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Maternal Serum Netrin-1 Levels in Pregnancies Complicated by Gestational Diabetes Mellitus

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Abstract:

Objective: Gestational diabetes mellitus (GDM) is associated with metabolic and vascular disturbances, yet its underlying biological mechanisms remain incompletely understood. Netrin-1, a multifunctional protein involved in angiogenesis, inflammation, and endothelial homeostasis, has been implicated in metabolic disorders; however, clinical data in GDM are limited. This study aimed to evaluate maternal netrin-1 levels in pregnancies complicated by GDM and to assess their associations with clinical parameters.

Methods: This prospective case–control study included 45 pregnant women with GDM and 80 healthy pregnant controls between 24 and 28 weeks of gestation. Serum netrin-1 concentrations were measured using enzyme-linked immunosorbent assay. Clinical characteristics, glycemic parameters, and pregnancy outcomes were recorded. Receiver operating characteristic (ROC) analysis assessed the discriminatory performance of netrin-1 for GDM, and correlation and multivariable regression analyses identified associations after adjustment for selected covariates.

Results: Serum netrin-1 levels were significantly lower in GDM group compared with controls ($P<0.001$). Netrin-1 concentrations showed significant inverse correlations with fasting plasma glucose ($r=-0.446$, $P=0.002$) and HbA1c ($r=-0.623$, $P<0.001$). ROC analysis identified an optimal cut-off value of 0.09 ng/mL for discriminating GDM, yielding a sensitivity of 68.9% and a high specificity of 98.8% (AUC=0.861), indicating good discriminative performance. In multivariable analysis adjusted for age, body mass index, and parity, log-transformed netrin-1 levels were associated with GDM ($P<0.001$).

Conclusion: Maternal netrin-1 levels are significantly reduced in pregnancies complicated by GDM and are closely associated with glycemic dysregulation. Netrin-1 may reflect underlying metabolic and vascular alterations and could be considered as a complementary biomarker rather than a standalone diagnostic tool in GDM.

Keywords: Gestational Diabetes Mellitus, Netrin-1, Pregnancy, Biomarkers, Glucose Metabolism

Gestational diabetes mellitus (GDM) represents a frequent metabolic disorder of pregnancy, characterized by impaired glucose regulation first detected during gestation [1]. The prevalence of GDM continues to increase worldwide, largely driven by rising obesity and sedentary lifestyles [2]. GDM is associated with significant maternal and neonatal morbidity, including preeclampsia, higher cesarean

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delivery rates, macrosomia, neonatal hypoglycemia, and an increased risk of future metabolic disease in both mother and offspring [3]. Despite improvements in screening and treatment strategies, the biological mechanisms underlying GDM are not yet fully elucidated.

Normal pregnancy involves gradual metabolic adaptation, including increasing insulin resistance and altered glucose handling to ensure adequate fetal nutrient supply [2, 4]. GDM develops when these metabolic adaptations exceed the compensatory capacity of pancreatic β-cells, resulting in hyperglycemia [2]. Beyond disturbances in glucose metabolism, GDM has increasingly been recognized as a condition associated with endothelial dysfunction, altered placental angiogenesis, and chronic low-grade inflammation [5]. These features overlap with metabolic abnormalities observed in prediabetes, suggesting shared pathophysiological pathways.

Netrin-1 is a laminin-like extracellular protein initially described as an axonal guidance molecule but later identified as a regulator of angiogenesis, inflammation, and endothelial homeostasis [6]. Netrin-1 is expressed in several tissues relevant to metabolic disease, including vascular endothelium, pancreatic tissue, kidneys, and placenta [7]. Experimental studies suggest that netrin-1 may have protective effects under metabolic stress by reducing inflammation, preserving endothelial function, and limiting oxidative injury [7, 8].

However, clinical evidence regarding circulating netrin-1 levels in metabolic disorders has yielded inconsistent results. Reduced serum netrin-1 concentrations have been reported in newly diagnosed type 2 diabetes mellitus (T2DM) and linked to insulin resistance, whereas other studies have observed elevated levels in individuals with impaired glucose tolerance, possibly reflecting a compensatory response [9-11]. In pregnancy, netrin-1 has been investigated both at the placental and circulating levels in hypertensive disorders and placental insufficiency; however, in GDM, existing evidence is limited to placental studies, with a lack of data on maternal circulating netrin-1 levels [12, 13].

Therefore, the present study aimed to compare maternal serum netrin-1 levels between women with GDM and healthy pregnant controls and to evaluate whether netrin-1 is associated with glycemic parameters and adverse pregnancy outcomes.

METHODS

Study Design and Sample Size

This prospective case–control study was conducted at a tertiary obstetrics and gynecology center between September 2023 and May 2024. Ethical approval was obtained from the institutional Clinical Research Ethics Committee (Approval No: 2011-KAEK-25, 2023/08-03). Written informed consent was obtained from all participants. All procedures were conducted in accordance with the ethical principles of the Declaration of Helsinki.

Sample size estimation was performed using GPower software (version 3.1.9.7). Assuming a medium effect size (Cohen's d = 0.6), an alpha level of 0.05, and 80% power, a minimum of 40 participants per group was required [13]. A larger sample was planned to account for potential exclusions.

Study Participants

Eligible participants were pregnant women aged 18–35 years with singleton pregnancies and a gestational age between 24 and 28 weeks. Inclusion criteria included a body mass index (BMI) ≤35 kg/m² and classification into either GDM or control group. The control group consisted of women with a negative 50 g glucose challenge test. GDM was diagnosed using the two-step screening approach. All participants underwent a 50 g GCT, followed by a diagnostic 100 g OGTT in those with positive screening results. GDM was defined as at least two elevated plasma glucose values during the 100 g OGTT (1h ≥180 mg/dL, 2h ≥155 mg/dL, 3h ≥140 mg/dL) [14].

Women with pregestational diabetes, overt diabetes, multiple pregnancy, hypertensive disorders, chronic systemic disease, active infection, smoking, fetal anomalies, placental abnormalities, or incomplete follow-up data were excluded.

After the exclusion process, 45 women with GDM and 80 healthy pregnant women were included in the final analysis.

Sociodemographic characteristics, obstetric history, gestational age, serum netrin-1 levels, and HbA1c values were recorded. Pregnancy outcomes, including mode of delivery, pregnancy complications (polyhydramnios, oligohydramnios, preterm birth, and preterm premature rupture of membranes), neonatal outcomes, APGAR scores, and need for neonatal

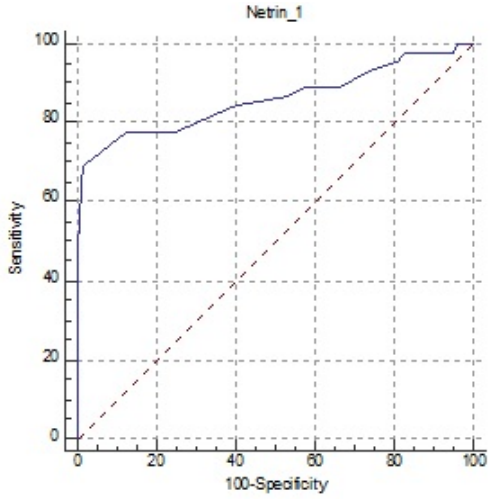

FIGURE 1. Receiver operating characteristic (ROC) curve showing the discriminatory performance of serum netrin-1 levels for gestational diabetes mellitus.

intensive care unit (NICU) admission were also collected.

Biochemical Measurements

Venous blood samples (5 mL) were collected into serum separator tubes, centrifuged at 3,500 rpm for 10 minutes, and stored at −80 °C until analysis. Serum netrin-1 levels were measured using a commercial Human Netrin-1 ELISA kit (ELK Biotechnology, ELK2210) according to the manufacturer's instructions, with absorbance read on a DAR 800 microplate reader. The intra- and inter-assay coefficients of variation were <8% and <10%, respectively.

Statistical Analysis

Continuous variables were presented as mean±standard deviation or median (minimum-maximum), and categorical variables as number and percentage. Normality was assessed using the Shapiro–Wilk test. Group comparisons were performed using Student's t-test or Mann–Whitney U test for continuous variables, and Chi-square or Fisher's exact test for categorical variables, as appropriate. ROC analysis was used to assess the discriminatory performance of serum netrin-1 for GDM, with the optimal cut-off determined by Youden's index. Multivariable logistic regression and Spearman correlation analyses were performed to identify associations after adjustment for selected covariates. For logistic regression analysis, serum netrin-1 levels were log-transformed due to non-normal distribution and skewness. Odds ratios were calculated per unit increase in the log-transformed netrin-1 values to improve interpretability and model stability. Statistical analyses were conducted using SPSS version 22.0 and MedCalc version 18. A P value <0.05 was considered statistically significant.

RESULTS

The demographic and clinical characteristics of the study groups are presented in Table 1. Maternal age, gravida, parity, and gestational week at sampling were comparable between GDM and control groups (all P>0.05). BMI, fasting glucose, and HbA1c were significantly higher in the GDM group compared with controls (P=0.011, P<0.001 and P=0.001, respectively). Serum netrin-1 levels differed significantly between groups, with lower netrin-1 levels observed in women with GDM (P<0.001).

Maternal and neonatal outcomes of the study groups are summarized in Table 2. The incidence of polyhydramnios and macrosomia was significantly higher in GDM group compared with controls (both P<0.001), and gestational age at delivery was significantly lower in women with GDM (P=0.001). Rates of postpartum hemorrhage, maternal intensive care unit admission, and NICU admission were also significantly higher in the GDM group (P=0.025, P=0.002, and P=0.023, respectively). Overall maternal and fetal complication rates were significantly increased in pregnancies complicated by GDM compared with controls (P<0.001 and P=0.008, respectively). These outcome analyses are exploratory in nature.

In ROC analysis, the optimal cut-off value of serum netrin-1 for distinguishing GDM from controls was 0.09 ng/mL, yielding a sensitivity of 68.9% and a high specificity of 98.8% (AUC=0.861, 95% CI: 0.787–0.916; P<0.001), indicating good discriminative performance (Figure 1).

Spearman correlation analysis demonstrated a moderate negative correlation between serum netrin-1 levels and fasting blood glucose levels (r= −0.446,

TABLE 1. Demographic and Clinical Characteristics of the Study Groups

Variables	Gestational diabetes mellitus (n=45)	Control (n=80)	P-value
Age (years)	30 (20–35)	29 (20–35)	0.932
Body mass index (kg/m²)	28.2 ± 2.56	26.9 ± 2.47	**0.011**
Gravida	2 (1–4)	2 (1–8)	0.455
Parity	1 (0–3)	1 (0–5)	0.136
Gestational week at sampling	27 (24–28)	26 (24–28)	0.154
Fasting glucose (mg/dL)	96 (70–204)	79 (53–125)	**<0.001**
HbA1c (%)	5.90 (4.20–7.78)	5.30 (4.10–7.10)	**0.001**
Netrin-1 (ng/mL)	0.09 (0.01–0.49)	0.13 (0.09–1.40)	**<0.001**

Data are shown as mean±standard deviation or median (minimum–maximum) where appropriate. Continuous variables were compared using Student's t-test or Mann–Whitney U test.

Statistically significant P-values are shown in bold.

TABLE 2. Maternal and Neonatal Outcomes of the Study Groups

	Gestational diabetes mellitus (n=45)	Control (n=80)	P-value
Polyhydramnios, n (%)	20 (44.4)	2 (2.5)	**<0.001**
Oligohydramnios, n (%)	1(2.2)	6 (7.5)	0.420
IUGR, n (%)	2 (4.4)	7 (8.8)	0.487
Macrosomia, n (%)	7 (15.6)	0 (0)	**<0.001**
Preterm rupture of membranes, n (%)	2 (4.4)	3 (3.8)	1.000
Gestational age at delivery (weeks)	38 (28-40)	39 (28-41)	**0.001**
Cesarean section, n (%)	31 (68.9)	48 (60.0)	0.323
Birth weight (g)	3380 (1170-4560)	3320 (1340-4030)	0.219
Preterm birth, n (%)	7 (15.6)	11 (13.8)	0.783
Postpartum bleeding, n (%)	6 (13.3)	2 (2.5)	**0.025**
APGAR score (5th minute)	10 (6-10)	10 (0-10)	0.333
Maternal intensive care unit, n (%)	10 (22.2)	3 (3.8)	**0.002**
Neonatal intensive care unit, n (%)	13 (28.9)	10 (12.5)	**0.023**
Overall maternal complications, n (%)	18 (40)	10 (12.5)	**<0.001**
Overall fetal complications, n (%)	28 (62.2)	30 (37.5)	**0.008**

Data are shown as median (minimum–maximum) or number (%) where appropriate. IUGR, Intrauterine growth restriction. Overall maternal complications included postpartum hemorrhage and maternal intensive care unit admission; overall fetal complications included macrosomia, polyhydramnios, oligohydramnios, IUGR, neonatal intensive care unit admission, and preterm birth. Overall fetal or maternal complications were defined as the presence of at least one adverse outcome. As multiple complications may coexist in the same pregnancy, the individual categories were not mutually exclusive.

Comparisons between groups were performed using the Mann–Whitney U test for continuous variables and the chi-square or Fisher's exact test for categorical variables. Statistically significant P-values are shown in bold.

TABLE 3. Multivariable Logistic Regression Analysis for Predictors of Gestational Diabetes Mellitus

Variable	B	S.E.	Wald	P-value	Odds Ratio (Exp(B))	95% CI for Exp(B)
Body mass index	0.199	0.092	4.709	**0.030**	1.22	1.02–1.46
Parity	0.039	0.216	0.033	0.855	1.04	0.68–1.59
Age	−0.013	0.051	0.070	0.792	0.99	0.89–1.09
Netrin-1	−0.871	0.236	14.281	**<0.001**	0.42	0.27–0.66
Constant	−2.976	2.871	1.075	0.300	0.05	—

Multivariable logistic regression analysis was adjusted for age, body mass index, and parity. Netrin-1 values were log-transformed due to skewed distribution prior to analysis. Odds ratios represent the change in risk of gestational diabetes mellitus per unit increase in the log-transformed netrin-1 level.

Statistically significant P-values are shown in bold.

P=0.002). In addition, netrin-1 levels showed a strong negative correlation with HbA1c levels (r= −0.623, P<0.001). No significant correlations were observed between netrin-1 levels and age, BMI, or parity (all P>0.05).

In multivariable logistic regression analysis adjusted for age, BMI, and parity, log-transformed serum netrin-1 levels were significantly associated with GDM (OR: 0.42, 95% CI: 0.27–0.66; P<0.001), indicating an inverse association. BMI also remained significantly associated with GDM, whereas age and parity were not significantly associated (both P>0.05) (Table 3).

DISCUSSION

In this prospective case–control study, maternal circulating netrin-1 levels at the time of GDM diagnosis were significantly lower in women with GDM compared with controls. ROC analysis demonstrated a good discriminative performance for serum netrin-1 (AUC=0.861), with high specificity but moderate sensitivity, suggesting that while netrin-1 may help identify individuals unlikely to have GDM, its clinical utility as a standalone diagnostic test is limited. Therefore, netrin-1 may be more appropriately considered as a complementary biomarker rather than a primary diagnostic tool in GDM.

After adjustment for maternal age, BMI, and parity, log-transformed serum netrin-1 levels remained significantly associated with GDM, indicating an inverse association between circulating netrin-1 and the presence of GDM. Additionally, netrin-1 levels were inversely correlated with fasting plasma glucose and HbA1c, indicating an association with glycemic dysregulation during pregnancy.

Netrin-1 is a laminin-related protein initially identified as a neuronal guidance cue in axonal migration. Given its regulatory roles in angiogenesis, inflammation, and metabolic homeostasis in peripheral tissues, netrin-1 has attracted increasing attention in the pathophysiology of metabolic disorders, including diabetes [6, 15].

However, clinical data regarding the relationship between netrin-1 and diabetes remain inconsistent [9-11, 16, 17]. A recent meta-analysis published in 2024, including eight studies, found no significant difference in circulating netrin-1 levels between patients with diabetes and healthy controls, although substantial heterogeneity was observed. Notably, the same analysis demonstrated significantly lower netrin-1 levels in individuals with prediabetes compared with healthy controls, suggesting that alterations in netrin-1 may occur early in the dysglycemic continuum [18]. In this context, pregnancy represents a unique metabolic and vascular state in which early disturbances in glucose homeostasis may be further accentuated.

Despite growing interest in netrin-1 in metabolic disease, data specifically addressing its role in GDM remain extremely limited. To date, only one study has directly explored the association between netrin-1 and GDM. Prieto *et al*. [12] demonstrated that netrin-1 exerted pro-angiogenic effects in human umbilical vein endothelial cells derived from pregnancies complicated by GDM, accompanied by downregulation of its anti-angiogenic receptor UNC5b.

However, given the cross-sectional design of the present study, causal or mechanistic interpretations cannot be made. Our findings should therefore be interpreted as evidence of an association between circulating netrin-1 levels and GDM rather than proof of a direct pathophysiological role.

These findings are consistent with a possible association between netrin-1 signaling and vascular adaptations in diabetic pregnancy. Our study extends this experimental evidence by providing, to our knowledge, the first clinical data demonstrating reduced maternal circulating netrin-1 levels in pregnancies complicated by GDM.

Our findings partially align with Nedeva *et al*., who reported lower netrin-1 levels in individuals with obesity and prediabetes, associated with carbohydrate metabolism disturbances and inversely correlated with BMI [19]. In contrast, in our cohort, netrin-1 levels were not significantly associated with BMI, and the optimal cut-off value for identifying GDM was lower. These discrepancies may reflect variations in study populations, metabolic phenotypes, and the unique physiological adaptations of pregnancy, including progressive insulin resistance, placental hormone secretion, and altered inflammatory signaling.

Although netrin-1 was not significantly associated with major obstetric or neonatal outcomes in this cohort, including macrosomia, preterm birth, or postpartum hemorrhage, these analyses were exploratory and not adjusted for multiple comparisons; therefore, findings should be interpreted cautiously. Therefore, no definitive conclusions can be drawn regarding the prognostic value of netrin-1 for pregnancy outcomes in GDM, and future studies specifically powered for clinical endpoints are warranted.

Strengths and Limitations

Strengths of this study include its prospective design, evaluation of both biochemical and clinical parameters, and adjustment for key maternal characteristics. Several limitations should also be acknowledged. First, the relatively modest sample size may limit statistical power, particularly for the analysis of obstetric and neonatal outcomes. However, the study was prospectively designed with an a priori sample size calculation, and the primary objective was to evaluate differences in circulating netrin-1 levels between pregnancies complicated by GDM and controls. Outcome analyses were exploratory in nature, and larger multicenter studies for clinical endpoints are warranted to further clarify the prognostic implications of netrin-1 in GDM. Second, serum netrin-1 levels were measured at a single time point during mid-pregnancy, precluding assessment of longitudinal changes throughout gestation. As pregnancy is characterized by dynamic metabolic and vascular adaptations, future longitudinal studies are needed to determine whether netrin-1 trajectories differ across gestation and whether early pregnancy levels may predict the subsequent development of GDM. Third, although BMI was adjusted for in regression models, residual confounding related to adiposity or other metabolic factors cannot be fully excluded.

CONCLUSION

This study provides preliminary clinical evidence that maternal netrin-1 levels are reduced in pregnancies complicated by GDM and are associated with markers of glycemic dysregulation. Although netrin-1 does not appear to function as a standalone diagnostic marker, our findings support its potential role as a complementary biomarker reflecting underlying metabolic alterations in GDM. Future multicenter studies with larger sample sizes and longitudinal designs are needed to validate these findings, and further investigate the associations between netrin-1, glucose metabolism, angiogenesis, and pregnancy outcomes.

Ethics Approval and Consent to Participate

This study was approved by the University of Health Sciences, Bursa Yuksek Ihtisas Training and Research Clinical Research Ethics Committee. (Decision No: 2011-KAEK-25 2023/08-03; date: 09.08.2023). All procedures were conducted in accordance with the ethical standards of the institutional and national research committee and with the 1964 Helsinki Declaration and its later amendments. Written informed consent was obtained from all participants.

Data Availability

The data that support the findings of this study are

available on request from the corresponding author, upon reasonable request.

Authors' Contribution

Study Conception: AE, NKE; Study Design: BC, AE; Supervision: BD, AE; Funding: BC, NKE; Materials: BC, NKE; Data Collection and/or Processing: BC, BD; Statistical Analysis and/or Data Interpretation: EKG, BD; Literature Review: EKG; Manuscript Preparation: EKG; and Critical Review: NKE, BD.

Conflict of Interest

The author(s) disclosed no conflict of interest during the preparation or publication of this manuscript.

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Generative Artificial Intelligence Statement

During the preparation of this work, the authors used ChatGPT (OpenAI) to assist with language editing, and restructuring of the manuscript draft. After using this tool, the authors thoroughly reviewed and edited the content as needed and take full responsibility for the final version of the manuscript. The all content of the study was produced by the author(s) in accordance with scientific research methods and academic ethical principles.

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REFERENCES

1. Wong T, Immanuel J, Wong VW, Barnes R, Simmons D. Diagnosis of gestational diabetes: Evidence and pitfalls. Best Pract Res Clin Endocrinol Metab. 2025;39(6):102069. doi: 10.1016/j.beem.2025.102069.

2. Mittal R, Prasad K, Lemos JRN, Arevalo G, Hirani K. Unveiling Gestational Diabetes: An Overview of Pathophysiology and Management. Int J Mol Sci. 2025;26(5):2320. doi: 10.3390/ijms26052320.

3. Umer A, Kanwal S, Ahmad A, et al. Maternal and Neonatal Outcomes Associated With Preeclampsia and Gestational Diabetes Mellitus: A Retrospective Cohort Study. Cureus. 2025;17(6):e86120. doi: 10.7759/cureus.86120.

4. Soma-Pillay P, Nelson-Piercy C, Tolppanen H, Mebazaa A. Physiological changes in pregnancy. Cardiovasc J Afr. 2016;27(2):89-94. doi: 10.5830/CVJA-2016-021.

5. Olmos-Ortiz A, Flores-Espinosa P, Díaz L, Velázquez P, Ramírez-Isarraraz C, Zaga-Clavellina V. Immunoendocrine Dysregulation during Gestational Diabetes Mellitus: The Central Role of the Placenta. Int J Mol Sci. 2021;22(15):8087. doi: 10.3390/ijms22158087.

6. Xia X, Hu Z, Wang S, Yin K. Netrin-1: An emerging player in inflammatory diseases. Cytokine Growth Factor Rev. 2022;64:46-56. doi: 10.1016/j.cytogfr.2022.01.003.

7. Claro V, Ferro A. Netrin-1: Focus on its role in cardiovascular physiology and atherosclerosis. JRSM Cardiovasc Dis. 2020;9:2048004020959574. doi: 10.1177/2048004020959574.

8. Toque HA, Fernandez-Flores A, Mohamed R, Caldwell RB, Ramesh G, Caldwell RW. Netrin-1 is a novel regulator of vascular endothelial function in diabetes. PLoS One. 2017;12(10):e0186734. doi: 10.1371/journal.pone.0186734.

9. Liu C, Ke X, Wang Y, et al. The level of netrin-1 is decreased in newly diagnosed type 2 diabetes mellitus patients. BMC Endocr Disord. 2016;16(1):33. doi: 10.1186/s12902-016-0112-z.

10. Ay E, Marakoğlu K, Kizmaz M, Ünlü A. Evaluation of Netrin-1 Levels and Albuminuria in Patients With Diabetes. J Clin Lab Anal. 2016;30(6):972-977. doi: 10.1002/jcla.21965.

11. White JJ, Mohamed R, Jayakumar C, Ramesh G. Tubular injury marker netrin-1 is elevated early in experimental diabetes. J Nephrol. 2013;26(6):1055-64. doi: 10.5301/jn.5000303.

12. Prieto CP, Casas BS, Falcón P, et al. Downregulation of the Netrin-1 Receptor UNC5b Underlies Increased Placental Angiogenesis in Human Gestational Diabetes Mellitus. Int J Mol Sci. 2019;20(6):1408. doi: 10.3390/ijms20061408.

13. Sert ZS. The role of serum netrin-1 level in the detection of early-onset preeclampsia. Eur J Obstet Gynecol Reprod Biol. 2022;270:164-168. doi: 10.1016/j.ejogrb.2022.01.010.

14. American Diabetes Association Professional Practice Committee. 2. Diagnosis and Classification of Diabetes: Standards of Care in Diabetes-2024. Diabetes Care. 2024;47(Suppl 1):20-42. doi: 10.2337/dc24-S002.

15. Ziegon L, Schlegel M. Netrin-1: A Modulator of Macrophage Driven Acute and Chronic Inflammation. Int J Mol Sci. 2021;23(1):275. doi: 10.3390/ijms23010275.

16. Yim J, Kim G, Lee BW, et al. Relationship Between Circulating Netrin-1 Concentration, Impaired Fasting Glucose, and Newly Diagnosed Type 2 Diabetes. Front Endocrinol (Lausanne). 2018;9:691. doi: 10.3389/fendo.2018.00691.

17. Chaitra D, Pai Divya N, Raghavendra H, Kamath D. Exploring The Role of Serum Netrin 1 Levels in Diabetic Foot

Syndrome And Uncomplicated Type 2 Diabetes Mellitus: A Comparative Analysis. Clin Ter. 2025;176(2):201-206. doi: 10.7417/CT.2025.5206.
18. Behnoush AH, Khalaji A, Shokri Varniab Z, Rahbarghazi A, Amini E, Klisic A. Urinary and circulatory netrin-1 as biomarker in diabetes and its related complications: a systematic review and meta-analysis. Endocrine. 2024;84(2):328-344. doi: 10.1007/s12020-023-03598-y.
19. Nedeva I, Gateva A, Assyov Y, Karamfilova V, Velikova T, Kamenov Z. Relationship between circulating netrin-1 levels, obesity, prediabetes and newly diagnosed type 2 diabetes. Arch Physiol Biochem. 2022;128(6):1533-1538. doi: 10.1080/13813455.2020.1780453.