ORIGINAL ARTICLE ASIAN JOURNAL OF MEDICAL SCIENCES

# A Study on Urinary Glycated Albumin to urinary albumin excretion in gestational diabetes mellitus

**Chandramallika Paul[1], Santanu Banerjee[2], Satinath Mukhopadhyay[3], Kalyan Goswami[4]**

*[1]Senior Resident, [4]Professor and Head, Department of Biochemistry, All India Institute of Medical Sciences (AIIMS)-Kalyani, India, [2]Professor and Head, Department of Biochemistry, Midnapore Medical College and Hospital, WB, India, [3]Professor, Department of Endocrinology and Metabolism, IPGMER & SSKM Hospital, Kolkata, India*

Submission: 26-10-2020 Revision: 15-12-2020 Publication: 01-02-2021

## ABSTRACT

**Background:** Gestational Diabetes Mellitus (GDM) is a public health problem in India with implications well pronounced in pregnancy and beyond. Biomarkers like Glycated Albumin (G.A.) can well monitor the glycaemic status and evaluate the transient hyperglycaemic spikes, which account for the diabetic complications. **Aims and Objectives:** In this study, we intend to study urinary G.A. excretion with respect to urinary albumin excretion expressed as UGA% in gestational diabetes mellitus. **Materials and Methods:** A prospective observational study was conducted for a period of 16 months on 177 pregnant women who attended antenatal clinics for the first time at a single centre.Among the surveyed population, 26 pregnant women subsequently developed GDM, and 31 healthy pregnant women who did not develop GDM were included in the study. **Results:** The UGA% between GDM and healthy mothers showed an increase in GDM with a p value $<0.05$ during the first and second trimesters. Pearson's correlation coefficient at 5% interval showed moderate to strong correlation for fasting plasma glucose (FBS) vs UGA% in 1st trimester($r = 0.61$) and 3rd trimester($r = 0.54$).**Conclusion:** The higher UGA% in GDM mothers in the early trimester may help monitor glycaemic status efficiently and timely. Long term follows up would be worthwhile to predict future progression to nephropathy, retinopathy, and neuropathy. Henceforth, UGA being a non-invasive marker may emerge as a more patient-friendly marker reducing the hassles of innumerable invasive tests to monitor the well-being of a mother as well as a foetus during pregnancy.

**Key words:** Gestational diabetes mellitus; Glycated albumin; Advanced glycation end products

**Access this article online**

**Website:** http://nepjol.info/index.php/AJMS

**DOI:** 10.3126/ajms.v12i2.32471

**E-ISSN:** 2091-0576

**P-ISSN:** 2467-9100



## INTRODUCTION

Gestational Diabetes Mellitus (GDM), is defined as carbohydrate intolerance of variable severity with onset or first recognition during pregnancy.[1] The number of patients with gestational diabetes mellitus is rapidly increasing worldwide in recent times. Prevalence of gestational diabetes mellitus (GDM) is known to vary widely depending on the region of the country, dietary habits, and socioeconomic status. In the Indian population, GDM has a prevalence rate of 9-18%.[2,3] The majority of the women with gestational diabetes ultimately develop overt type 2 diabetes (T2 DM) and there is evidence for long-term complications that include obesity and diabetes in their offspring.[1] The odds of developing subsequent T2DM for women with GDM is approximately 5 times higher than that for women with normoglycemic pregnancies in the first 5 years and the chances increase further with time.[4] So monitoring these patients till delivery and postpartum (6-12 weeks) can help detect risk for long-term T2DM related complications like retinopathy, neuropathy, nephropathy, and cardiovascular complications.

The objectives of managing diabetic pregnancies are as follows:

**Address for Correspondence:**
Dr. Chandramallika Paul, NH-34 Connector, Basantapur, Saguna, Kalyani, District Nadia, West Bengal 741245. **Mobile:** +91-8017010811
**E-mail:** chandramallikapaul@gmail.com

I. Prevention of perinatal complications in mothers and fetuses/infants
II. Prevention of the development of diabetes mellitus and metabolic syndrome of the mothers and the neonates in their future.[5,6]

Fasting hyperglycemia early in pregnancy likely represents overt diabetes. Selective screening for Gestational diabetes between 24 and 28 weeks in those women not known to have glucose intolerance earlier in pregnancy is recommended.[1]

Albuminuria in pregnancy can be physiological and pathological owing to the pre-eclamptic changes and diabetic complications in gestational diabetes. However, exclusion of the definite cause of albuminuria in pregnancy has yet not been possible and is the focus of constant research.

Microalbuminuria (urinary albumin excretion of 30 mg–300 mg/day) is the earliest marker of the renal involvement of diabetes. Interestingly, while microalbuminuria is more predictive of reaching cardiovascular endpoints than kidney endpoints, macroalbuminuria (total protein urinary excretion >500 mg/day) has been demonstrated to be more associated with reaching kidney end points.[6-8] Therefore, an early detection and reduction or prevention of protein or albumin urine excretion is highly desirable.

The period of pregnancy is usually limited to 280 days. So a precise marker for glycaemic control is urgently needed in early diagnosis and management of GDM patients to curb the risk of complications to both mother and fetus. Recent results[9-11] highlight the dramatic and long-lasting effects that transient hyperglycaemic spikes can have on vascular walls. Glycated albumin (G.A.) with a much more rapid turnover rate (half-life of 15-20 days) provides an index of rapid glycaemic control marker over a shorter period of time (2 weeks) within the limited gestational period itself.[12,13]

This prospective observational study was undertaken to assess the glycaemic status in gestational diabetic pregnancies in a timely manner before the setting in of diabetic complications by a non-invasive marker i.e, UGA%. The correlation of UGA% with the corresponding blood sugar levels in fasting and postprandial state was also assessed. The prevalence of albuminuria was assessed by measuring the spot urinary ACR levels.

## MATERIALS AND METHODS

A prospective study was conducted for pregnant women who attended antenatal service for the first time in their first trimester and subsequently being followed up till their third trimester at SSKM hospital, Kolkata, for a period of 16 months. Women with pre-existing diabetes, hypertension and renal or liver impairment were excluded from this study. All pregnant women were screened for GDM at 20 weeks of gestation.American Diabetes Association (ADA) criteria for 75 g 2h OGTT was used for diagnosing GDM.

The woman's age, body mass index(BMI), family history of diabetes, previous GDM, previous birth of an overweight baby of 4kg or more, previous stillbirth, unexplained perinatal loss, results of FBS and PPBS and evidence of gestational hypertension (B.P.> 140/90 mmHg or taking antihypertensive medication at the initial antenatal visit in the first trimester) were documented.

The calculation of UGA% took into account the measurement of urinary glycated albumin, UGA (sandwich ELISA based) expressed in percentage of total urinary micro-albumin (in semi-auto analyzer by turbidimetric immunoassay). The urine samples and blood samples were collected in the last week of respective trimesters. In the third trimester, samples were collected before the delivery of their babies.

The study was approved by the Institutional Ethics Committee.

## STATISTICAL METHODS

### Sample size

Being an observational study, we did not do any formal sample size calculation. On the basis of available time and logistics, the recruitment target was being kept at 30 GDM subjects and 30 healthy pregnant mothers who served as controls for comparison of urinary parameters.

All the maternal parameters taken into consideration were categorized into their respective trimesters. To examine whether all the data were normally distributed, Kolmogorov-Smirnoff goodness-of-fit test was applied. For comparative analysis of all the parameters between the two independent groups i.e., between the GDM group and Control group, student's unpaired t test was used. Differences were considered significant with P-value $< 0.05$. To determine the correlation between UGA% and blood glucose levels, Pearson's correlation coefficient $r$ was measured. *The* '$r$' values indicate that correlation is significant at the 5% level (i.e., likely to hold in the underlying population as well). The correlation was considered moderate to strong if $r$ lies between 0.5-1.0, good if $r$ lies between 0.3-0.5, poor if $r < 0.3$.

All analyses were conducted in Statistica version 6.

## RESULTS

In total, 177 pregnant women were included in the study who attended the antenatal clinic for the first time in their first trimester. 26 pregnant women subsequently developed GDM in their late 2nd trimester while the remaining 151 pregnant women remained healthy throughout their gestational period. 31 pregnant mothers were considered as a control to the GDM population from the remaining healthy population. The detailed results are summarized in Table 1.

Considering the age between the GDM group and control group, the GDM population had a mean age group of 27.85±3.684 years and control group had a mean age group of 25.03 ± 5.839, thus showing a significant difference with P value of 0.038.

**Table 1: Baseline profile of the study Subjects**

| Parameter/s | GDM- group (n = 26) | Control group (n = 31) | *p* value |
|---|---|---|---|
| Age (years) | | | |
| Range | 22.0–38.0 | 17.0–40.0 | 0.038 |
| Mean±SD | 27.85±3.684 | 25.03±5.839 | |
| BMI(kg/m$^2$) T1 | | | |
| Range | 16.27-34.3 | 14.58-28.6 | 0.163 |
| Mean±SD | 23.06±4.791 | 21.47±3.667 | |
| BMI (kg/m$^2$) T2 | | | |
| Range | 17.41-36.7 | 16.25-30.1 | 0.160 |
| Mean±SD | 24.84±5.036 | 23.19±3.716 | |
| BMI (kg/m$^2$) T3 | | | |
| Range | 18.16-38.7 | 17.50-32.9 | 0.204 |
| Mea±SD | 26.54±5.160 | 24.96±4.091 | |
| FBS T1(mg/dl) | | | |
| Range | 64-102 | 58-95 | < 0.0001 |
| Mean±SD | 81.23±12.685 | 76.16±10.761 | |
| FBS T2(mg/dl) | | | |
| Range | 58-109 | 54-96 | < 0.0001 |
| Mean±SD | 88.42±14.443 | 75.06±10.804 | |
| FBS T3(mg/dl) | | | |
| Range | 67-118 | 59-91 | < 0.0001 |
| Mean±SD | 94.19±14.445 | 76.03±8.408 | |
| PPBS T1(mg/dl) | | | |
| Range | 84-140 | 71-06 | < 0.0001 |
| Mean±SD | 103.65±21.973 | 98.68±14.575 | |
| PPBS T2(mg/dl) | | | |
| Range | 99-190 | 71-116 | < 0.0001 |
| Mean±SD | 115.42±23.202 | 98.9±14.588 | |
| PPBS T3(mg/dl) | | | |
| Range | 108-195 | 73121 | < 0.0001 |
| Mean±SD | 131.73±30.828 | 99.35±12.771 | |
| UGA% T1 | | | |
| Range | 0.03-0.39 | 0.01-0.1 | < 0.0001 |
| Mean±SD | 0.14±0.093 | 0.06±0.031 | |
| UGA% T2 | | | |
| Range | 0.04-0.49 | 0.03-0.8 | 0.040 |
| Mean±SD | 0.208±0.138 | 0.13± 0.141 | |
| UGA% T3 | | | |
| Range | 0.03-0.74 | 0.03-0.7 | 0.785 |
| Mean±SD | 0.28±0.194 | 0.29±0.204 | |
| ACR T1(mg of alb/g of cr) | | | |
| Range | 22.59-559 | 11.25-257.5 | 0.018 |
| Mean±SD | 140.71±133.55 | 75.64±60.66 | |
| ACR T2 (mg of alb/g of cr) | | | |
| Range | 25.890-459.76 | 12.45-379.4 | 0.050 |
| Mean±SD | 159.48±123.259 | 105.25±79.41 | |
| ACR T3 (mg of alb/g of cr) | | | |
| Range | 23.03-890 | 24.04-402.0 | 0.020 |
| Mean±SD | 264±235.25 | 151.50±107.81 | |

Abbreviations: S.D. (Standard deviation), BMI (Body mass index ),FBS(Fasting blood glucose) , PPBS(Post prandial blood glucose), ACR(Albumin-creatinine ratio) , UGA%(Urinary Glycated albumin % calculated as Glycated albumin in percent of total Urinary Albumin excretion)
*p* value in the last column is from Student's unpaired *t* test.
T1(1st trimester) ,T2(2nd trimester) , T3(3rd trimester)

The other parameters, including the BMI, blood sugar levels (both FBS and PPBS), UACR, UGA% were measured thrice from time to time in the three trimesters. A comparison of BMI between the two groups did not significantly differ with a $p > 0.05$.

The blood sugar levels (FBS and PPBS) between the two groups showed a significant difference in all three trimesters with a p-value <0.0001. ACR values between the two groups also showed a significant difference in the three trimesters. Figure 1 depicts the trimester wise mean values of ACR for control and GDM patients.

### Urinary glycated albumin assay

The concentrations of standards are expressed on the abscissa and the corresponding O.D. values on the ordinate. The linear regression equation of a standard curve is then calculated and O.D. value of the sample is substituted in the equation to calculate its concentration. A representative standard curve is shown in the following Figure 2.

However, the UGA% calculated showed a significant difference with a p-value <0.05 during the first two trimesters with a much more significant difference in the 1st trimester with a p-value <0.0001 while in the last trimester there was no difference (p-value of 0.785). Figure 3 depicts the trimester wise mean values of UGA % for control and GDM patients

Table 2 and 3 depicts the 'Pearson's correlation coefficient *r values* between blood glucose levels and UGA% in GDM population. In the control group, no correlation was observed.

**Table 2: Pearson's correlation coefficient values for Trimester wise FBS vs. UGA Percentage (%) Group 1: GDM [n=26]**

| | UGA_T1 | UGA_T2 | UGA_T3 |
|---|---|---|---|
| FBS_T1 | 0.61 | | |
| FBS_T2 | | 0.36 | |
| FBS_T3 | | | 0.54 |

*Each Cell denotes Pearson's correlation coefficient (*r* - Value) as shown in Table 2 and Table 3.
Correlation was considered moderate to strong if *r* lie between 0.5-1, good if *r* lies between 0.3-0.5, poor if *r* < 0.3.

**Table 3: Pearson's correlation coefficient values for Trimester wise PPBS vs. UGA Percentage (%) Group 1 GDM [n=26]**

| | UGA_T1 | UGA_T2 | UGA_T3 |
|---|---|---|---|
| PPBS _T1 | 0.53 | | |
| PPBS _T2 | | 0.33 | |
| PPBS _T3 | | | 0.55 |

## DISCUSSION

An increase in insulin resistance during pregnancy leading to Gestational Diabetes Mellitus is a common pregnancy-related complication prevalent nowadays. This is a transient hyperglycemic episode within the limited pregnancy span of 280 days but may have long-lasting effects if not controlled early. Glycation is the process of non-enzymatic Maillard reaction initially involving the attachment of

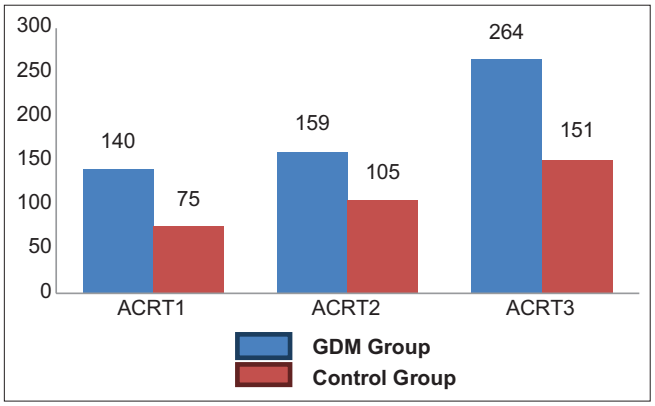


**Figure 1:** Trimester wise Mean Values of UACR for Control and GDM patients

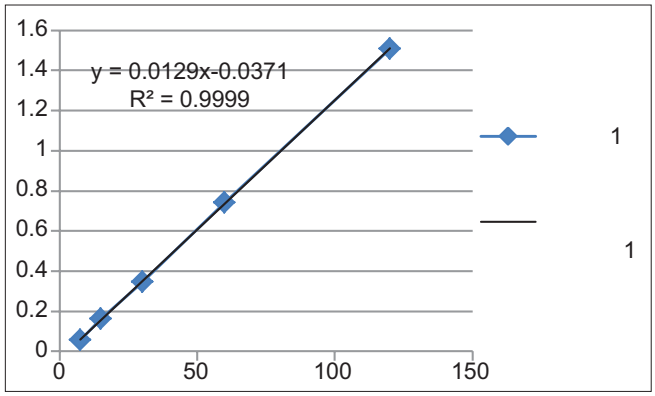


**Figure 2:** A representative standard curve showing G.A. Assay in Urine

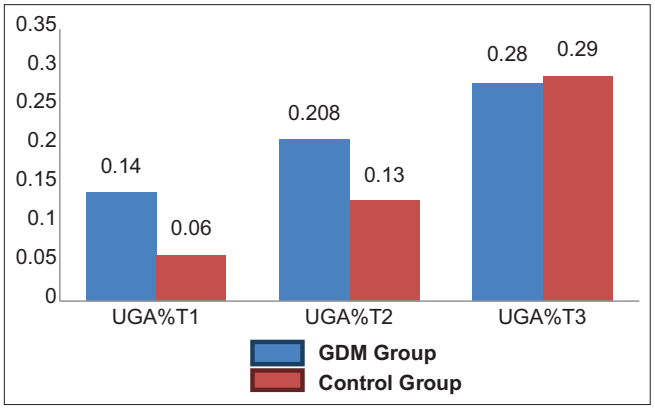


**Figure 3:** Trimester wise Mean Values of UGA % for Control and GDM patients

glucose and other carbohydrate compounds such as galactose and fructose with amino groups on protein and lipids.[9,14] Early glycation and oxidation produce Schiff bases and Amadori products. Further glycation and through a series of molecular rearrangement, irreversible products are known as Advanced Glycation End Products (AGEs) are formed [Figure. 4].

Human Serum Albumin (HSA), with a long half-life of 21 days and high concentration in plasma, is very sensitive to glycation. Approximately 4.5 times more Glycated albumin (G.A.) is produced as compared to other proteins like glycated hemoglobin (with Hb) when exposed to equal amounts of glucose.[15] The glycation of albumin induces several structural modifications, including an increase in the net negative electrical charge (pi 3.0 - 4.2) than the native albumin (pi 4.7 - 4.9).[10,16-18]

According to the standardized established concepts to date, Age and BMI can reasonably predict the likelihood of developing GDM.[19] GDM complicates about 3-5% of pregnancies. In the South Asian Population, the risk of developing GDM increases in women above the age group of 20 years. Generally, exhibiting higher BMI categories is at increased risk for GDM. In our study, considering the age between the GDM group and the control group, the GDM population shows a higher age group adhering to the findings of previous studies.However, GDM and healthy mothers exhibited comparable BMI without any significant difference between the two groups with $P > 0.05$. Although we have data on BMI for all the participants during gestation, we only have limited data on their pre-conceptional BMI and other anthropological parameters like neck circumferences, waist circumferences.

To measure the extent of albuminuria in our study population, the urinary albumin to creatinine ratio, ACR, was measured. The UACR showed a corresponding increment in all the 3 trimesters in both the study groups considered. In pregnancy, since the GFR increases, as the pregnancy progresses, the rate of urinary albumin excretion increases considerably with the advancing trimester.[20,21] The values of UACR are not very well defined in gestation and vary with gestation,[22] so a clear cut distinction about the rise in ACR cannot be made.

However, the extent of albuminuria was more in the GDM group as compared to the control group in all the trimesters.

To predict the prevalence of GDM and impending nephropathy in our study population, the percentage of urinary Glycated albumin in terms of the total urinary albumin, UGA%, was evaluated in all the three trimesters. There is a significant difference in UGA% in the 1st and 2nd trimesters, while in the last trimester, there was no difference. The increase in UGA% during early trimesters can be well explained and attributed to the renal handling of modified glycated albumin. Since GA acquires a higher negative charge (pi 3.0 - 4.2) than the native albumin (pi 4.7 - 4.9) G.A. is filtered to a lesser extent than the native albumin. But once filtered, glycated albumin is not reabsorbed in the proximal tubule.[10,16] In a very recent study[23] it was concluded that serum albumin being the most abundant plasma protein usually has a long half-life due to neonatal Fc receptor (FcRn) mediated transcytosis across the proximal tubule cells of the kidney. Structural modifications due to glycation result in decreased affinity to the FcRn receptor, and hence an increased proximal tubule mediated clearance of glycated albumins.

Thus for all practical purposes, the amount of glycated albumin appearing in the urine is primarily governed by glomerular handling. An increased GFR in pregnancy and increased prevalence of albuminuria ultimately results to an increased G.A. formation and clearance in the hyperglycemic environment of gestational diabetes mellitus. Many studies [6,8,11-14,18,24] have well documented the usefulness of G.A. in evaluating the transient hyperglycemic spikes, which account for the long-lasting effects on

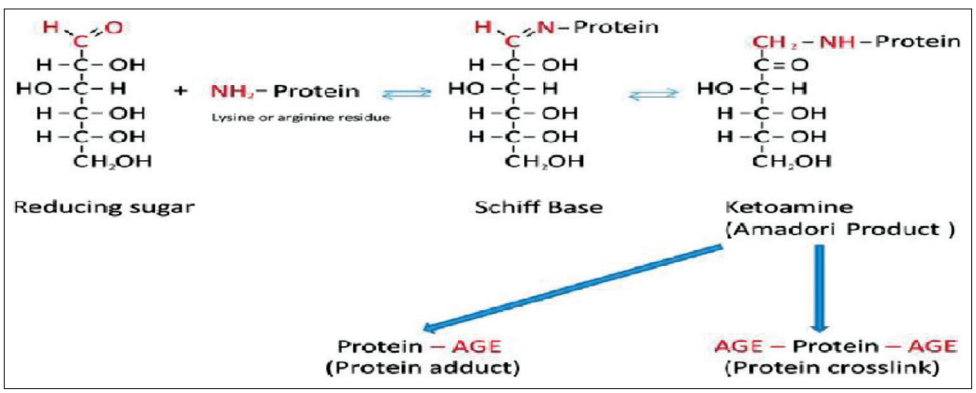


**Figure 4:** Schematic representation of Amadori product formation by Maillard reaction

vascular walls leading to late-stage diabetic vascular complications, more accurately.

Hence UGA % may be used to predict the glycaemic status during early gestational weeks even before the clinical presentation of GDM, which usually manifests in late 2nd or 3rd trimesters. While a high ACR may indicate the extent of albuminuria in both diabetic and non-diabetic progression of chronic kidney disease, UGA may pin-point the imminent and impending event of gestational diabetes mellitus and may help in curbing the fatal complications on both the mother and fetus.

## CONCLUSION

This entire study work essentially tries to highlight that urinary glycated albumin (UGA%) can emerge as a powerful predictive marker for early diagnosis of impending gestational diabetes mellitus, thus curbing the risk and complications at a much earlier stage even before the onset of clinical manifestations. In addition, UGA being a non-invasive marker, may be cost-effective if applied on a mass scale and lessen the hardships of innumerable invasive test procedures during pregnancy.

## ACKNOWLEDGEMENT

The authors take this opportunity to thank GDM Clinic, IPGMER & SSKM Hospital for their whole hearted support for this study

## REFERENCES

1. American Diabetes Association. Gestational diabetes mellitus. Diabetes Care. 2003; 26 Suppl 1: S103-S105.
   https://doi.org/10.2337/diacare.26.2007.S103
2. Rajput R, Yadav Y, Nanda S and Rajput M. Prevalence of gestational diabetes mellitus & associated risk factors at a tertiary care hospital in Haryana. Indian J Med Res. 2013;137(4):728-733.
3. Seshiah V, Balaji V, Balaji MS, Paneerselvam A, Arthi T, Thamizharasi M and Datta M. Prevalence of gestational diabetes mellitus in South India (Tamil Nadu)-a community-based study. J Assoc Physicians India. 2008; 56:329-333.
4. Bellamy L, Casas JP, Hingorani AD and Williams D. Type 2 diabetes mellitus after gestational diabetes: a systematic review and meta-analysis. Lancet. 2009;373(9677):1773-1779.
   https://doi.org/10.1016/S0140-6736(09)60731-5
5. Catalano PM, Kirwan JP, Haugel-de Mouzon S and King J. Gestational diabetes and insulin resistance: role in short- and long-term implications for mother and fetus. J Nutr. 2003;133(5 Suppl 2):1674S-1683S.
   https://doi.org/10.1093/jn/133.5.1674S
6. Hiramatsu Y, Shimizu I, Omori Y, Nakabayashi M and JGA (Japan Glycated Albumin) Study Group. Determination of reference intervals of glycated albumin and hemoglobin A1c in healthy pregnant Japanese women and analysis of their time courses and influencing factors during pregnancy. Endocr J. 2012; 59(2):145-151.
   https://doi.org/10.1507/endocrj.K10E-410
7. de Jong PE, Gansevoort RT and Bakker SJ. Macroalbuminuria and microalbuminuria: do both predict renal and cardiovascular events with similar strength? J Nephrol. 2007;20(4):375-380.
8. Savage S, Estacio RO, Jeffers B and Schrier RW. Urinary albumin excretion as a predictor of diabetic retinopathy, neuropathy, and cardiovascular disease in NIDDM. Diabetes Care. 1996;19(11):1243-1248.
   https://doi.org/10.2337/diacare.19.11.1243
9. Goldin A, Beckman JA, Schmidt AM and Creager MA. Advanced glycation end products: sparking the development of diabetic vascular injury. Circulation. 2006;114(6):597-605.
   https://doi.org/10.1161/CIRCULATIONAHA.106.621854
10. Bundschuh I, Jäckle-Meyer I, Lüneberg E, Bentzel C, Petzoldt R and Stolte H. Glycation of serum albumin and its role in renal protein excretion and the development of diabetic nephropathy. Eur J Clin Chem Clin Biochem. 1992;30(10):651-656.
11. El-Osta A, Brasacchio D, Yao D, Pocai A, Jones PL, Roeder RG, et al. Transient high glucose causes persistent epigenetic changes and altered gene expression during subsequent normoglycemia. J Exp Med. 2008;205(10):2409-2417.
    https://doi.org/10.1084/jem.20081188
12. Vos FE, Schollum JB and Walker RJ. Glycated albumin is the preferred marker for assessing glycaemic control in advanced chronic kidney disease. NDT Plus. 2011;4(6):368-375.
    https://doi.org/10.1093/ndtplus/sfr140
13. Davies CS, Harris CL and Morgan BP. Glycation of CD59 impairs complement regulation on erythrocytes from diabetic subjects. Immunology. 2005;114(2):280-286.
    https://doi.org/10.1111/j.1365-2567.2004.02086.x
14. Friedman S, Jones HW, Golbetz HV, Lee JA, Little HL and Myers BD. Mechanisms of proteinuria in diabetic nephropathy. II. A study of the size-selective glomerular filtration barrier. Diabetes. 198332 Suppl 2:40-46.
    https://doi.org/10.2337/diab.32.2.S40
15. Iberg N and Flückiger R. Nonenzymatic glycosylation of albumin in vivo. Identification of multiple glycosylated sites. J Biol Chem. 1986;261(29):13542-13545.
16. Ghiggeri GM, Candiano G, Delfino G and Queirolo C. Electrical charge of serum and urinary albumin in normal and diabetic humans. Kidney Int. 1985;28(2):168-177.
    https://doi.org/10.1038/ki.1985.137
17. Kowluru A, Kowluru R, Bitensky MW, Corwin EJ, Solomon SS and Johnson JD. Suggested mechanism for the selective excretion of glucosylated albumin. The effects of diabetes mellitus and aging on this process and the origins of diabetic microalbuminuria. J Exp Med. 1987;166(5):1259-1279.
    https://doi.org/10.1084/jem.166.5.1259
18. Guerin-Dubourg A, Catan A, Bourdon E and Rondeau P. Structural modifications of human albumin in diabetes. Diabetes Metab. 2012;38(2):171-178.
    https://doi.org/10.1016/j.diabet.2011.11.002
19. Yong HY, Mohd Shariff Z, Mohd Yusof BN, Rejali Z, Tee YYS, Bindels J, et al. Independent and combined effects of age, body mass index and gestational weight gain on the risk of gestational diabetes mellitus. Sci Rep. 2020;10(1):8486.
    https://doi.org/10.1038/s41598-020-65251-2
20. Erman A, Neri A, Sharoni R, Rabinov M, Kaplan B, Rosenfeld JB, et al. Enhanced urinary albumin excretion after 35 weeks of

gestation and during labour in normal pregnancy. Scand J Clin Lab Invest. 1992;52(5):409-413.

https://doi.org/10.3109/00365519209088376

21. Bomback AS, Rekhtman Y, Whaley-Connell AT, Kshirsagar AV, Sowers JR, Chen SC, et al. Gestational diabetes mellitus alone in the absence of subsequent diabetes is associated with microalbuminuria: results from the Kidney Early Evaluation Program (KEEP). Diabetes Care. 2010;33(12):2586-2591.

    https://doi.org/10.2337/dc10-1095

22. Wong VW, Chong S, Jalaludin B, Russell H and Depczynski B. Urine albumin-creatinine ratio in women with gestational diabetes: its link with glycaemic status. Aust N Z J Obstet Gynaecol. 2014;54(6):529-533.

    https://doi.org/10.1111/ajo.12243

23. Wagner MC, Myslinski J, Pratap S, Flores B, Rhodes G, Campos-Bilderback SB, et al. Mechanism of increased clearance of glycated albumin by proximal tubule cells. Am J Physiol Renal Physiol. 2016;310(10): F1089-F1102.

    https://doi.org/10.1152/ajprenal.00605.2015

24. Inaba M, Okuno S, Kumeda Y, Yamada S, Imanishi Y, Tabata T, et al. Glycated albumin is a better glycemic indicator than glycated hemoglobin values in hemodialysis patients with diabetes: effect of anemia and erythropoietin injection. J Am Soc Nephrol. 2007 18(3):896-903.

    https://doi.org/10.1681/ASN.2006070772

**Author's Contribution:**
**CP, SM-** Planned the study; **CP-** Collected the data and wrote the report; **SB, SM, KG-** Analysed, edited and revised the manuscript. All authors: involved in the conception, design, write-up, and revision of this article.

**Work attributed to:**
Department of Biochemistry and Obstetrics and Gynaecology, Institute of Post Graduate Medical Education and Research, Kolkata, India.

**Orcid ID:**
Dr. Chandramallika Paul- https://orcid.org/0000-0002-1558-1878
Prof. Santanu Banerjee- https://orcid.org/0000-0002-0960-2900
Prof. Satinath Mukhopadhyay- https://orcid.org/0000-0001-8033-5748
Prof. Kalyan Goswami- https://orcid.org/0000-0002-5052-0675

**Source of Funding:** Nil, **Conflict of Interest:** None declared.