum creatinine in type 2 diabetic patients divided in groups of those on default antidiabetic treatment compared with those on regular antidiabetic treatment and to assess its correlation in type 2 of diabetic nephropathy.

**Methods:** Two hundred diabetic patients above 40 years of age and 200 age matched control subjects with levels of glycosylated hemoglobin < 6.5% and on regular antiglycemic therapy were selected. Fasting plasma sugar was estimated by the glucose oxidase (GOD) - glucose peroxidase (POD). Glycosylated hemoglobin and microalbuminuria level was measured by the immunoturbidimetric method and serum creatinine estimation was done by the Jaffe's kinetic method. *p* value was drawn using the student's paired t-test.

**Results:** There is a strong correlation between the increase in the levels of glycosylated hemoglobin with the corresponding rise in the levels of microalbuminuria and serum creatinine.

**Conclusion:** Periodic surveillance of the levels of microalbuminuria should be carried out in the type 2 diabetic patients to prevent further damage by early detection of diabetic nephropathy.

**Keywords:** Glycosylated hemoglobin, Microalbuminuria, Diabetic nephropathy, Glycemic control

## INTRODUCTION

Diabetes mellitus is a metabolic disorder of multiple etiology characterized by chronic hyperglycaemia with disturbance in carbohydrate, fat and protein metabolism resulting from defects in insulin secretion, insulin action or both.<sup>1</sup> Long term effects of diabetes are responsible for the macro and microvascular complications.<sup>2</sup>

Diabetic Nephropathy is a major microvascular complications of diabetes.<sup>3</sup> Its earliest manifestation is the appearance of low amounts of albumin in the urine of patients (> 30 mg/day but < 300 mg/day) referred to as microalbuminuria.<sup>4</sup> It is easily measured but frequently

overlooked as a tool for assessment of impairment of kidney functions and is associated with progression of chronic kidney disease (CKD) to more advanced stages or to end-stage renal disease (ESRD).<sup>5</sup>

Along with microalbuminuria, serum creatinine concentration is also a widely accepted parameter to measure nephropathy. It is interpreted as a measure of the glomerular filtration rate (GFR) and is used as an index of renal function in clinical practice.<sup>6</sup>

The pathogenesis of diabetic nephropathy is mediated by persistent hyperglycemia.<sup>7</sup> The level of glycosylated hemoglobin is proportional to the average blood glucose

concentration over the previous four weeks to three months. This gives an assessment of the glycemic control of the patient.<sup>8</sup>

Thus, the effect of intensive glycemic control in the development of microvascular complications establishes the clinical utility of HbA1c as a tool to assess the risk of complications of diabetes.<sup>9</sup> With regular screening of levels of glycosylated hemoglobin and microalbuminuria, diabetic nephropathy can be prevented.

## METHOD

This is a cross sectional analytical study, carried out in the Department of Pathology, Jawaharlal Nehru Medical College, Datta Meghe Institute of Medical Sciences.

Two hundred diabetic patients above 40 years of age with a minimum duration of 5 years of diabetes from the time of diagnosis, admitted in the Department of /Medicine and Surgery were recruited in the study<sup>28</sup>.

### Exclusion criteria<sup>30</sup>

1. Diabetic patients with overt albuminuria i.e. urinary albumin excretion > 300 mg/24 hrs.
2. Diabetic patients with anemia were excluded as the level of glycosylated hemoglobin is dependent on the life span of RBCs.

Institutional Ethical Committee clearance was taken and consent was obtained from the participant. A proforma regarding the demographic data, duration, details of treatment and complications of diabetes was recorded.

Two hundred control subjects were selected from an age matched population on the basis of the levels of glycosylated hemoglobin < 6.5% who were on regular antidiabetic therapy

As per American Diabetes Association, diabetes was defined by levels of fasting plasma glucose  $\geq$  126 mg/dl.<sup>31</sup>

Venous blood and the first morning urine samples were collected. Fasting and post meal plasma sugar estimation was done by the GOD - POD method using the Glucose kit (Mono reagent) by Asritha. Fasting plasma glucose levels between 70mg/dl to 100 mg/dl was considered normal, between 100mg/dl to 125 mg/dl was taken as prediabetes and  $\geq$  126 mg/dl was taken as a criterion for diagnosis of diabetes.<sup>31</sup>

Serum creatinine estimation was done by modified 2-point Jaffe's kinetic method using alkaline picrate. Normal serum creatinine for adults was taken as 0.5 – 1.0 mg/dl.<sup>32</sup>

Glycosylated hemoglobin level was measured by the immunoturbidimetric method in the Randox RX Daytona

clinical chemistry automatic analyser. Level of Glycosylated hemoglobin from 5.7% to 6.4% was considered as prediabetes and levels  $\geq$  6.5% as a diagnostic criterion for diabetes.<sup>31</sup>

Urine albumin was assessed by the reagent strip method using the reagent strips from Teco Diagnostics based on protein error- of- indicator principle and expressed in mg/dl. Dipsticks show reactivity only in the presence of macroalbuminuria. Thus, patients with positive dipstick test were excluded from the study. When dipstick showed no reaction, the sample was tested for micro- or normoalbuminuria.

Microalbuminuria was assessed by turbidimetric immunoassay based on the principle of agglutination reaction using the Turbilyte-MA kit by Tulip diagnostics and expressed in terms of mg/L.

The reference value of urinary albumin in normal population was taken to be <20mg/L. Microalbuminuria was considered when urine albumin excretion was 20-200 mg/L or 30-300 mg/24 hrs.<sup>6,30</sup>

The values of the serum creatinine, glycosylated hemoglobin and microalbuminuria of the patients were stratified into group A and B based on the fasting sugar levels of the patients. Group A comprised of patients with fasting blood sugar levels between 126-200mg/dl and Group B had patients whose fasting blood sugar levels were more than 200mg/dl.<sup>28</sup>

An attempt was made to correlate the variables selected for the study (HbA1c and microalbuminuria) with the extent of renal damage through the values of serum creatinine.

The statistical analysis for the value of significance (*p* value) was drawn using the student's paired t-test. The intervariable relationship was attempted for HbA1c and microalbuminuria.

## RESULTS

Two hundred known diabetic patients above 40 years of age were taken as cases. Two hundred age matched subjects with known diabetes on oral anti glycaemic drugs were selected as control subjects for comparison of the results with the study subjects in regard to nephropathy.

The mean fasting and post meal plasma sugar level across all the ages of the case subjects was  $176.69 \pm 58.28$ mg/dl and  $281.37 \pm 64.8$  mg/dl respectively. There was no homology of values for distribution across the age ranges for mean fasting and post meal plasma sugar levels.

The mean glycosylated hemoglobin of the case subjects having diabetes for 5-10 years was  $13.32 \pm 5.98\%$  and the corresponding mean value of microalbuminuria was  $127.14 \pm 52.92$  mg/L. This is highly statistically significant with the *p* value less than 0.0001, *df* =128.

**Table 1: Distribution of demographic characters of case subjects and control subjects.**

Age group (years)	Sex		Duration of diabetes (years)						Positive Family history of diabetes		Total no. of patient	
	Male	Female	5-10		>10		Study subjects	Control subjects	Study subjects	Control subjects	Study subjects	Control subjects
	Study subjects	Control subjects	Study subjects	Control subjects	Study subjects	Control subjects	Study subjects	Control subjects	Study subjects	Control subjects	Study subjects	Control subjects
31-40	4	3	1	2	5	-	-	5	5	5	5	5
41-50	34	30	13	17	47	-	-	47	17	16	47	47
51-60	31	26	20	25	7	2	44	49	41	37	51	51
61-70	50	38	25	37	5	7	70	68	68	61	75	75
71-80	14	10	4	8	1	8	17	10	15	14	18	18
>80	1	2	3	2	-	1	4	3	4	4	4	4
Total no. of patient	134 (67%)	109 (54.5%)	66 (33%)	91 (45.5%)	65 (32.5%)	18 (9%)	135 (67.5%)	182 (91%)	150 (75%)	137 (68.5%)	400	

**Table 2: Distribution of the case subjects and control subjects according to the plasma sugar levels over the age ranges.**

Age range (years)	Plasma sugar levels (mg/dl)				Total number of patients	
	Fasting Plasma Sugar Levels		Post meal Plasma Sugar Levels		Case subjects	Control subjects
	Case subjects	Control subjects	Case subjects	Control subjects	Case subjects	Control subjects
31-40	191.6 ± 117.4	119.4± 7.79	329.6 ± 93.52	148.8± 14.97	5	5
41-50	173.68± 66.99	120± 6.86	281.8± 66.17	152.55± 8.9	47	47
51-60	179.58± 57.78	118.31± 6.7	293.92± 63.82	150.23±10.65	51	51
61-70	173.24± 48.61	118.82± 6.65	269.34± 61.59	152.34± 9.58	75	75
71-80	188.27± 55.29	119.5± 7.7	279.22± 61.05	151.72±9.77	18	18
>80	169.25± 71.31	118± 6.05	291 ± 82.45	145.25± 6.99	4	4
Mean	176.69± 58.28	119.03± 6.77	281.37 ± 64.8	151.57±9.82	400	

**Table 3: Comparison of glycosylated hemoglobin with microalbuminuria according to the duration of diabetes in study subjects and control subjects.**

Duration of diabetes (years)	HbA1c (%)		Microalbuminuria (mg/L)		Total		P value
	Case subjects	Control subjects	Case subjects	Control subjects	Case subjects	Control subjects	
5 – 10	13.32± 5.98	5.79± 0.32	127.14± 52.92	17.94±2.31	65	18	< 0.0001
>10	15.10 ± 4.34	5.91± 0.25	153.34 ± 32.90	19.18±2.74	135	182	< 0.0001

**Table 4: The comparison of the levels of microalbuminuria versus the variables of fasting plasma sugar, glycosylated hemoglobin and serum creatinine in Group A and Group B of the study subjects.**

Micro Albuminuria (mg/L)	Group A			Group B			Total 200
	Fasting Plasma Sugar* (mg/dl)	Glycosylated Hemoglobin** (%)	Serum Creatinine*** (mg/dl)	Fasting Plasma Sugar* (mg/dl)	Glycosylated Hemoglobin** (%)	Serum Creatinine*** (mg/dl)	
21-50 (38.67±6.4)	130.61± 4.23	7.28± 0.33	2.46 ±1.27	-	-	-	
Total	14			-			13
51-80 (63.85± 11.33)	130.75±4.86	9.47 ± 3.62	3.36±0.94	-	-	-	
Total	8			-			8
81-110 (98.41±10.29)	134.11±15.80	10.67± 3.37	4.84± 1.00	-	-	-	
Total	17			-			17

111-140 (128.15± 7.32)	141.42± 20.06	12.02± 2.61	6.26± 0.33	238.66±41.99	15.33±1.53	6.53±0.45
Total	21			6		27
141-170 (157.82±7.94)	151.09± 18.84	14.37± 3.23	7.40± 0.85	240.28± 32.84	15.63± 3.98	7.37±1.14
Total	52			21		73
171-200 (182.21± 7.38)	159.17±17.73	16.02± 4.02	8.23±1.5	275.51±57.71	21.2± 4.61	8.92±0.60
Total	35			27		62
Mean Microalbuminuria	136.85 ± 45.48			166.37 ± 19.47		

\*P value < 0.0001; \*\*P value < 0.0001; \*\*\*P value < 0.0001

In the 135 patients having diabetes for over 10 years, the mean glycosylated hemoglobin was 15.10 ± 4.34% and the corresponding mean value of microalbuminuria was 153.34 ± 32.90 mg/L. This is statistically significant with the p value less than 0.0001, df= 268.

The comparisons through the graded quantification of microalbuminuria, shows a linear correlation between the fasting plasma sugar and glycosylated hemoglobin in Group A as well as in Group B patients of the study population. The value of significance is high for these variables when compared with the increasing grade of microalbuminuria and also intervariable comparisons of fasting sugar, glycosylated hemoglobin and kidney function indicator of serum creatinine. Group B patients who had the fasting plasma glucose value of 238.66±41.99 mg/dl with further change in its value correlated with glycosylated hemoglobin to possess the similar ascending value relationship. However, there were overlaps for the values of mean fasting plasma sugar, glycosylated hemoglobin and serum creatinine of Group A subjects with that of Group B subjects for the same variables when microalbuminuria was over 111mg/L with mean of 128.15± 7.32mg/L.

**Table 5: Comparison of study population versus age matched control subjects.**

		Case subjects	Control subjects	P value
Duration of diabetes	5- 10 years	65 (32.5%)	18 (9%)	
	>10 years	135(67.5%)	182(91%)	
Positive Family history		150 (75%)	137 (68.5%)	
Fasting Plasma sugar level		176.69± 58.28	119.03± 6.77	< 0.0001
Post meal plasma sugar level		281.37 ± 64.8	151.57±9.82	< 0.0001
Glycosylated hemoglobin		14.54 ± 4.99	5.94±0.26%	< 0.0001
Microalbuminuria		144.82±42.20	19.07± 2.6	< 0.0001
Serum creatinine		6.09 ± 2.03	1.47±0.14 mg/dl.	< 0.0001

It is observed that the mean fasting plasma glucose value appeared well controlled in the control subjects on

regular oral antiglycemic drugs with the mean values of 119.03± 6.77mg/dl. All the other variables indicating diabetes control and renal damage were on the lower side. The p values suggested that all these values in the study population are significantly raised suggesting high grade nephropathy, decreased renal function and damage.

## DISCUSSION

The studies reviewed for the present work have shown that the complications of diabetic nephropathy begin to set in if the patient has had diabetes for over 5 years as quoted by Neil et al.,<sup>12</sup> Forsblom et al.,<sup>15</sup> Stratton et al.,<sup>16</sup> Tabaei et al.,<sup>17</sup> Bruno et al.,<sup>18</sup> Verhave et al.,<sup>20</sup> Wu et al.,<sup>22</sup> Retnakaran et al.,<sup>23</sup> Parvanova et al.,<sup>24</sup> Parving et al.,<sup>25</sup> Naveen et al.<sup>28</sup> and that the duration of diabetes reflects on diabetic nephropathy and its severity.<sup>12, 15, 16, 17, 18, 20, 22, 23, 24, 25, 28</sup> The observations of the present study are in concordance with these studies. However, the studies of Mattock et al.<sup>11</sup> and Rossing et al.<sup>21</sup> stated that the presence of microalbuminuria is not associated with the known duration of diabetes.

The study observed that the levels of glycosylated hemoglobin and microalbuminuria were found to increase with the increase in the duration of diabetes. This correlation was highly significant and supported by the studies of Mattock et al.,<sup>11</sup> Neil et al.,<sup>12</sup> Forsblom et al.,<sup>15</sup> Tabei et al.,<sup>17</sup> Bruno et al.,<sup>18</sup> Wu et al.,<sup>22</sup> Parvanova et al.,<sup>24</sup> Naveen et al.<sup>28</sup> In the studies conducted by Tahara et al.,<sup>13</sup> a rise in the levels of glycosylated hemoglobin was seen along with the increase in the duration of diabetes. Adler et al.<sup>19</sup> reported a rise in the levels of microalbuminuria along with the duration of diabetes at a rate of 2.0% per year. The increase in these values is explained by the pathogenesis of formation of glycated hemoglobin. Longer duration of uncontrolled diabetes pathogenetically correlates with the higher values of glycated hemoglobin.

This study shows that the level of glycosylated hemoglobin also increases with levels of fasting plasma sugar levels. This was supported in the studies of Aleyassine et al.<sup>10</sup> and Tahara et al.<sup>13</sup> This was also associated with increasing levels of serum creatinine which was highly significant, thus indicating renal damage. This was similar to the findings of the studies of Forsblom et al.,<sup>15</sup> Bruno et al.,<sup>18</sup> Adler et al.,<sup>19</sup> Rossing et

al.,<sup>21</sup> Retnakaran et al.,<sup>23</sup> Parvanova et al.,<sup>24</sup> Parving et al.<sup>25</sup> and Makita et al.<sup>29</sup> A highly significant correlation was also seen with the rise in the levels of microalbuminuria along with the increase in fasting plasma sugar. This indicates increased renal damage with increase in fasting plasma sugar levels. This was supported by the studies of Adler et al.,<sup>19</sup> Rossing et al.,<sup>21</sup> Parvanova et al.,<sup>24</sup> Retnakaran et al.,<sup>23</sup> Parving et al.,<sup>25</sup> Forsblom et al.,<sup>15</sup> Makita et al.<sup>29</sup> and Bruno et al.<sup>18</sup>

Microalbuminuria as an indicator of nephropathy and renal damage has been concomitantly correlated with fasting plasma sugar, glycosylated hemoglobin and occasionally with serum creatinine by many workers. The study of Retnakaran et al.<sup>23</sup> stated that these variables rise together if not intervened by antiglycemic drugs. However, multivariate model of the same study has found that the rise in glycosylated hemoglobin was an independent predictor of microalbuminuria. Parving et al.<sup>25</sup> in his study suggested that at the mean level of HbA1c of 7.5%, microalbuminuria could be demonstrated in 39% of the patients and overt albuminuria in 9.8% of the patients while other remained normoalbuminuric. In the main study done by Parvanova et al.,<sup>24</sup> glycosylated hemoglobin and serum creatinine were significantly and independently associated with microalbuminuria. The study done by Bruno et al.<sup>18</sup> used a multivariate conditional logistic regression analyses to calculate the effect of the mean value of glycosylated hemoglobin in the progression of normoalbuminuria to microalbuminuria. It was found that the relative risk of progression to overt nephropathy was twofold higher in micro- than in normoalbuminuric patients. Rossing et al.<sup>21</sup> also used a multivariate analysis which revealed that higher values of albuminuria (581 mg/24hrs) and glycosylated hemoglobin (8.9%) along with baseline Glomerular Filtration Rate were significantly associated with an increased rate of decline in kidney function. It was noted in the follow up of this study that the baseline serum creatinine values doubled in 28% of these and they developed End Stage Renal Disease. Wu et al.<sup>22</sup> in his study had noted that glycemic control prevents development of nephropathy. Forsblom et al.<sup>15</sup> in his study showed that long term glycemic control was an independent risk factor for the progression to microalbuminuria. Naveen et al.<sup>28</sup> in their observed that the levels of microalbuminuria increased significantly with poor glycemic control and correlated with elevated serum creatinine levels indicating renal damage.

The observed differences in the levels of plasma sugar, glycosylated hemoglobin, microalbuminuria and serum creatinine between study subjects and control subjects is attributable to the good glycemic control which prevents the rise in the levels of microalbuminuria and serum creatinine and subsequently prevents the development of diabetic nephropathy as one of the complication of diabetes mellitus. This was supported by the study of Naveen et al.<sup>28</sup> which was carried out using the similar methods as the present study.

*Funding: No funding sources*

*Conflict of interest: None declared*

*Ethical approval: The study was approved by the Institutional Ethics Committee*

## CONCLUSION

- The study concluded that with increase in the duration of diabetes and fasting plasma sugar levels, the levels of glycosylated hemoglobin increase correspondingly.
- Increase in the levels of glycosylated hemoglobin is associated with a corresponding rise in the levels of microalbuminuria and serum creatinine is seen which predicts the progression of renal damage.
- Microalbuminuria as an indicator of nephropathy and renal damage has been linearly correlated with elevated fasting plasma sugar, glycosylated hemoglobin and serum creatinine.
- Better glycemic control is seen in patients on regular antidiabetic treatment than defaulters, thus preventing the rise in the levels of microalbuminuria and serum creatinine and subsequent development of diabetic nephropathy as a complication of diabetes mellitus.
- It is therefore suggested that surveillance of the levels of microalbuminuria should be carried out periodically in the type 2 diabetic patients to prevent further damage by early detection of diabetic nephropathy.

## REFERENCES

1. Maitra A, Abbas A. The Endocrine System. Kumar V, Abbas A, Fausto N. Robins and Cotran Pathologic Basis of Disease. 8<sup>th</sup> edition. Elsevier. India.
2. Report of a WHO Consultation, Part 1: Diagnosis and Classification of Diabetes Mellitus. World Health Organization, Department of Noncommunicable Disease Surveillance, Geneva.
3. Mogensen CE, Christensen CK, Vittinghus E. The stages in diabetic renal disease: with emphasis on the stage of incipient diabetic nephropathy. *Diabetes*. 1983;32(2):64-78.
4. Moser M, Sowers J, Black H. Microalbuminuria, Chronic Renal disease and the effects of metabolic syndrome on cardiovascular events. *The Journal of Clinical Hypertension*. 2007;9(7):551-6.
5. James MT, Hemmelgran BR, Tonelli M. Early recognition and prevention of chronic kidney disease. *Lancet*. 2010;375:1296-309.
6. Taylor, E. Howard. Clinical Chemistry. New York: John Wiley and Sons. 1989;4:58-62.
7. Rossini AA. Special speculations on etiology of Diabetes Mellitus. *Diabetes*. 1998;37:257.
8. Saudek C, Kalyani R, Derr R. Assessment of Glycemia in Diabetes Mellitus. *JAPI*. 2005;53:299-305.
9. UK Prospective Diabetes Study (UKPDS) Group. Intensive blood glucose control with sulphonylureas

- or insulin compared with conventional treatment and risk of complications in in patients with type 2 diabetes (UKPDS 33). *Lancet.* 1998;352:837-53.
10. Aleyassine H, Gardiner R, Tonks D, Koch P. Glycosylated hemoglobin in diabetes: Correlation with fasting plasma glucose, serum lipids and glycosuria. *Diabetes Care.* 1980;3(4):508-14.
  11. Mattock M, Morrish N, Viberti G, Keen H, Fitzgerald A and Jackson G. Prospective Study of Microalbuminuria as Predictor of Mortality in NIDDM. *Diabetes.* 1992;41:736-41.
  12. Neil A, Hawkins M, Potok M, Thorogood M, Cohen D, Mann J. A Prospective Population- Based Study of Microalbuminuria as a Predictor of Mortality in NIDDM. *Diabetes Care.* 1993;16(7):996-1003.
  13. Tahara Y, Shima K. Kinetics of HbA1c, Glycated Albumin, and Fructosamine and Analysis of Their Weight Functions against Preceding Plasma Glucose Level. *Diabetes Care.* 1995;18(4):440-7.
  14. Beisswenger P, Makita Z, Curphey T, Moore L, Jean S, Johnsen T, et al. Formation of Immunochemical Advanced Glycosylation End Products Precedes and Correlates with Early Manifestations of Renal and Retinal Disease in Diabetes. *Diabetes.* 1995;44:824-9.
  15. Forsblom C, Groop P, Ekstrand A, Tötterman K, Sane T, Saloranta C, et al. Predictors of Progression from Normoalbuminuria to Microalbuminuria in NIDDM. *Diabetes Care.* 1998;21(11):1932-38.
  16. Stratton I, Adler A, Neil A, Matthews D, Manley S, Cull C, et al. On behalf of the UK Prospective Diabetes Study Group. Association of glycaemia with macrovascular and microvascular complications of type 2 diabetes (UKPDS 35): prospective observational study. *BMJ.* 2000;321:405-12.
  17. Tabaei B, Al-Kassab A, Ilag L, Zawacki C, Herman W. Does Microalbuminuria Predict Diabetic Nephropathy? *Diabetes Care.* 2001;24(9):1560-6.
  18. Bruno G, Merletti F, Biggeri A, Barger G, Ferrero S, Pagano G, et al. Progression to Overt Nephropathy In Type 2 Diabetes. *Diabetes Care.* 2003;26(7):2150-5.
  19. Adler A, Stevens R, Manley S, Bilous R, Cull C, Holman R. On behalf of UKPDS group. Development and progression of nephropathy in type 2 diabetes: The United Kingdom Prospective Diabetes Study (UKPDS 64). *Kidney International.* 2003;63:225-32.
  20. Verhave J, Gansevoort R, Hillege H, Bakker S, Zeeuw D, De Jong P. The Prevend Study Group. An elevated urinary albumin excretion predicts de novo development of renal function impairment in the general population. *Kidney International.* 2004;66(92):S18–S21.
  21. Rossing K, Christensen P, Hovind P, Tarnow L, Rossing P, Parving H. Progression of nephropathy in type 2 diabetic patients, *Kidney International,* 2004;66:1596–605.
  22. Wu A, Kong N, de Leon F, Pan C, Tai T, Yeung V, et al. The MAPS Investigators. An alarmingly high prevalence of diabetic nephropathy in Asian type 2 diabetic patients: the MicroAlbuminuria Prevalence (MAP) Study. *Diabetologia.* 2005;48:17–26.
  23. Retnakaran R, Cull C, Thorne K, Adler A, Holman R. The UKPDS Study Group. Risk Factors for Renal Dysfunction in Type 2 Diabetes U.K. Prospective Diabetes Study 74. *Diabetes.* 2006;55:1832-9.
  24. Parvanova A, Trevisan R, Iliev I, Dimitrov B, Vedovato M, Tiengo A, et al. Insulin Resistance and Microalbuminuria. A Cross-Sectional, Case-Control Study of 158 Patients With Type 2 Diabetes and Different Degrees of Urinary Albumin Excretion. *Diabetes.* 2006;22:1456-62.
  25. Parving H, Lewis J, Ravid M, Remuzzi G, Hunsicker L. The DEMAND investigators. Prevalence and risk factors for microalbuminuria in a referred cohort of type II diabetic patients: A global perspective, *Kidney International.* 2006;69:2057–63.
  26. Nathan D, Turgeon Hand Regan S. Relationship between glycated haemoglobin levels and mean glucose levels over time. *Diabetologia.* 2007;50:2239–44.
  27. Kaur S, Mahajan M. Declining Age of Onset of Type 2 Diabetes Mellitus in the North-West Punjabi Population. *Journal of Clinical and Diagnostic Research.* 2011;5(3):425-9.
  28. Naveen P, Kannan N, Vamseedhar A, Bhanu PG and Aravind K. Evaluation of Glycated hemoglobin and Microalbuminuria as early risk markers of Nephropathy in Type 2 Diabetes Mellitus. *International Journal of Biological & Medical Research.* 2012;3(2):1724-6.
  29. Makita Z, Radoff S, Rayfield E, Yang Z, Skolnik E, Delaney V, et al. Advanced glycosylation end products in patients with Diabetic Nephropathy. *The New England Journal of Medicine.* 1991;836-42.
  30. Sacks D, Arnold M, Bakris G, Bruns D, Horvath A, Kirkman M, et al. Guidelines and Recommendations for Laboratory Analysis in the Diagnosis and Management of Diabetes Mellitus. *Diabetes Care.* 2011;34:61-99.
  31. American Diabetes Association. Diagnosis and Classification of Diabetes Mellitus. *Diabetes Care.* 2013;36(1):S67-S74.
  32. Oh M. Evaluation of renal function, water, electrolytes and acid-base balance. McPherson R. and Pincus M. *Henry’s Clinical Diagnosis and Management by Laboratory Methods,* 22<sup>nd</sup> edition. India: Elsevier; 2012.

**Cite this article as:** Kriplani D, Kriplani A, Gupta V, Samal N, Bhake A, Mahajan S. Correlation of glycosylated hemoglobin with microalbuminuria to predict renal damage in diabetic patients. *Int J Res Med Sci* 2015;3(8):2014-9.