

Serum Pentosidine as a Potential Biomarker of Vascular Complications in Type 2 Diabetes Mellitus: A Case-Control Study

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ABSTRACT

Background: Diabetes mellitus is a type 2 results in progressive vascular damage due to hyperglycaemia, oxidative stress, inflammation, endothelial dysfunction, and advanced glycation. Pentosidine, an advanced glycation end product, could be a surrogate marker for total glycoxidative stress that cannot be explained by traditional glycemic parameters. The study aim to assess the serum levels of pentosidine and its correlation with vascular complications in type 2 diabetes mellitus.

Methods: The study was conducted among 150 patients for the hospital-based case-control study that comprised 50 healthy individuals, 50 T2DM patients without complications, and 50 patients with complications from T2DM. The concentration of serum pentosidine was determined by means of competitive ELISA, while HbA1c, fasting blood glucose, and postprandial blood glucose levels were measured.

Results: The study findings showed the 33.06 ng/ml in uncomplicated T2DM subjects and 301.48 ± 26.42 ng/ml in complicated T2DM ($P < 0.001$). Also HbA1c increased from $4.82 \pm 0.55\%$ to $8.68 \pm 0.59\%$ and $10.82 \pm 0.58\%$. PEN was noted to show correlation with diabetes duration ($r=0.752-0.816$) and HbA1c ($r=0.731-0.769$).

Conclusion: The study concluded that high level of serum pentosidine was related with poor glycemic control and diabetic complications, contributing to the efficacy as an adjunctive biomarker.

Key-words: Pentosidine; Type 2 diabetes mellitus; Vascular complications; Glycemic control; Advanced glycation end-products

INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a state of chronic metabolic stress which gradually damages the vascular system by interacting pathways of hyperglycaemia, oxidative stress, inflammation, endothelial dysfunction

and advanced glycation end-product (AGE) accumulation. The main parameters to assess risk conventionally are glycated haemoglobin (HbA1c), blood pressure, lipid profile, estimated glomerular filtration rate (eGFR), and urinary albumin excretion, but these parameters are not sufficient to define cumulative glycaemic injury or susceptibility to vascular complications. This limitation has rendered biochemical markers of combined long-term glycation and glycoxidative stress interesting. Of these, pentosidine is an AGE that is cross-linking and fluorescent, formed from oxidative reactions of reducing sugars with amino acid residues and may provide a way to gain information

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beyond that of blood glucose or HbA1c levels but also about chronic tissue and vascular glycation^[1-3]. These are thought to be especially important in diabetes, because they contribute to extracellular-matrix cross-linking, stiffening of the blood vessels, alterations in endothelial signaling, production of reactive oxygen species and activation of the AGE–receptor for advanced glycation end products (RAGE) pathway. These processes trigger NF-κB dependent inflammatory signaling, deplete nitric oxide availability and drive endothelial dysfunction and atherosclerotic vascular remodeling^[1,2,4].

An association between AGE burden and diabetic vascular disease is becoming well established with clinical evidence. In the large patient cohort of 1471, who had T2DM, there was a graded correlation between higher quartiles of AGE and increasing severity of DR by SAF with an area under the receiver operating characteristic curve of 0.728^[5] for vision-threatening DR. In a similar manner, circulating AGEs have been shown to be related to renal deterioration. The composite AGE score was independently associated with 30% reduction in eGFR (HR 1.42, 95% CI 1.13-1.78), macroalbuminuria (HR 1.53, 95% CI 1.13-2.06) and high-risk chronic kidney disease (HR 1.88, 95% CI 1.37-2.57) in the ACCORD cohort^[6].

Pentosidine could be particularly useful as it is a chemically-defined AGE, not a composite of signals of fluorescence. In the T2DM, pentosidine has been reported as associated with structural and functional changes of the cardiovascular system. One cross-sectional study showed that there was a relationship between pentosidine and the increase in brachial-ankle pulse-wave velocity (PWV) and arterial root diameter and a decrease in left ventricular ejection fraction (LVEF) in 122 T2DM patients^[7]. In addition, the analysis of blood concentrations of pentosidine and vascular events has shown possible association of pentosidine to macroangiopathic diseases and pulse-wave velocity that reflects system-wide vascular damage^[8]. In addition, it has recently been shown that total and free circulating AGEs levels are elevated in diabetic patients with microvascular and macrovascular diabetic complications which further supports the biological plausibility of using specific molecules of AGEs as adjunctive biomarkers^[9].

Therefore, a case-control study of serum pentosidine in T2DM with and without vascular complications would be helpful in elucidating any biochemical differences in the

presence of vascular complication in addition to those metabolic parameters. The objective of the present study was to investigate the association between pentosidine with vascular complications in T2DM and its potential as a secondary biomarker in patients at higher risk for vascular complications.

MATERIALS AND METHODS

Study design- The study was hospital-based case-control study which was conducted to analyze Serum Pentosidine as an effective biomarker of vascular complications among Type 2 Diabetes Mellitus. The study was conducted among 150 patients, with 100 patients as type 2 diabetes mellitus (T2DM) and 50 patients as healthy control. Diabetic patients were categorized as 50 cases without vascular complications and 50 cases each with microvascular or macrovascular complications, consisting of diabetic retinopathy, nephropathy, neuropathy, coronary artery disease, and stroke. The study was conducted in the tertiary care hospital, for duration of 1 year. Both written and verbal consent required for the study. Patients were selected on the basis of the specific predefined inclusion and exclusion criteria.

Inclusion criteria

- The adults of age 35 to 74 years of type 2 diabetes mellitus
- T2DM patients with or without vascular complications
- Patients with complications including retinopathy, nephropathy, or neuropathy
- Patients with macrovascular complications such as coronary artery disease or stroke was included
- Informed consent required for the study

Exclusion criteria

- Patients with the previous history of history of acute or chronic illnesses were excluded
- Uninformed consent were excluded

Procedure- The study ethical approval was obtained and written informed consent from the participants was selected. Subjects were classified as either T2DM subjects without any vascular complications, T2DM subjects with microvascular/macrovascular complications or age/gender-matched healthy controls.

Blood sample in the volume of approximately 5 mL was collected through venipuncture under aseptic precautions. The samples were subjected to centrifugation at 3000 rpm for 15 minutes; serum/plasma was collected and stored in -20°C until used for analyses. Levels of PEN were analyzed by ELK Biotechnology Pentosidine Kit with competitive ELISA method. Measurement of HbA1c, Fasting blood glucose (FBG), and Post prandial blood glucose (PPBG) was conducted out at the Central Clinical Laboratory by the use of the Beckman Coulter AU5800 Series Clinical Chemistry Analyzer.

Statistical analysis- Data analysis was performed using SPSS version 27. Continuous variables were expressed as mean $\pm$ standard deviation (SD). The independent t-test was used to compare continuous variables, and Pearson's correlation was evaluated between PEN and clinical or biochemical parameters. Multiple linear and logistic regression analyses were conducted to identify

independent predictors and factors related to diabetes. A p-value <0.05 was considered statistically significant.

RESULTS

Table 1 demonstrated the difference in the demographic and clinical parameters for the 3 study groups. The mean value of age enhanced from 48.72 ± 9.12 years controls to 50.46 ± 9.41 years in cases without complications and 57.28 ± 9.52 years for cases with complication, which indicated old age profile. Males were 52.0%, 50.0%, and 56.0% for the groups. The mean value of diabetes was high among cases with complications (9.52 ± 4.68 years), rather than those without complications (2.34 ± 1.62 years). Patients with diabetic cases showed high BMI, ($26.54\text{--}26.88 \text{ kg/m}^2$) compared to control ($24.68 \pm 3.08 \text{ kg/m}^2$), which suggested high adiposity among diabetes (Table 1).

Table 1: Comparison of Demographic and Clinical Characteristics among Controls, Cases without Complications, and Cases with Complications

Parameters	Controls (n=50)	Cases without Complications (n=50)	Cases with Complications (n=50)	p-value (a-b)	p-value (a-c)	p-value (b-c)
Age (Years)	48.72 ± 9.12	50.46 ± 9.41	57.28 ± 9.52	-	-	-
Male, n (%)	26 (52.0%)	25 (50.0%)	28 (56.0%)	-	-	-
Female, n (%)	24 (48.0%)	25 (50.0%)	22 (44.0%)	-	-	-
Duration of Diabetes (Years)	-	2.34 ± 1.62	9.52 ± 4.68	-	-	-
BMI (kg/m^2)	24.68 ± 3.08	26.54 ± 2.61	26.88 ± 2.76	-	-	-

Table 2 demonstrated marked differences for the PEN and glycemic parameters and the mean value of PEN enhanced from $122.64 \pm 25.18 \text{ ng/ml}$ in controls to $289.72 \pm 33.06 \text{ ng/ml}$ in cases without complications and $301.48 \pm 26.42 \text{ ng/ml}$ cases with complications. HbA1c enhanced with increase $4.82 \pm 0.55\%$ in control and $8.68 \pm 0.59\%$ and $10.82 \pm 0.58\%$. FBG enhanced from $85.14 \pm 7.64 \text{ mg/dl}$ to $120.46 \pm 18.72 \text{ mg/dl}$ and $133.18 \pm 13.26 \text{ mg/dl}$. The PPBG enhanced from $123.72 \pm 8.06 \text{ mg/dl}$ to $148.64 \pm 16.42 \text{ mg/dl}$ and $160.28 \pm 14.38 \text{ mg/dl}$ (Table 2).

Table 3 indicated with high positive relationship between the PEN levels and the duration of the T2DM in the case group ($r = 0.752$) and control group ($r = 0.816$). Also high positive correlations between HbA1c and the concentration of PEN ($r = 0.731$ and 0.769 , respectively). This indicated a significant relationship between the concentration of PEN and poor glycemic control in T2DM patients. The table showed moderate correlations between the FBG and the concentration of PEN in cases without complications ($r = 0.671$) and weak correlations among cases with complications ($r = 0.238$). There was no relationship between BMI and the concentration of PEN ($r = 0.061$ and 0.014 , respectively) (Table 3).

Table 2: Comparison of Proenkephalin, Glycemic Control, Fasting Blood Glucose, and Postprandial Blood Glucose Levels among Controls and Diabetic Cases With and Without Complications

Parameters	Controls (a) (n=50)	Cases without Complications (b) (n=50)	Cases with Complications (c) (n=50)	p-value (a-b)	p-value (a-c)	p-value (b-c)
PEN (ng/ml)	122.64 ± 25.18	289.72 ± 33.06	301.48 ± 26.42	<0.001	<0.001	<0.05
HbA1c (%)	4.82 ± 0.55	8.68 ± 0.59	10.82 ± 0.58	<0.001	<0.001	<0.001
FBG (mg/dl)	85.14 ± 7.64	120.46 ± 18.72	133.18 ± 13.26	<0.001	<0.001	<0.001
PPBG (mg/dl)	123.72 ± 8.06	148.64 ± 16.42	160.28 ± 14.38	<0.001	<0.001	<0.001

Table 3: Correlation of PEN Levels with Duration of Type 2 Diabetes, BMI, and Glycemic Parameters among Cases With and Without Complications

PEN v/s Parameters	Cases without Complications (r value)	Cases with Complications (r value)
T2DM Duration	0.752	0.816
BMI	0.061	0.014
HbA1c	0.731	0.769
FBG	0.671	0.238
PPBG	0.114	0.137

Table 4 showed the multivariate regression analysis and both of the PEN and HbA1c was noted to be significant predictor. PEN showed positive regression coefficient of 0.021 ± 0.009 and the t value as 2.42 with P = 0.0168, which indicated the independent association. HbA1c

showed the highest relation among all variables along with regression coefficient of 2.8764 ± 0.226 , t = 12.73, and p < 0.0001. FBG showed the positive coefficient of 0.03042. The regression coefficient of PPBG is -0.00912 and also not significant (Table 4).

Table 4: Multivariate Regression Analysis of PEN and Glycemic Parameters Associated with the Study Outcome

Parameters	Coefficient	SE	95% CI	t value	p-value
PEN	0.021	0.009	0.003–0.039	2.42	0.0168
HbA1c	2.8764	0.226	2.431–3.322	12.73	<0.0001
FBG	0.03042	0.016	-0.001–0.062	1.9	0.0589
PPBG	-0.00912	0.017	-0.042–0.024	-0.54	0.5901

Table 5 showed the binary logistic regression analysis revealed that the predictors of complications in diabetes mellitus patients included PEN, HbA1c, and FBG. The predictor PEN had a positive correlation with the presence of complications and had a coefficient of 0.086, an odds ratio (OR) of 1.09, and P value of 0.0278, indicated the higher likelihood of complications as PEN

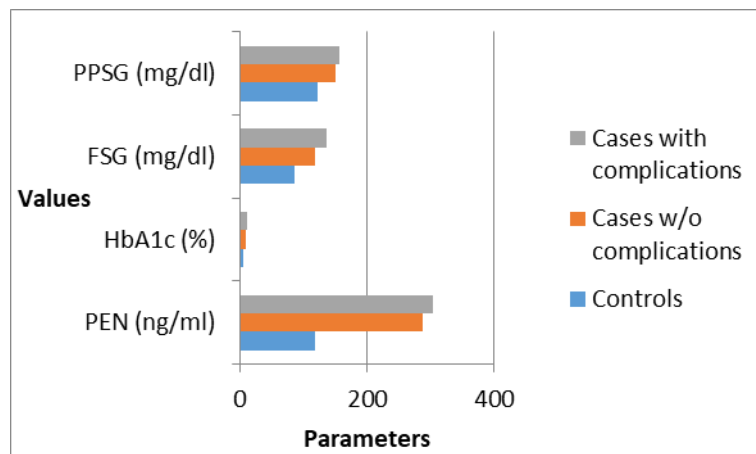
values. HbA1c had the highest positive correlation with the complications, with the predictor having a coefficient of 5.62, OR of 275.4, and p-value less than 0.0001. FBG also showed a significant correlation with complications (coefficient 0.105, OR 1.11, p = 0.0322). PPBG showed a weak correlation (OR 1.03, p-value = 0.3620) (Table 5).

Table 5: Binary Logistic Regression Analysis of PEN and Glycemic Parameters Associated with Diabetic Complications

Parameters	Coefficient	SE	Wald Test	p-value	Odds Ratio	95% CI
PEN	0.086	0.039	4.84	0.0278	1.09	1.01–1.18
HbA1c	5.62	1.08	27.05	<0.0001	275.4	33.26–2280.17
FBG	0.105	0.049	4.59	0.0322	1.11	1.01–1.22
PPBG	0.031	0.034	0.83	0.362	1.03	0.96–1.10

Fig. 1 showed the progressive increase for the 4 variables from the controls to uncomplicated cases to complicated cases in the form of a stepwise pattern. The level of PEN increased from 118.45 ng/ml in controls to 287.30 ng/ml in uncomplicated cases and 304.12 ng/ml in complicated cases with an increase of over 2.5 times. The levels of HbA1c increased from 5.02% to 8.45% and 10.98%,

reflecting increasing poor glycemic control. There were increases in the values of fasting serum glucose from 86.10 to 118.75 to 135.40 mg/dl and postprandial serum glucose from 121.80 to 150.20 to 156.35 mg/dl. Highest increase between the controls and the cases group, while the variation between the two cases groups were relatively smaller (Fig. 1).

**Fig. 1:** Comparison of PEN, HbA1c, FSG and PPSG levels among controls, cases without complications and cases with complications

DISCUSSION

The current case-control study assesses the association between pentosidine and vascular complications in T2DM patients. This association is biologically supported as pentosidine is a stable fluorescent AGE formed during glycoxidative reactions that may be a marker of past metabolic stress rather than recent glycaemia. Unlike HbA1c, which primarily reflects glycaemic exposure over about the last 2–3 months, factors affecting AGE accumulation include prolonged hyperglycaemia, oxidative stress, carbonyl stress, renal clearance and tissue turnover. Thus, increased pentosidine levels might reflect a part of chronic vascular injury that is poorly captured by other traditional glycaemic indices^[1,2]. Thus, the major implication of this study should be directed at the association of pentosidine with vascular

complications, rather than at the study of pentosidine as a single parameter of hyperglycaemia. There are two complementary pathways by which AGE are associated with vascular pathology. Non-enzymatic glycation changes long-lived proteins like collagen and elastin by creating intermolecular cross-links that result in greater arterial stiffness. Second, AGE–RAGE interaction is linked to intracellular oxidative and inflammatory pathways, such as NADPH oxidase and nuclear factor- κ B signalling, which can raise the production of reactive oxygen species, cytokines of inflammation, endothelial adhesion molecules and vascular permeability^[2,4]. These mechanisms provide a plausible explanation for an association between increased serum pentosidine and clinical vascular disease.

The results are in agreement with recent clinical data on circulating pentosidine. In the DIABAGE study, pentosidine has been associated significantly with coronary disease and overall macro angiopathy, and correlated with pulse-wave velocity, which has been shown to be an established surrogate of arterial stiffness [8]. In other words, pentosidine was linked to the development of coronaropathy (OR 1.61) as well as any macroangiopathy (OR 1.52). The correlation between pentosidine and PWV was $r=0.28$ ($P<0.001$) suggesting a relationship between the circulating AGE burden and the mechanical abnormalities of the vessels. The conclusions were corroborated by a separate T2DM study of 122 patients that found serum pentosidine was positively correlated with brachial-ankle pulse-wave velocity and various measures of vascular and cardiac remodelling, but inversely correlated with left ventricular ejection fraction [7].

The clinical relevance of the association of AGE accumulation and microvascular disease is also significant. In a study of 1,471 patients with T2DM, elevated AGE levels, by skin autofluorescence, were correlated with the severity of DR. The odds ratios for retinopathy in the different AGE quartiles were 1.00, 1.69, 1.58 and 1.60, respectively, and the trend was significant; AGE measurement also showed an AUC of 0.728 for diabetic retinopathy [5]. Skin autofluorescence does not directly measure serum pentosidine, but is a marker of systemic AGE accumulation, clinically associated with retinal microangiopathy. Mechanistic link is endothelial dysfunction, injury of the pericytes, oxidative stress, inflammatory signalling, and disruption of blood-retinal barrier due to AGE [11].

Consideration should be given to renal disease in interpreting serum pentosidine. The kidney plays an important role in the elimination of circulating products of AGE and a decrease in renal function can lead to elevated circulating levels of AGE products. A large study of patients with T2DM showed that the composite AGE score was independently associated with a worsening of renal function, 30% eGFR decline, macroalbuminuria and high-risk CKD after accounting for HbA1c and established clinical factors [6]. Likewise, an increase in AGE concentrations has been seen with the progression of CKD in subsequent studies, and some free AGE species are found to have particularly high increases in advanced renal disease [12]. Accordingly, when the present study

shows increased pentosidine in those with vascular complications, care should be taken to not conclude that the overall increase is due to vascular disease, but rather that renal function is an important variable to consider as a potential confounder.

There are also longitudinal and meta-analytic supporting evidence of cardiovascular relevance of AGEs. Data from 14 prospective studies (79,389 individuals) revealed that increased AGE levels (measured by SA) were significantly linked to MACE, with a pooled HR of 1.54 (95% CI 1.31–1.81). This association was more pronounced in individuals with diabetes, with an HR of 1.88 (95% CI 1.31 – 2.69) [13]. Importantly, this evidence is for AGE burden, and not for pentosidine alone, so should not be interpreted as evidence that pentosidine is an independent predictor of cardiovascular events in its own right.

Analytical research in recent years also supports the idea that individual AGE compounds should also be investigated, and not just global measurements of AGEs. In a 2024 study, using a liquid chromatography–tandem mass spectrometry, the concentration of free and total AGEs was found to be higher in patients with both microvascular and macrovascular diabetic complications [9]. However, kidney studies with prospective data indicate that composite AGE measures may be superior to AGE compounds in predicting renal decline [6]. This is significant because pentosidine may have biological specificity, whereas a multimarker AGE panel may have wider predicting power due to the different precursor pathways of different AGEs and their differential sensitivity to renal function and metabolic status.

The present results should therefore be considered as supportive data to accompany pentosidine as a potential adjunctive biomarker, rather than an alternative to the use of HbA1c, lipid parameters, blood pressure, urinary albumin to creatinine ratio, eGFR, retinal assessment or established cardiovascular risk assessment. Recent reviews highlight the high analytical heterogeneity of AGE measurement, including variations in biological matrices, assay platforms, calibration protocols, and molecular targets of the assays [14,15]. In a recent systematic review and meta-analysis published in 2026, high levels of AGEs were consistently linked to diabetic microvascular and macrovascular studies, with an accent on the variability in measurement methods of AGEs [15].

Overall, biological and clinical findings suggest a relationship between pentosidine related glycoxidative burden and diabetic vascular damage. The major strength of this case-control design is that it could identify any systemic differences in serum pentosidine levels in T2DM patients with and without vascular complications and whether this difference remained after adjusting for variables including age, diabetes duration, HbA1c, renal function, hypertension and dyslipidaemia. Larger, prospective studies with standardized LC-MS assays and with longitudinal outcomes of the vasculature are needed before serum pentosidine can be used in routine clinical risk stratification.

CONCLUSIONS

The study concluded with the relationship between high PEN levels, bad glycemic control and diabetic complications in patients with T2DM. The levels of PEN gradually increased from controls (122.64 ± 25.18 ng/ml), cases without complications (289.72 ± 33.06 ng/ml) and cases with complications (301.48 ± 26.42 ng/ml) and had significant differences between the groups. There were significant differences in the levels of HbA1c, fasting blood glucose (FBG) and postprandial blood glucose (PPBG) between the groups ($p < 0.001$). There were strong positive correlations of PEN with duration of T2DM and HbA1c especially in cases with complications while there was no correlation with BMI. From multiple regression analysis, it was found that PEN and HbA1c were significant independent predictors of the study outcome. Also, binary logistic regression analysis found that high levels of PEN, HbA1c and FBG were significantly related to the risk of diabetic complications but there was no significance in relation with PPBG.

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