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Association of Serum Uric Acid Level with Diabetic Retinopathy in Patients with Type 2 Diabetes Mellitus

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KEYWORDS

Serum uric acid, Diabetic retinopathy, Type 2 diabetes mellitus, Biomarker, Glycaemic correlation, Microvascular complications

ABSTRACT:

Background: Diabetic retinopathy is a major microvascular complication of type 2 diabetes mellitus (T2DM), leading to vision impairment and blindness. Emerging evidence suggests that elevated serum uric acid levels may contribute to its development and progression.

Objectives: To assess serum uric acid levels in patients with T2DM, evaluate its association with diabetic retinopathy, and determine its relationship with the severity of diabetic retinopathy.

Methods: This was an observational, cross-sectional study conducted in the Department of General Medicine, among 140 T2DM between July 2023 – December 2024 a comprehensive clinical assessment, including vitals, systemic examination, anthropometry and laboratory tests (FBS, PPBS, HbA1c and serum uric acid) was conducted followed by an ophthalmological evaluation with fundus examination to assess diabetic retinopathy. Analysis was done using Stata v16.

Results: The study found a significant association between serum uric acid levels and diabetic retinopathy in T2DM patients. Serum uric acid levels were markedly higher in patients with DR (8.4 ± 1.3 mg/dL) compared to those without DR (5.4 ± 1.9 mg/dL) ($P < 0.001$), and levels increased progressively with disease severity. Patients with PDR had higher serum uric acid levels (9.2 ± 1.8 mg/dL) compared to those with NPDR (8.1 ± 0.9 mg/dL) ($P < 0.001$). Within NPDR, serum uric acid levels increased from mild (7.6 ± 0.7 mg/dL) to moderate (8.4 ± 0.7 mg/dL) to severe NPDR (10.4 ± 0.3 mg/dL) ($P < 0.001$). Serum uric acid levels also showed a significant positive correlation with glycaemic parameters, including fasting blood sugar (rp = 0.394, $P < 0.001$), postprandial blood sugar (rp = 0.419, $P < 0.001$), and glycated haemoglobin (rp = 0.411, $P < 0.001$). ROC analysis demonstrated that serum uric acid is a strong predictor of DR (AUC = 0.908), PDR (AUC = 0.702), and severe NPDR (AUC = 0.935), with high sensitivity and specificity.

Conclusion: This study found a significant association between elevated serum uric acid levels and the presence and severity of diabetic retinopathy in T2DM patients. Serum uric acid may serve as a potential biomarker for early identification and risk stratification of diabetic retinopathy.

INTRODUCTION

Diabetes mellitus (DM) is a group of metabolic disorders characterized by chronic hyperglycaemia due to defects in insulin secretion, insulin action, or both. The prevalence of diabetes mellitus has increased dramatically over the past two decades and is expected to continue rising, with projections estimating that

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approximately 642 million people will be diagnosed with diabetes by the year 2040.(1) Type 2 diabetes mellitus (T2DM) constitutes the majority of diabetes cases worldwide, driven by lifestyle factors such as increasing obesity rates and decreased physical activity. The metabolic dysregulation associated with diabetes leads to multiple complications,(2) significantly impacting both individual health and healthcare systems globally. One of the major concerns associated with diabetes is its complications, which are broadly classified into macrovascular and microvascular complications. Macrovascular complications include cardiovascular diseases, such as coronary artery disease and stroke, which significantly contribute to the increased mortality and morbidity in diabetic patients. However, the real burden of diabetes lies in its microvascular complications, including diabetic retinopathy, nephropathy, and neuropathy.(3) Among these, diabetic retinopathy is a leading cause of vision impairment and blindness in working-age adults.(4)

Diabetic retinopathy is a progressive microvascular complication of diabetes that affects the retinal vasculature, leading to vision impairment and potential blindness if left untreated. In the United States, diabetes is one of the leading causes of blindness in individuals aged 20–74 years.(5) In India, the prevalence of diabetic retinopathy ranges from 12% to 37%.(6) The risk of developing moderate vision loss over three years in diabetic individuals is approximately 25%.(7) The duration of diabetes significantly influences the likelihood of developing retinopathy, with a 25% incidence observed within five years and an 80% incidence within 15 years in individuals with type 1 diabetes mellitus.(8)

The pathophysiological changes seen in diabetic retinopathy are primarily due to chronic hyperglycaemia, which results in retinal ischemia and increased vascular permeability. The loss of pericytes, retinal hypoxia, and upregulation of vascular endothelial growth factor (VEGF) contribute to the development of neovascularization, leading to further complications such as vitreous haemorrhage, fibrosis, and retinal detachment.(9, 10) Diabetic retinopathy is categorized into two main types: non-proliferative diabetic retinopathy (NPDR) and proliferative diabetic retinopathy (PDR). NPDR is an early stage characterized by microaneurysms, haemorrhages, cotton wool spots, and venous abnormalities, whereas PDR is marked by neovascularization and fibrous tissue proliferation, significantly increasing the risk of vision loss.(5)

Hyperuricemia, defined as elevated serum uric acid (SUA) levels above 7 mg/dL, has been recognized as an important risk factor for various diseases, including cardiovascular disease, renal dysfunction, and peripheral vascular disease.(11-14) Emerging research has also suggested a potential role of hyperuricemia in the development and progression of diabetic retinopathy.(11) Uric acid, a byproduct of purine metabolism, acts as both an antioxidant and a pro-oxidant, contributing to oxidative stress, endothelial dysfunction, and inflammation, all of which are implicated in the pathogenesis of diabetic complications. The mechanisms linking hyperuricemia to diabetic retinopathy involve oxidative stress, endothelial dysfunction, and activation of inflammatory pathways, which exacerbate retinal ischemia and vascular permeability.(9) Given the increasing prevalence of diabetes and its associated complications, there is a growing need to identify potential risk factors that contribute to the progression of diabetic retinopathy. Against this background, the present study aimed to assess serum uric acid levels in T2DM patients and their association with diabetic retinopathy, while secondarily evaluating the severity of diabetic retinopathy in relation to serum uric acid levels.

MATERIALS AND METHODS

This was a single centre, hospital based, observational, cross-sectional study conducted in the outpatient department and/or inpatient wards of the Department of General Medicine, Aarupadai Veedu Medical College and Hospital, Puducherry, India between July 2023 and December 2024. The study was approved by the Institutional Human Ethics Committee (IHEC) with reference number AV/IHEC/2023/051 dated 25/05/2023. The participants were given the Participant Information Sheet (PIS) in their native language, and its contents were verbally explained to ensure their understanding and satisfaction. Enrolment into the study proceeded upon receipt of written informed consent.

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Patients more than 18 years of age, of both gender, diagnosed with T2DM were included. However, pregnant patients; patients on xanthine oxidase inhibitors, thiazides, oral contraceptive pills; with lymphoma; history of organ transplantation; with nephrotic syndrome; chronic obstructive pulmonary disease; chronic kidney disease; systemic hypertension; and liver disease were excluded.

The study estimated a minimum sample size of 140 T2DM patients, based on a 15.5% prevalence of diabetic retinopathy (from Raman et al.), with a 6% absolute precision and a 6% significance level, accounting for a 10% attrition rate. We used nonprobability sampling technique – purposive sampling/consecutive enumeration to enrol patients. A detailed history was recorded for each patient, including demographic details, duration of diabetes, medical history, and any associated comorbidities. A thorough clinical assessment was conducted, including vital parameters, systemic examination, and relevant anthropometric measurements. Laboratory investigations, including fasting blood sugar (FBS), postprandial blood sugar (PPBS), glycated haemoglobin (HbA1c), and serum uric acid levels, were performed for all participants. Patients were referred to the ophthalmology department to evaluate the presence/type of diabetic retinopathy. A comprehensive ophthalmological examination was conducted after pupil dilation, and the fundus was examined using an indirect ophthalmoscope to determine the presence and severity of diabetic retinopathy. Findings were recorded systematically for further analysis.

Statistical analysis: The data collected was manually entered in Microsoft Excel, coded, recoded, and analysed using Software for Statistics and Data Science (Stata) v16 (StataCorp, 2019). Descriptive analysis was presented using numbers and percentages for categorical variables; mean and standard deviation (SD) for continuous variables (based on data normality tested using Kolmogorov–Smirnov test and the Shapiro–Wilk test). Chi square test of significance (two-sided) for categorial and independent 't' tests (two-sided) for continuous variables was applied to test for association between independent and dependent study variables. Pearsons's correlation coefficient was estimated to assess the correlation between serum uric acid levels and glycaemic parameters. Receiver operating characteristics (ROC) analysis was conducted to determine the area under the curve of serum uric acid levels in predicting DR, type of DR (PDR), and severity of NPDR (severe NPDR). Statistical significance was considered at $p<0.05$.

RESULTS

The study included 140 participants with a mean age of 55.7 ± 8.7 years, of whom 80% were younger than 60 years. The sample comprised 54.3% males and 45.7% females. None of the patients reported smoking or alcohol use. Most participants (80.7%) had a known diagnosis of T2DM, while 19.3% were newly diagnosed. The mean FBS was 236.8 ± 72.7 mg/dL, PPBS was 311.2 ± 87.9 mg/dL, and glycated haemoglobin was 8.6 ± 1.5%. The mean serum uric acid level was 7.5 ± 2.0 mg/dL. Diabetic retinopathy was observed in 70.7% of participants, with 28.3% having PDR and 71.7% having NPDR. Among NPDR cases, 47.9% had mild NPDR, 49.3% had moderate NPDR, and 2.8% had severe NPDR.

Serum uric acid levels were significantly higher in patients with diabetic retinopathy (DR) compared to those without DR (8.4 ± 1.3 mg/dL vs. 5.4 ± 1.9 mg/dL, $p < 0.001$). Among DR cases, patients with proliferative diabetic retinopathy (PDR) had higher serum uric acid levels (9.2 ± 1.8 mg/dL) than those with non-proliferative diabetic retinopathy (NPDR) (8.1 ± 0.9 mg/dL, $p < 0.001$). Within NPDR cases, serum uric acid levels increased with disease severity, measuring 7.6 ± 0.7 mg/dL in mild NPDR, 8.4 ± 0.7 mg/dL in moderate NPDR, and peaking at 10.4 ± 0.3 mg/dL in severe NPDR ($p < 0.001$), indicating a strong association between higher serum uric acid levels and the severity of diabetic retinopathy.

The study found a significant positive correlation between serum uric acid levels and various glycaemic parameters. FBS showed a moderate positive correlation with serum uric acid levels ($r_p = 0.394$, $P < 0.001$). Similarly, PPBS demonstrated a slightly stronger positive correlation ($r_p = 0.419$, $P < 0.001$). Glycated haemoglobin also exhibited a significant positive correlation with serum uric acid levels (rp =

0.411, P < 0.001), indicating that higher blood glucose levels were associated with increased serum uric acid levels.

The ROC analysis demonstrated that serum uric acid levels were a strong predictor of diabetic retinopathy, PDR, and severe NPDR. For diabetic retinopathy, the AUC was 0.908 (95% CI: 0.829–0.988), with a cutoff value of >6.0 mg/dL, showing a sensitivity of 97.0% and specificity of 85.4% (P < 0.001). In predicting PDR, the AUC was 0.702 (95% CI: 0.561–0.842), with a cutoff of >8.0 mg/dL, sensitivity of 67.9%, and specificity of 40.8% (P = 0.002). For severe NPDR, the AUC was 0.935 (95% CI: 0.832–1.000), with a cutoff of >9.0 mg/dL, sensitivity of 79.2%, and specificity of 91.3% (P = 0.037).

DISCUSSION

The present study aimed to determine the association between serum uric acid levels and diabetic retinopathy in patients with T2DM. The study included 140 patients diagnosed with T2DM, with a mean age of 55.7 years (SD = 8.7). A significant proportion (80%) of the participants were below 60 years of age, which aligns with previous studies indicating that T2DM predominantly affects middle-aged individuals, particularly in India.(15) The gender distribution was nearly balanced, with 54.3% males and 45.7% females. This finding is consistent with global epidemiological trends where T2DM prevalence is similar in both genders but with slight variations based on region and socioeconomic factors.(16) None of the study participants reported smoking or alcohol use. Given that smoking and alcohol consumption are known risk factors for the progression of DR,(17, 18) their absence in the present study suggests that other metabolic factors, such as hyperglycaemia and serum uric acid levels, might have played a more significant role in the observed DR prevalence. A majority of participants (80.7%) were known cases of T2DM, whereas 19.3% were newly diagnosed. This observation suggests that a considerable proportion of individuals remain undiagnosed until they develop complications, highlighting the need for early screening and intervention.(19)

The mean FBS level among the participants was 236.8 mg/dL (SD = 72.7), while the mean PPBS level was 311.2 mg/dL (SD = 87.9). These values indicate poor glycaemic control in the studied population, as optimal FBS and PPBS levels should be below 126 mg/dL and 200 mg/dL, respectively, according to the American Diabetes Association.(20) Furthermore, the mean glycated haemoglobin level was 8.6% (SD = 1.5%), which exceeds the recommended target of <6.5% for good glycaemic control.(20) Poor glycaemic control is a well-established risk factor for the development and progression of DR, as hyperglycaemia induces retinal damage through oxidative stress, inflammation, and microvascular dysfunction.(21)

The mean serum uric acid level in the study population was 7.5 mg/dL (SD = 2.0), which is above the normal reference range (typically 3.5–7.0 mg/dL in males and 2.5–6.0 mg/dL in females). Elevated serum uric acid levels have been implicated in the pathogenesis of DR through mechanisms involving endothelial dysfunction, oxidative stress, and chronic inflammation.(22) Several studies have reported a positive correlation between hyperuricemia and DR, suggesting that serum uric acid may serve as an independent risk factor for the development of microvascular complications in T2DM.(23)

In the present study, DR was present in 70.7% (n = 99) of the participants. This high prevalence aligns with previous Indian studies, where DR has been reported in 50%–80% of T2DM patients with poor glycaemic control.(24, 25) Among those with DR, 71.7% had NPDR, while 28.3% had PDR. NPDR is an earlier stage of DR characterized by microaneurysms, haemorrhages, and retinal oedema, whereas PDR represents a more severe stage marked by neovascularization and an increased risk of vision loss.(5) Further stratification of NPDR cases showed that 47.9% had mild NPDR, 49.3% had moderate NPDR, and 2.8% had severe NPDR. This distribution suggests that most patients with DR were in the early to moderate stages, which is crucial from a clinical perspective, as timely intervention at this stage can prevent progression to PDR and associated visual impairment.(26)

The study found that mean serum uric acid levels were significantly higher in patients with DR (8.4 ± 1.3

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mg/dL) compared to those without DR (5.4 ± 1.9 mg/dL). This result aligns with previous research indicating that hyperuricemia is associated with an increased risk of DR in diabetic patients.(27) Elevated serum uric acid levels may contribute to DR pathogenesis through mechanisms such as oxidative stress, endothelial dysfunction, and inflammation, which have been shown to accelerate retinal microvascular damage. The relationship between serum uric acid levels and DR severity was further supported by the observation that patients with PDR had significantly higher serum uric acid levels (9.2 ± 1.8 mg/dL) compared to those with NPDR (8.1 ± 0.9 mg/dL) (P < 0.001). These findings are consistent with studies demonstrating that serum uric acid levels increase with DR progression, suggesting that uric acid plays a role in the worsening of retinal damage.(28)

A particularly striking finding of this study was the progressive increase in serum uric acid levels with NPDR severity. The mean serum uric acid level was lowest in patients with mild NPDR (7.6 ± 0.7 mg/dL), increased in those with moderate NPDR (8.4 ± 0.7 mg/dL), and was highest in those with severe NPDR (10.4 ± 0.3 mg/dL), with a statistically significant P-value of <0.001. This dose-response relationship suggests a potential causal role of serum uric acid in DR pathogenesis.(29) Studies have indicated that hyperuricemia exacerbates microvascular damage by increasing endothelial dysfunction, activating pro-inflammatory cytokines, and promoting oxidative stress – all of which are key contributors to DR progression.(14) Furthermore, high serum uric acid levels may contribute to increased vascular permeability and retinal ischemia, which are characteristic features of severe NPDR and PDR.(30)

The study also demonstrated significant positive correlations between serum uric acid levels and glycaemic parameters, including FBS, PPBS, and glycated haemoglobin. The correlation coefficients were rp = 0.394 (P < 0.001) for FBS, rp = 0.419 (P < 0.001) for PPBS, and rp = 0.411 (P < 0.001) for HbA1c, indicating that higher blood glucose levels were associated with increased serum uric acid levels. These findings suggest that poor glycaemic control is associated with higher serum uric acid levels, a relationship that has been documented in several studies.(31) Hyperglycaemia promotes uric acid synthesis through increased purine metabolism and reduced renal clearance, leading to hyperuricemia.(32) Given that hyperglycaemia is a well-established risk factor for DR, the observed correlation between serum uric acid and glycaemic markers further supports the role of serum uric acid in DR pathogenesis.

The ROC analysis demonstrated that serum uric acid levels are a strong predictor of DR, PDR, and severe NPDR. The results indicated that for DR, the AUC was 0.908 (95% CI: 0.829–0.988), with a cutoff value of >6.0 mg/dL, a sensitivity of 97.0%, and a specificity of 85.4% (P < 0.001), suggesting that SUA levels above 6.0 mg/dL can reliably distinguish diabetic patients with and without DR, making it a highly sensitive and specific biomarker. Previous studies have reported similar findings, highlighting SUA as a potential screening tool for early DR detection.(33) In predicting PDR, the AUC was 0.702 (95% CI: 0.561–0.842), with a cutoff of >8.0 mg/dL, sensitivity of 67.9%, and specificity of 40.8% (P = 0.002). Although SUA was a significant predictor of PDR, the sensitivity and specificity were lower compared to DR, suggesting that while SUA is useful for identifying DR in general, additional factors such as duration of diabetes, lipid profiles, and inflammatory markers may need to be considered when predicting PDR risk.(5) For severe NPDR, the AUC was 0.935 (95% CI: 0.832–1.000), with a cutoff of >9.0 mg/dL, sensitivity of 79.2%, and specificity of 91.3% (P = 0.037). These findings suggest that SUA levels above 9.0 mg/dL are highly predictive of severe NPDR, reinforcing its potential role as a biomarker for disease severity. Given that severe NPDR often progresses to PDR if untreated, SUA monitoring could help in risk stratification and timely intervention.(26)

The results of this study emphasize the need for routine SUA screening in diabetic patients, particularly those at risk for DR. Given the high sensitivity and specificity of SUA in predicting DR, early intervention strategies, including uric acid-lowering therapies such as allopurinol and febuxostat, could be explored as potential approaches for DR prevention and management.(11) Moreover, the strong correlation between SUA and glycaemic parameters highlights the importance of comprehensive diabetes management, including strict blood sugar control, dietary modifications, and pharmacological interventions to mitigate both hyperuricemia and DR risk.

The study has certain limitations that should be acknowledged. As a single-centre, hospital-based study conducted in a tertiary healthcare facility, the findings may not be generalizable to broader populations, particularly those in community settings or rural areas with different healthcare access and disease patterns. The cross-sectional design limits the ability to establish a causal relationship between serum uric acid levels and diabetic retinopathy, as it only provides a snapshot of the association rather than tracking disease progression over time. The sample size, though statistically adequate, may still limit the robustness of subgroup analyses, particularly in assessing different stages of diabetic retinopathy. Additionally, the study did not account for potential confounding factors such as inflammatory markers, lipid profiles, and renal function parameters, which could influence both serum uric acid levels and diabetic retinopathy risk. The exclusion of patients with systemic hypertension, chronic kidney disease, and other comorbidities, while necessary to maintain a homogeneous study population, may have limited the ability to explore interactions between these conditions and diabetic retinopathy. Furthermore, lifestyle factors such as dietary habits, physical activity levels, and genetic predisposition, which could impact serum uric acid levels and diabetes-related complications, were not assessed.

CONCLUSION

This study demonstrated a significant association between elevated serum uric acid levels and the presence and severity of diabetic retinopathy in patients with type 2 diabetes mellitus. Higher serum uric acid levels were observed in patients with diabetic retinopathy compared to those without, and levels progressively increased with the severity of the condition, particularly in those with proliferative diabetic retinopathy and severe non-proliferative diabetic retinopathy. The study also found a strong correlation between serum uric acid levels and glycaemic parameters, suggesting a potential link between poor glycaemic control and hyperuricemia. Furthermore, receiver operating characteristic analysis indicated that serum uric acid could serve as a reliable biomarker for predicting diabetic retinopathy and its severity, with high sensitivity and specificity. These findings highlight the potential role of serum uric acid monitoring in the early identification of high-risk patients, allowing for timely interventions to prevent disease progression.

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Table 1: Sociodemographic and clinical characteristics

		Frequency (N = 140)	**Percentage**
		(n)	**(%)**
Age (in years), Mean (SD)		55.7 (8.7)	
Age (in years)	≤60	112	80.0
	>60	28	20.0
Gender	Female	64	45.7
	Male	76	54.3
Diabetes mellitus	Newly diagnosed	27	19.3
	Known case	113	80.7
Fasting Blood Sugar (mg/dL), Mean (SD)		236.8 (72.7)	
Postprandial Blood Sugar (mg/dL), Mean (SD)		311.2 (87.9)	
Glycated Haemoglobin (%), Mean (SD)		8.6 (1.5)	
Serum Uric Acid (mg/dL), Mean (SD)		7.5 (2.0)	
Diabetic retinopathy (N = 140)	Present	99	70.7
	Absent	41	29.3
Type of diabetic retinopathy (N = 99)	PDR	28	28.3
	NPDR	71	71.7
Severity of NPDR (N = 71)	Mild	34	47.9
	Moderate	35	49.3
	Severe	2	2.8
SD, Standard deviation; PDR, Proliferative Diabetic Retinopathy; NPDR, Non-Proliferative Diabetic Retinopathy			

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Table 2: Association between serum uric acid levels, presence of diabetic retinopathy, its types and severity of NPDR

		Serum Uric Acid (mg/dL)	P value
		Mean (SD)	
Diabetic retinopathy (N = 140)	Present	8.4 (1.3)	<0.001*
	Absent	5.4 (1.9)	Ref
Type of diabetic retinopathy (N = 99)	PDR	9.2 (1.8)	<0.001*
	NPDR	8.1 (0.9)	Ref
Severity of NPDR (N = 71)	Mild	7.6 (0.7)	<0.001*
	Moderate	8.4 (0.7)	<0.001*
	Severe	10.4 (0.3)	Ref
PDR, Proliferative Diabetic Retinopathy; NPDR, Non-Proliferative Diabetic Retinopathy			

Table 3: ROC analysis showing area under the curve of serum uric acid levels in predicting diabetic retinopathy, PDR and severe NPDR

	AUC (95% CI)	Cut off	Sensitivity (%)	Specificity (%)	P value
Diabetic retinopathy	0.908 (0.829 to 0.988)	≥6.0	97.0	85.4	<0.001*
PDR	0.702 (0.561 to 0.842)	≥8.0	67.9	40.8	0.002*
Severe NPDR	0.935 (0.832 to 1.000)	≥9.0	79.2	91.3	0.037*
PDR, Proliferative Diabetic Retinopathy; NPDR, Non-Proliferative Diabetic Retinopathy; AUC, Area under the curve; CI, Confidence interval					

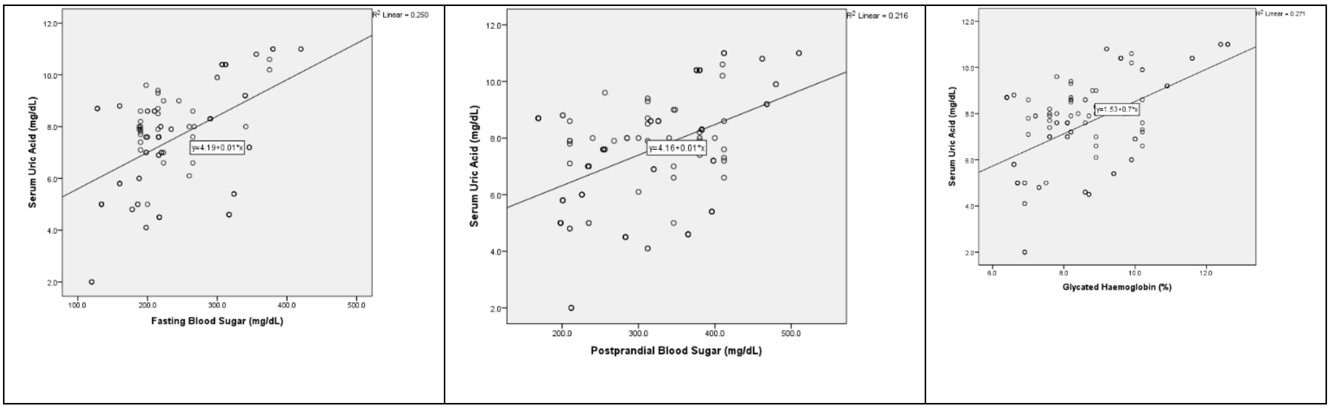

Figure 1: Correlation between serum uric acid levels, fasting blood sugar levels, postprandial blood sugar levels, and glycated haemoglobin

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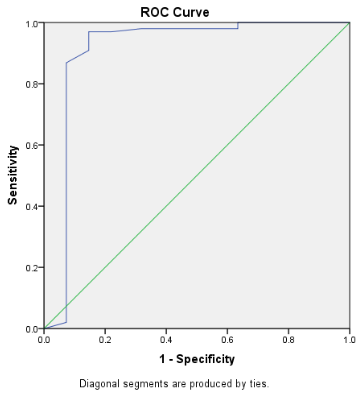
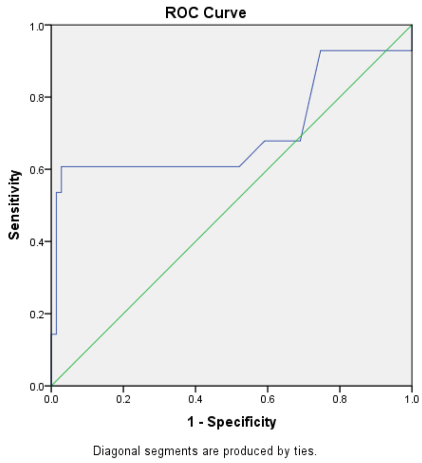
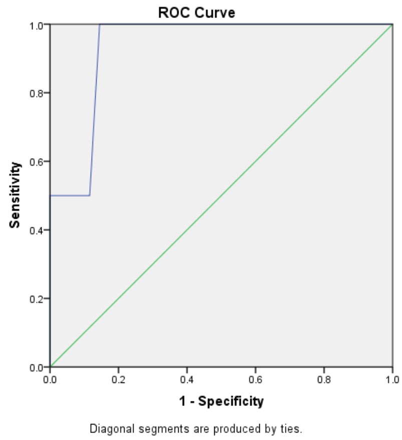
Serum uric acid levels in predicting diabetic retinopathy	**Serum uric acid levels in predicting PDR**	**Serum uric acid levels in predicting severe NPDR**
 	 	

Figure 2: ROC analysis showing area under the curve of serum uric acid levels in predicting diabetic retinopathy, PDR and severe NPDR