ASIAN JOURNAL OF PHARMACEUTICAL AND CLINICAL RESEARCH

INNOVARE ACADEMIC SCIENCES
Knowledge to Innovation

Vol 18, Issue 7, 2025

Online - 2455-3891
Print - 0974-2441

Research Article

PENTRAXIN LEVELS AS PREDICTORS OF CARDIOVASCULAR RISK IN TYPE 2 DIABETES MELLITUS

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Received: 23 February 2025, Revised and Accepted: 23 May 2025

ABSTRACT

Objectives: Pentraxin-3 (PTX3) is an acute phase protein, considered as a vascular proinflammatory reflector, and its link with dyslipidemia environment and oxidative stress truths the risk of cardiovascular disease. This study evaluates the association of PTX3 and atherogenic markers (total cholesterol [TC]/high-density lipoprotein [HDLc], low-density lipoprotein [LDLc]/HDLc, triglycerides [TGL]/HDLc, non-HDLc, atherogenic coefficient) in normoalbuminuric micro and macroalbuminuria diabetic individuals.

Methods: The analytical observational research work included 86 participants with a duration of diabetes mellitus <5 years without complications. Participants were grouped based on glycated hemoglobin and urine albumin creatinine ratio as normoalbuminuric, micro, and macroalbuminuria. Lipid profile analytes were measured in the Beckman Coulter AU series and PTX3 was analyzed using a standard kit. The atherogenic coefficient is the ratio between non-HDLc and HDLc that epitomizes cardiovascular risk.

Results: Elevated PTX3 values in poor glycemic diabetic individuals with albuminuria patient. PTX3 results statistically correlated positively with TC/HDLc, TGL/HDLc, non-HDLc/HDLc (atherogenic coefficient), and non-HDLc. These findings strengthen the potential link between PTX3 and atherogenic risk in diabetic patients with poor glycemic status. The receiver's operative characteristics curve supported PTX3 cut-off of 2.49 ng/mL, the area under the curve was 0.739 with a diagnostic sensitivity of 79.1% and specificity of 72.1%, with a significant p value.

Conclusion: PTX3 is considered a promising marker of the inflammatory environment due to poor glycemic and lipidemic environment, thereby relating to the likelihood of complications in diabetes. This study substantiates that PTX3 is a valuable marker for assessing early cardiovascular risk and emphasizes the importance of lifestyle modification and therapeutic intervention.

Keywords: Atherogenic coefficient, Cardiovascular risk, HDL cholesterol, Pentraxin 3, Type 2 diabetes mellitus.

 DOI: http://dx.doi.org/10.22159/ajpcr.2025v18i7.54053. Journal homepage: https://innovareacademics.in/journals/index.php/ajpcr

INTRODUCTION

On a global scale diabetes mellitus (DM) appears to be the primary contributor of rising morbidity and mortality. A rise in uncontrolled diabetic status accelerates the pathological root toward vascular complications. The predominant trend of occurrence of DM had increased following the global covid pandemic threat [1]. Chronic hyperglycemia leads to disastrous vascular complications built due to the overproduction of advanced glycation end products and decreased nitric oxide availability. Pre-clinical studies emphasize the current pressing challenge as endothelial dysfunction contributed by an intricate pathological mechanism that integrates uncoupling of nitric oxide synthase and endothelial enflamed inflammatory status characterized by inflammatory molecular patterns, high oxidized lipoprotein, and cytokine levels [2,3]. This imbalance of vasodilators and vasoconstrictors vascular endothelial inflammatory state in turn contributes to cardiovascular disease (CVD).

Pentraxin 3 (PTX3) is an acute-phase protein that forms a part of the pentraxin superfamily. As to foil to CRP, which is secreted by hepatocytes activated by interleukin-6 (IL6), recent studies figure that PTX3 excites in atherosclerotic plaques, macrophages, and endothelial cells [4]. PTX3 gets liberated in response to inflammation, thus chiefly known as the "facilitator of acute inflammation". This has been demonstrated that a number of inflammatory stimuli, such as lipopolysaccharide, IL 1β, and tumor necrosis factor, elevate PTX3 levels.

In context with diabetic patients, the hyperglycemic status vitalizes inflammatory reaction, inducing the release of PTX3 to manage inflammation. The pentraxin level mirrors the vascular inflammatory reaction which may serve as an ideal marker to monitor diabetics. The levels of this peptide also root higher in patients with dyslipidemia environment. One of the most common fundamental pathology of CVD is atherosclerosis which is tightly linked to a chronic low-grade inflammatory environment. As a concern to medical and public health research, its prime importance to score the inflammatory marker of atherosclerosis is the need of the hour to predict CVD. As per the "Kidney Disease Outcomes Quality Initiative (KDOQI) guidelines" urine albumin creatinine ratio clearly reflects the pathological processes related to endothelial dysfunction and diffuse vascular damage; thereby linked to cardiovascular risk [5].

Studies have explored the diverse vascular effects of PTX3, as a vascular reflector to proinflammatory signals, positive association with dyslipidemia effects, and oxidative stress marker [6]. This study evaluates the association of PTX3 and atherogenic markers (TC/HDLc, LDLc/HDLc, TGL/HDLc, non-HDLc, atherogenic coefficient) in type 2 diabetes with normoalbuminuria, microalbuminuria and macroalbuminuria.

METHODS

Design

The research work was an analytical cross-sectional study design carried out from November 2023 to September 2024. With Departmental Scientific Committee consensus, the research proposal cleared the College Scientific Committee and Institutional Ethical Committee approval.

Ethical approval

Each participant was briefed of the objectives of the research study as part of the protocol and received informed consent before commencement of the work. The traceability of the Institutional Ethics Committee approval number is ST0923-700.

The sample size has been derived with the formula, n=[n=(Z 1−α/2+Z 1−β)^2 (2S)^2/d2] utilizing the mean and SD of PTX3 in normal and DM, 4.11±2.4 ng/mL and 4.26±2.2 ng/mL respectively [7]. ($Z_{\alpha/2}$=1.96), Z 1−β=(1.65), S=SD1 + SD2, d=mean1 – mean2, by applying the formula sample size of 43was calculated.

A total of 86 patients presenting to the general medicine outpatient department with symptoms of type 2 DM were selected. Participants were allotted as Group A with glycated hemoglobin (HbA1c)<6.5% and Group B with HbA1c≥6.5%; whereas the participants with chronic kidney disease, hypertension, acute inflammatory disease, CVD were excluded.

Laboratory investigations

Blood samples were collected following an overnight fasting of 10–12 h. Biochemical variables were assayed in Beckman Coulter DXC 700 using dedicated reagents. Fasting plasma glucose levels were estimated by hexokinase enzymatic method, Lipid parameters such as total cholesterol, triglycerides, HDL-cholesterol, and LDL-cholesterol were measured by a standardized method in Beckman Coulter DXC700 integrated autoanalyzer. Serum pentraxin levels were measured in the Bio-Rad enzyme-linked immunosorbent assay (ELISA) technique using the Human ELISA kit. This ELISA assay kit had a sensitivity of 0.113 ng/mL. This assay showed a strong reliability with both intra-assay <8% and inter-assay coefficient of variance of <10%. The atherogenic coefficient or index is the measure of risk to develop CVD which is calculated as the difference of total cholesterol and HDLc divided by HDLc. Normal healthy individuals have an atherogenic coefficient between 2 and 2.5 units [8]. Urine albumin creatinine ratio with a cutoff <30 µg/mg of creatinine was considered normoalbuminuric.

Statistics

The Statistical Package for the Social Sciences (SPSS 22.0) was employed to analyze the data. The Shapiro–Wilk test was employed for checking the data for normality, and the results showed that they were not. As a result, non-parametric techniques were implemented for analysis. Mann–Whitney U test was used for comparing the difference and measured in the Inter-Quartile range across the groups. Correlation of the analyzed clinical chemistry analytes has been accomplished through spearman's correlation. The receiver operating characteristic curve was utilized to determine the diagnostic sensitivity and specificity of the analyte of interest PTX3 as a biochemical marker of vascular endothelial damage in DM.

RESULTS

The participants were type 2 DM attending the Diabetology outpatient wing of SRM Medical College Hospital and Research Centre. By consecutive sampling technique, 86 participants were included in the research study. Out of 86 participants, 46.5% were men and 53.5% were women presented with <5 years of duration of DM. Following the general physical examination of the participants, basic anthropometric details were recorded in the patient proforma sheet. As per Table 1: Type 2 diabetic men had a body mass index (BMI) with a median of 27.69 kg/m^2 compared to women with aBMI of median value of 22.2 kg/m^2

In this study, median PTX3 value in men was 3.08 ng/mL with interquartile range (2.59, 3.36) whereas in women the median PTX value was 1.78 ng/mL with interquartile range (1.52, 2.08) Fig. 1.

Table 3 represents the statistically significant elevation of PTX3, atherogenic indices, and atherogenic coefficient in T2DM macroalbuminuric individuals.

Table 1: Comparison of anthropometric data in type 2 diabetes mellitus

Parameter	Male (n=40)	Female (n=46)	p-value
Height (m^2)	1.63 (1.59, 1.70)	1.53 (1.48, 1.58)	<0.001**
Weight (kg)	69.2 (63.8, 75.7)	67 (61.3, 70.1)	0.106^{NS}
Body mass index Kg/m^2	26.2 (24.1, 27.8)	27.9 (26.1, 30.4)	0.014*

MannWhitney U test: results are expressed in median (interquartile range - IQR), p<0.05 - statistically significant, NS indicating non-significant results, *significant findings, **highly significant findings (p<0.001)

Table 2: Comparison of HbA1c and pentraxin values in men and women

Parameter	Men n=40	Women n=46	p-value
HbA1c (%)	6.9 (6.6, 8.8)	7.45 (6.57, 8.85)	0.665 (NS)
Pentraxin (ng/mL)	3.08 (2.59, 3.36)	1.78 (1.52, 2.08)	0.001**

Mann–Whitney U test: median (interquartile range - IQR), NS indicating non-significant results **high significant result (p<0.001). HbA1c: Glycated hemoglobin

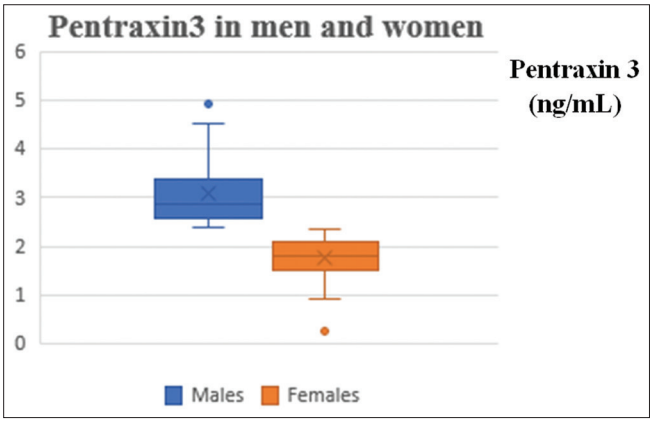

Fig. 1: Pentraxin levels in type 2 diabetes mellitus

According to Table 4, based on urine albumin creatinine ratio (urine ACR) the participants were sub-categorized as Group I with Urine ACR≤30 µg/mg of creatinine, Group II with urine ACR more than 30–300 µg/mg of creatinine and Group III with urine ACR more than 300 µg/mg of creatinine. The data comparison based on the urine albumin creatinine ratio, HDL-based lipid ratios non-HDLc, TC/HDLc, TGL/HDLc, TC-HDL/HDL (Atherogenic coefficient) revealed statistically significant median difference between Group I and III through Kruskal–Wallis test and subsequently *post-hoc* analysis. Fig. 2 shows the graphical representation of PTX3 data in T2DM sub-categorized based on the severity of urine ACR. In this study significantly elevated PTX3 levels were observed in macroalbuminuric as compared with normoalbuminuric or microalbuminuria individuals.

Table 5 revealed a positive correlation of PTX3 with atherogenic parameters and a negative correlation with HDLc, minimal positive correlation with TC/HDL (r=0.255), and atherogenic coefficient (r=0.22),whereas PTX3 revealed a statistically significant positive correlation with urine albumin creatinine ratio (r=0.355).

Area under the curve analysis

As in Fig. 3 and Table 6, the Receiver's operating characteristics of PTX3 revealed that at a cut-off of 2.49 ng/mL, area under the curve was 0.739 with a diagnostic sensitivity of 79.1% and specificity of 72.1%, with significant p-value. Frequency distribution revealed that 55.81% of type 2 DM had PTX3 results more than the diagnostic

Table 3: Comparison of pentraxin and atherogenic coefficient in diabetic individuals based on HbA1c levels

Parameters	HbA1c<6.5% (n=32)	HbA1c≥6.5% (n=54)	p-value
Total cholesterol (mg/dL)	145.5 (118.5, 167.75)	160.85 (132.25, 182.75)	0.04*
Triglycerides (mg/dL)	119.82 (81.75, 145.25)	135.24 (90.5, 144.5)	0.302(NS)
HDLc (mg/dL)	41.6 (34.75,45.25)	42.51 (34.25, 51)	0.57(NS)
LDLc (mg/dL)	101.64 (77.75,123.25)	105.74 (83, 117.75)	0.59(NS)
Non-HDLc (mg/dL)	103.89 (83, 124)	118.33 (88.25, 148.3)	0.02*
TC/HDLc	3.57 (3.02, 4.15)	3.83 (3.2, 4.4)	0.039*
TGL/HDL	3.03 (2.05, 3.53)	3.35 (2.2, 3.67)	0.34(NS)
LDL/HDL	2.52 (1.87, 3.18)	2.5 (1.97, 2.91)	0.98 (NS)
Atherogenic coefficient	2.2 (1.75, 2.92)	2.82 (2.2, 3.4)	0.048*
PTX3 (ng/mL)	2.13 (1.68, 2.49)	2.52 (1.85, 2.99)	0.037*
Variables	FPG: Fasting plasma glucose, PPPG: Post prandial plasma glucose, HbA1c: Glycated hemoglobin, LDL: Low density lipoprotein, HDL: High density lipoprotein, uACR: Urine albumin creatinine ratio, PTX3: Pentraxin-3		

Table 4: Data comparison of HbA1c, lipid atherogenic parameters and pentraxin-3 in type 2 diabetes mellitus

Parameters	Urine ACR<30 (n=40)	Urine ACR 30–300 (n=36)	Urine ACR>300 (n=10)	p-value
FPG (mg/dL)	122.5 (101.25, 150)	140 (123.5, 178.5)	187.5 (179.5, 239.5)	0.001**
PPPG (mg/dL)	174 (131.25, 235.5)	217 (191.25, 267)	279 (241, 295.25)	0.001**
HbA1c (%)	6.8 (6.25, 7.37)	8.1 (7.0, 9.92)	10.95 (9.62, 11.87)	0.001**
NON-HDL (mg/dL)	103.5 (85.25, 127.75)	112 (75.5, 128.5)	134 (121.75, 169)	0.039*
TC/HDL	3.51 (3.002, 4.26)	3.40 (3.08, 4.36)	5.41 (4.27, 5.82)	0.001**
TGL/HDL	2.85 (1.90, 3.58)	2.78 (2.18, 3.19)	3.88 (3.46, 5.11)	0.025*
LDL/HDL	2.35 (2, 3.26)	2.30 (1.90, 3.05)	2.96 (2.67, 3.42)	0.077 (NS)
Atherogenic coefficient	2.51 (2, 3.26)	2.40 (2.08, 3.36)	4.41 (3.27, 4.82)	0.001**
uACR (µg/mg of creatinine)	8 (5.25, 14.95)	65 (39.5, 130)	824.5 (430, 142)	0.001**
PTX3 (ng/mL)	2.2 (1.91, 2.4)	2.5 (1.8, 3.2)	3.41 (2.6, 3.6)	0.001**
Variables	FBG: Fasting plasma glucose, PPBG: post prandial plasma glucose, HbA1c: Glycated hemoglobin, uACR: Urine albumin creatinine ratio, PTX3: Pentraxin-3			

Kruskal–Wallis test: values are given in median (interquartile range - IQR), p<0.05 - statistically significant indicating non-significant results, *significant findings, **highly significant findings (p<0.001). TC: Total cholesterol.

Table 5: Correlation of pentraxin with Atherogenic parameters and urine ACR

Parameters	ρ-value	p-value
HDL (mg/dL)	−0.231	0.039*
Non-HDL (mg/dL)	0.05	0.64 (NS)
TC/HDL	0.255	0.022*
TGL/HDL	0.210	0.06 (NS)
LDL/HDL	0.03	0.77 (NS)
Atherogenic coefficient	0.266	0.022*
Urine ACR (µg/mg of creatinine)	0.355**	<0.001**
Spearman's rank correlation: Rho–ρ±1: Perfect correlation, ρ=0.7–1.0 strong correlation, ρ=0.4–0.7 moderate correlation, ρ=0.1–0.4 weak correlation, ρ=0: no correlation		

p-value: *significant results (p<0.05) **highly significant results (p<0.001) (NS–not significant). ACR: Albumin creatinine ratio

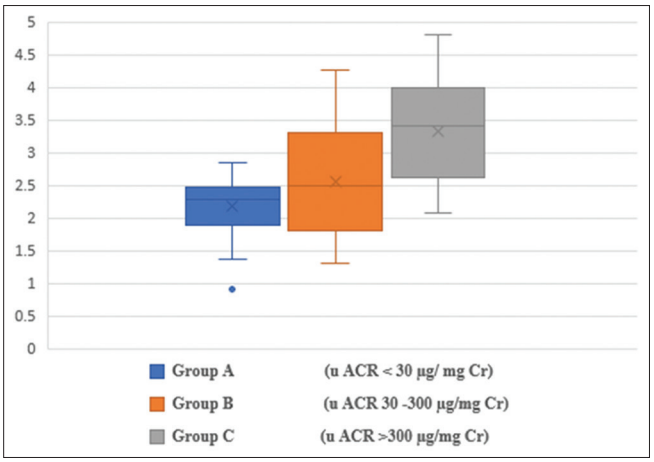

Fig. 2: Pentraxin levels in type 2 diabetes mellitus based on urine albumin creatinine ratio

cut-off ≥2.49 ng/mL, whereas 44.19% of type 2 DM had PTX3 cut-off <2.49 ng/mL.

DISCUSSION

The study expands the interplay between PTX3 and atherogenic markers that underscores the intricate mechanism of vascular endothelial cell inflammation paving way toward CVD. PTX3 is considered a crucial marker of vascular endothelial inflammation. Evaluation of PTX3 and atherogenicity in T2DM are diagnostic to identify individuals with metabolic disturbances with the likelihood of vascular endothelial cell dysfunction. Thereby therapeutic targets to alleviate inflammation and promote vascular tissue repair can be studied as the future scope.

Researchers have identified PTX3 as a protein of the long pentraxin family. It is a cognate molecule of CRP, being a multifunctional regulatory protein PTX3 has a prime role in inflammatory event and the organization of extracellular matrix and remodeling of tissue damage [9]. In fact, one of the findings from researchers is that fibroblast growth factor 2 (FGF2) is involved in the promotion of angiogenesis and revascularization of vascular inflammation. Moreover, that PTX3 was reported as a competitor of FGF2, thereby affecting the activation of smooth muscle cells after arterial injury [10].

PTX3 levels were compared between men and women with DM. The analysis revealed that men exhibited significantly higher levels of PTX-3 compared to women. This finding suggests that men may be more susceptible to CVD due to the heightened vascular inflammatory environment indicated by elevated PTX-3 levels [11].

Growing evidence suggests that excess adiposity is associated with inflammatory background which in turn provokes the release

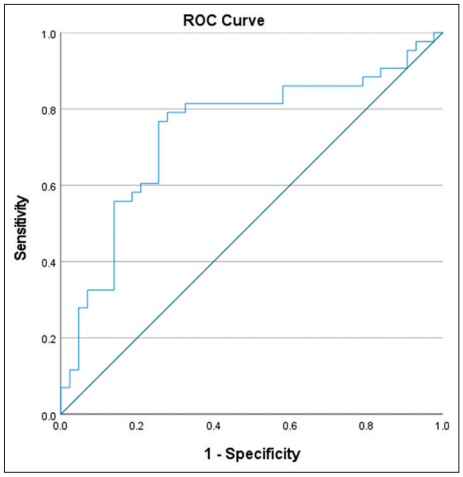

Fig. 3: Receiver's operating characteristics for pentraxin in type 2 diabetes mellitus

Table 6: Receiver's operating characteristics curve diagnostic sensitivity and specificity of pentraxin 3 in type 2 diabetes mellitus

Marker	Cut-off value	Diagnostic sensitivity (%)	Diagnostic specificity (%)	Area under curve	Significance p-value
PTX3 ng/mL	2.49	79.1	72.1	0.739**	<0.001

Area under curve: *0.5 denotes no discrimination; **0.7–0.8 denotes acceptable; ***0.8–0.9 denotes exceptional; >0.9 denotes remarkable

of adipose-derived adipokines and chemokines. Atherosclerotic pathology mirrors systemic inflammation as suggested Pentraxins are considered as a biochemical inflammatory marker that parallels an inflammatory environment [12]. Increased PTX3 has been noticed in response to acute phase inflammation, tumor necrosis factor, IL 1, and lipopolysaccharide, especially from macrophages and vascular endothelial cells. This substantiates with our research findings of increased PTX3 in individuals with overweight and obese BMI. These outcomes could be accounted by the subclinical inflammation attributed to obesity and overweight as well as the inflammatory response-induced release of PTX3 from vascular cells.

In large studies cardiovascular Health study and Multi-Ethic Study of Atherosclerosis, PTX3 was identified as a prognostic tool to cardiovascular mortality and all-cause death [13].

Based on the glycemic status of type 2 Diabetic individuals, the PTX3 levels were compared and found to be elevated in poor glycemic state. This elevated PTX3 levels reflect as marker of inflammation and vascular endothelial damage in diabetic patients. High PTX3 levels are associated with poor glycemic state and also complications such as CVD, nephropathy, and retinopathy [14]. Thus PTX3 is considered as inflammatory and vascular endothelial damage marker. It is considered as a potential target in assessing the CVD risk. The hallmark characteristic of insulin resistance [15] plays a prime role in boosting the underlying vascular pathology promoting inflammatory response thereby PTX3 levels are elevated. This makes PTX3 as potential indicator of the severity of DM.

In this study, PTX3 levels were significantly negatively correlated with HDLc that relates to less protective cardiovascular environment. Moreover, T2DM with elevated PTX3 are associated with cardiovascular risk factor as reflected by elevated atherogenic ratio such as TC/HDLc, LDL/HDLc, and TGL/HDL. Another less comprehensible content about PTX3 is its nature to impair ultrastructural vasculature because of cardioprotective role which is in contrast to our study [16]. PTX3 is found to have a significant positive correlation with TC/HDL and atherogenic coefficient that strengthens the background support in favor of PTX3 as predictor of CVD [17,18].

Growing evidence has linked inflammation to endothelial dysfunction and decreased levels of nitric oxide [19]. Investigators have enlightened at this point innate immunity-based response resulting in elevated PTX3, which is implicated in vascular endothelial dysfunction through matric metalloproteinase pathway. PTX3 is recognized to contribute to endothelial dysfunction by decreased nitric oxide production [20]. PTX3 had demonstrated statistically positive correlation with atherogenic index TC/HDL and atherogenic coefficient that strengthens the background support of PTX3 as a predictor of CVD.

Meta-analysis study had reported that for every increase in 1 unit of PTX3 in the circulation would increase the occurrence of cardiovascular death by 28% and cardiovascular events by 2%. Likewise Multi-ethnic study of atherosclerosis cardiovascular health study had illustrated a strong relation between PTX3 and cardiovascular mortality, thus PTX-3 is considered a biomarker for identifying individuals with CVD progression [21].

This study is unique in a way that it had linked the PTX levels with endothelial dysfunction and albuminuria in type 2 diabetic patients. Moreover, the diagnostic sensitivity of PTX3 was found to be 79.1% and specificity 72.1% at a cut-off value 2.49 ng/mL. Elevated PTX3 levels reflect vascular inflammation and endothelial dysfunction, which are precursors to CVD. Therefore, PTX-3 serves as a valuable tool for the early detection of cardiovascular risk in diabetic patients.

Limitations of the study are that less participants were recruited thereby prospective data analysis has to be done in a large scale to quantify the vascular endothelial markers that predict CVD.

CONCLUSION

PTX3 is considered a promising marker of the inflammatory environment due to poor glycemic and lipidemic environment, thereby relating to the likelihood of complications in diabetes. Analysis of Pentraxin provides a comprehensive picture of patient's metabolic health status and preventive treatment strategies can be planned accordingly. Future studies on a large scale might throw more light on the role of PTX3 with respect to vascular endothelial cell damage.

CONFLICT OF INTEREST

Authors declare none.

FUNDING

Utilized the autoanalyzer Beckman Coulter of the Central Lab services.

AUTHOR CONTRIBUTION

Vyshnavi M S: Sample collection, analytical phase
Arul Senghor KA: Conceptualization, formal analysis, writing – original draft
Vinodhini VM: Supervision, project administration
Renuka P: Writing – review & editing
Kumar JS: Resource visualization

REFERENCES

1. Rajueni K, Ambekar R, Solanki H, Momin AA, Pawar S. Assessment of the possible causes of diabetes mellitus developed in patients post Covid-19 treatment in a tertiary care hospital. Int J Pharm Pharm Sci. 2021 Sep;13(9):11-5. doi: 10.22159/ijpps.2021v13i9.42508

2. Saeedi P, Petersohn I, Salpea P, Malanda B, Karuranga S, Unwin N, *et al*. Global and regional diabetes prevalence estimates for 2019 and projections for 2030 and 2045: Results from the international diabetes federation diabetes atlas, 9th edition. Diabetes Res Clin Pract. 2019 Nov;157:107843. doi: 10.1016/j.diabres.2019.107843, PMID 31518657
3. Theofilis P, Sagris M, Oikonomou E, Antonopoulos AS, Siasos G, Tsioufis C, *et al*. Inflammatory mechanisms contributing to endothelial dysfunction. Biomedicines. 2021 Jul 6;9(7):781. doi: 10.3390/biomedicines9070781, PMID 34356845, PMCID PMC8301477
4. Carrizzo A, Procaccini C, Lenzi P, Fusco C, Villa F, Migliarino S, *et al*. PTX3: An inflammatory protein modulating ultrastructure and bioenergetics of human endothelial cells. Immun Ageing. 2019 Feb 2;16:4. doi: 10.1186/s12979-019-0144-0, PMID 30733816, PMCID PMC6359806
5. Lin X, Song W, Zhou Y, Gao Y, Wang Y, Wang Y, *et al*. Elevated urine albumin creatinine ratio increases cardiovascular mortality in coronary artery disease patients with or without type 2 diabetes mellitus: A multicenter retrospective study. Cardiovasc Diabetol. 2023 Aug 10;22(1):203. doi: 10.1186/s12933-023-01907-3, PMID 37563647, PMCID PMC10416404
6. Fornai F, Carrizzo A, Forte M, Ambrosio M, Damato A, Ferrucci M, *et al*. The inflammatory protein PENTRAXIN 3 in cardiovascular disease. Immun Ageing. 2016 Aug 24;13(1):25. doi: 10.1186/s12979-016-0080-1, PMID 27559355, PMCID PMC4995820
7. Trojak A, Waluś-Miarka M, Kapusta M, Miarka P, Kawalec E, Idzior-Waluś B, *et al*. Serum PENTRAXIN 3 concentration in patients with type 2 diabetes and nonalcoholic fatty liver disease. Pol Arch Intern Med. 2019 Aug 29;129(7-8):499-505. doi: 10.20452/pamw.14913, PMID 31469122
8. Bo MS, Cheah WL, Lwin S, Moe Nwe T, Win TT, Aung M. Understanding the relationship between atherogenic index of plasma and cardiovascular disease risk factors among staff of an university in Malaysia. J Nutr Metab. 2018 Jul 4;2018:7027624. doi: 10.1155/2018/7027624, PMID 30116641, PMCID PMC6079547
9. Garlanda C, Bottazzi B, Magrini E, Inforzato A, Mantovani A. PTX3, a humoral pattern recognition molecule, in innate immunity, tissue repair, and cancer. Physiol Rev. 2018 Apr 1;98(2):623-39. doi: 10.1152/physrev.00016.2017, PMID 29412047, PMCID PMC5985957
10. Presta M, Foglio E, Churruca Schuind A, Ronca R. Long pentraxin-3 modulates the angiogenic activity of fibroblast growth Factor-2. Front Immunol. 2018 Oct 8;9:2327. doi: 10.3389/fimmu.2018.02327, PMID 30349543, PMCID PMC6187966
11. Waluś-Miarka M, Trojak A, Miarka P, Kapusta M, Kawalec E, Idzior-Waluś B, *et al*. Correlates of PENTRAXIN 3 serum concentration in men and women with type 2 diabetes. Innate Immun. 2020 Jul;26(5):351-7. doi: 10.1177/1753425919891628, PMID 31874582, PMCID PMC7903534
12. Dervisoglu P, Elmas B. PENTRAXIN 3 as a marker for cardiovascular disease risk in overweight and obese children. Acta Cardiol Sin. 2021 Mar;37(2):177-83. doi: 10.6515/ACS.202103-37(2).20201006A, PMID 33716459, PMCID PMC7953122
13. Ristagno G, Fumagalli F, Bottazzi B, Mantovani A, Olivari D, Novelli D, *et al*. PENTRAXIN 3 in cardiovascular disease. Front Immunol. 2019 Apr 17;10:823. doi: 10.3389/fimmu.2019.00823, PMID 31057548, PMCID PMC6481278
14. Karamfilova V, Assyov Y, Nedeva I, Gateva A, Ivanova I, Cherkezov N, *et al*. Increased serum PENTRAXIN 3 is associated with prediabetes and type 2 diabetes in obese patients with nonalcoholic fatty liver disease. Metab Syndr Relat Disord. 2022 Mar;20(2):132-6. doi: 10.1089/met.2021.0086, PMID 34818080
15. Bhagyashree N, Ramaswamy C, Ramya K, Ganesh M. Comparison of insulin resistance with the severity of metabolic syndrome: Insulin resistance. Asian J Pharm Clin Res. 2020 Sept;13(9):73-5. doi: 10.22159/ajpcr.2020.v13i9.38444
16. Witasp A, Carrero JJ, Michaëlsson K, Ahlström H, Kullberg J, Adamsson V, *et al*. Inflammatory biomarker pentraxin 3 (PTX3) in relation to obesity, body fat depots and weight loss. Obesity (Silver Spring). 2014 May;22(5):1373-9. doi: 10.1002/oby.20695, PMID 24415446
17. Lee R, Ahn HR, Shin MH, Kim HN, Lee YH, Choi SW, *et al*. Association of plasma pentraxin-3 level with lipid levels and cardiovascular risk factors in people with no history of lipid-lowering medication: The dong-gu study. J Atheroscler Thromb. 2019 Aug 1;26(8):738-45. doi: 10.5551/jat.47167, PMID 30674758, PMCID PMC6711843
18. Ki J, Sivagami K, Kumar M, Aparnavi P, Jeevithan S. Connecting the dots: Investigating the relationship between HbA1c and lipid profile. Asian J Pharm Clin Res. Dec 2024;17(12):151-4. doi: 10.22159/ajpcr.2024v17i12.50249
19. Anderson MR, Kim JS, Podolanczuk A, Ding J, Al-Naamani N, Allison M, *et al*. Nonlinear associations between computed tomography-measures of adiposity and long pentraxin-3 in the multi-ethnic study of atherosclerosis. Obes Sci Pract. 2024;10(1):e708. doi: 10.1002/osp4.708, PMID 38263991, PMCID PMC10804353
20. Carrizzo A, Lenzi P, Procaccini C, Damato A, Biagioni F, Ambrosio M, *et al*. PENTRAXIN 3 induces vascular endothelial dysfunction through a P-selectin/matrix metalloproteinase-1 pathway. Circulation. 2015 Apr 28;131(17):1495-505; discussion 1505. doi: 10.1161/circulationaha.114.014822, PMID 25747934
21. Li L, Liu H, Zhang Q, Jin H, Tao H, Chen H, *et al*. Pentraxin-3 and outcomes in CKD: A systematic review and meta-analysis. Kidney Med. 2024 Feb 22;6(4):100800. doi: 10.1016/j.xkme.2024.100800, PMID 38645733, PMCID PMC11026967