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Original Research Article

ASSOCIATION OF ELEVATED SERUM FETUIN-A LEVELS WITH OBESITY IN TYPE 2 DIABETES MELLITUS: A COMPARATIVE CROSS-SECTIONAL STUDY FROM CENTRAL INDIA

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ABSTRACT

Background: Fetuin-A, a glycoprotein secreted by the liver, has emerged as a potential metabolic biomarker associated with obesity and Type 2 Diabetes Mellitus (T2DM). Elevated serum levels of Fetuin-A have been linked to various metabolic alterations, including increased body mass index (BMI), hyperglycemia, elevated HbA1c, dyslipidemia, and insulin resistance. This study aimed to assess the association between elevated serum Fetuin-A levels and obesity in individuals with T2DM.

Materials and Methods: A comparative cross-sectional study was conducted involving 100 participants, including obese T2DM patients and healthy controls. Serum Fetuin-A levels were quantified using the enzyme-linked immunosorbent assay (ELISA) method.

Results: Serum Fetuin-A levels were significantly higher in obese T2DM patients compared to healthy controls ($p < 0.05$). A positive correlation was observed between elevated Fetuin-A levels and key metabolic parameters, including BMI, fasting blood glucose, HbA1c, and lipid profile abnormalities.

Conclusion: High serum Fetuin-A levels are significantly associated with obesity and adverse metabolic profiles in patients with Type 2 Diabetes Mellitus. Fetuin-A may serve as a potential biomarker for identifying metabolic risk in obese diabetic individuals.

Keywords: Fetuin-A, Type 2 Diabetes Mellitus, Obesity, Biomarker, Insulin Resistance.

INTRODUCTION

In India, diabetes is the most important pathological state affecting more than 62 million people in India.[1] In other developed and developing countries, diabetes in combination with hypertension are the most common non communicable disease. Diabetes is a multi-factorial disease including genetic defect, environmental factor, obesity, urban migration and changes in life style.[2] are responsible for the development of diabetes. Pathogenic factors are also included like autoimmune destruction of the beta cells of pancreas and insulin resistance in cell.[3] The major abnormalities in diabetes are metabolic defects in carbohydrate, lipid and protein because of the low action of insulin on target tissue. Inadequate or defective insulin secretion diminishes tissue response for insulin and for this complex metabolic pathway is responsible.4 The major abnormalities of diabetes are hyperglycemia, polyuria, polydipsia, weight loss, polyphagia and blurred vision.[5]

Obesity is a serious clinical condition where patients have accumulated overabundance adipose tissue that cause serious complications such as metabolic syndrome, type 2 diabetes mellitus and hypertension.[6] The prevalence of obesity in Indian adults aged 20-69 years will be 27.4%-30.4% in 2040.[7] According to the statistics released by the International Diabetes Federation (IDF) in December 2020, there were about 590 million diabetic patients worldwide, who mainly suffered from type 2 diabetes mellitus 75% were from developingcountries.[8]

Obesity and type 2 diabetes mellitus have become a burden globally as healthcare costs have increased with a growing population.[9] Recently published data imply that most novel biomarkers such as fetuin-A with other metabolic markers help to understand their role in the pathophysiology of vascular disease. Recently, it has been reported that fetuin-A–deficient mice demonstrate enhanced insulin sensitivity.[10] These data indicate that fetuin-A might be a negative regulator of insulin signaling in cells. Serum fetuin-A (also called alpha-2 heremansschmid glycoprotein, AHSG) is a glycoprotein with many functions, which is exclusively secreted from liver cell in human. Fetuin-A has been considered to play an important role in the protection from vascular calcification by solubilizing calcium and phosphorus in serum.[11] It was also reported that fetuin-A also inhibit insulin receptor dependent tyrosine kinase activity through inhibiting the auto phosphorylation of tyrosine kinase and insulin receptor substrate-1(IRS-1) and induced a lower-grade inflammation, which resulted in insulin resistance. Insulin resistance is a conspicuous characteristic of prediabetic states. So, that serum fetuin – A is associated with prediabetes as well as type 2 diabetes.

Role of Fetuin-A in type 2 diabetes

Insulin resistance is one of the most important pathophysiological mechanisms of type 2 diabetes. It is also related with dyslipidemia and hypertensive disease. Fetuin-A is an important biomarker for risk of type 2 diabetes. Higher level of fetuin-A associated with insulin resistance and HOMA –IR. In addition to that it is also positively related with HbA1c, TG,LDL-cholesterol, fasting glucose, but negatively related with fasting plasma insulin and LDL-cholesterol.[14]

Objectives

- To evaluate the association between elevated serum Fetuin-A levels and obesity in patients with Type 2 Diabetes Mellitus in Central India.

MATERIALS AND METHODS

Study Design

This is a **comparative cross – sectional study** conducted at a tertiary care center in Central India.

Study Population and Sample Size

The study population comprised obese individuals with Type 2 Diabetes Mellitus (T2DM) and age- and sex-matched healthy controls. All eligible participants who underwent physical examination and laboratory investigations at the tertiary care center in Central India during the study period were included. A total of 200 participants were enrolled, with 100 patients in the obese T2DM group and 100 individuals in the healthy control group.

Study Duration

The study was carried out over a period of six months, from April 2022 to November 2023.

Inclusion Criteria for Study

1) Adults aged up to 70 years.
2) Newly diagnosed cases of Type 2 Diabetes Mellitus.
3) Diagnosis of Type 2 Diabetes Mellitus confirmed as per American Diabetes Association (ADA) criteria, based on fasting blood glucose (FBG), HbA1c, and lipid profile parameters.

Exclusion Criteria for Study

1) Patients diagnosed with any condition other than Type 2 Diabetes Mellitus, such as acute myocardial infarction, renal failure, liver disease, critical illness, tuberculosis, carcinoma, or any severe infection.
2) Women who are pregnant or lactating.
3) Patients with clinically diagnosed anaemia or haemoglobin levels below standard reference ranges.

Ethical Considerations

- Approval was taken from the Institutional Ethics Committee (IEC).
- Informed written consent in subject's vernacular language was taken after apprising them of the nature and purpose of study.

Data Collection Parameters

➢ Data were collected using a pre-tested, structured proforma after obtaining written informed consent from all participants.

A. Baseline sociodemographic information included:

- Age
- Sex
- Educational status
- Occupation
- Socioeconomic status
- Lifestyle factors (diet and physical activity)
- Family history of diabetes or other chronic illnesses

B. Anthropometric measurements:

- Weight and height were measured using standard procedures.
- **Body Mass Index (BMI)** was calculated using the formula:
  BMI = Weight (kg) / Height² (m²)
- Obesity was defined as BMI ≥ 25 kg/m² (WHO criteria for Asia – pacific criteria).

C. Biochemical investigations were performed using fasting venous blood samples:

- **Fasting Blood Glucose (FBG):** Measured using the glucose oxidase-peroxidase (GOD-POD) colorimetric method.
- **HbA1c:** Estimated using a turbidimetric inhibition immunoassay.
- **Lipid Profile:** Total cholesterol, triglycerides, and HDL-C measured enzymatically.
- **Plasma Insulin:** Assessed using a solid-phase enzyme-linked immunosorbent assay (ELISA).
- **Serum Fetuin-A:** Quantified using sandwich ELISA technique.
- **Insulin Resistance (HOMA-IR):** Calculated using the formula:

HOMA-IR = (Fasting Insulin [µIU/mL] × Fasting Glucose [mmol/L]) / 22.5

- All biochemical analyses were conducted in a single accredited laboratory following standard quality control protocols.
- **Statistical Analysis -** Data entry and analysis were performed using SAS 9.2, SPSS 15.0, and R version 4.4.2.
- Descriptive statistics were used to summarize baseline variables.
  - **Continuous variables:** Summarized as mean with standard deviation.
  - **Categorical variables:** Summarized in terms of proportion, frequency and percentage.
- Independent t-tests were used to compare means between groups. A p-value of < 0.05 was considered statistically significant.

RESULTS

Table 1: Age-wise Comparison of Biomarker Levels Between Subjects and Controls

Biochemical Parameters	Age	Obese type 2 DM (Mean±SD)	Healthy (Mean±SD)	p-value
FBG (mg/dl)	41 – 50	209.34 ± 66.52	89.21 ± 9.35	<0.001
	51 – 60	203.55 ± 60.99	90.84 ± 8.97	<0.001
	61 – 70	206.28 ± 64.27	88.71 ± 9.15	<0.001
HbA1c (%)	41 – 50	8.87± 1.272	5.27 ± 0.14	<0.001
	51 – 60	8.85 ± 1.26	5.28 ± 0.17	<0.001
	61 – 70	8.77 ± 1.28	5.28 ± 0.15	<0.001
TC (mg/dl)	41 – 50	177.55 ± 42.43	134.41 ± 25.80	<0.001
	51 – 60	176.41 ± 45.71	137.75 ± 24.26	<0.001
	61 – 70	178.17 ± 38.27	131.79 ± 23.49	<0.001
TG (mg/dl)	41 – 50	267.59 ± 88.06	103.74 ± 21.62	<0.001
	51 – 60	275.17 ± 90.18	107.28 ± 22.02	<0.001
	61 – 70	257.96 ± 84.85	104.20 ± 21.77	<0.001
HDL (mg/dl)	41 – 50	36.75 ± 10.50	50.94 ± 6.24	<0.001
	51 – 60	36.23 ± 11.52	51.19 ± 5.97	<0.001
	61 – 70	38.64 ± 12.04	51.37 ± 6.09	<0.001
LDL (mg/dl)	41 – 50	101.18 ± 41.06	90.94 ± 21.12	0.028
	51 – 60	99.79 ± 46.16	89.46 ± 22.13	0.046
	61 – 70	100.98 ± 36.52	91.60 ± 20.61	0.027
VLDL (mg/dl)	41 – 50	52.91 ± 23.94	19.39 ± 3.95	<0.001
	51 – 60	56.69 ± 24.29	19.42 ± 4.48	<0.001
	61 – 70	50.85 ± 23.74	19.37 ± 3.91	<0.001
Insulin (mµ/l)	41 – 50	30.24 ± 3.03	10.69 ± 2.68	<0.001
	51 – 60	30.33 ± 3.03	10.71 ± 3.12	<0.001
	61 – 70	30.29 ± 3.01	11.55 ± 3.59	<0.001
HOMA-IR	41 – 50	28.27 ± 1.83	12.01 ± 3.42	<0.001
	51 – 60	55.87 ± 25.39	18.58 ± 4.13	<0.001
	61 – 70	58.74 ± 24.82	10.77 ± 3.31	<0.001
Fetuin-A (µg/ml)	41 – 50	335.06 ± 6.67	263.77 ± 6.31	<0.001
	51 – 60	335.37 ± 6.10	264.81 ± 6.39	<0.001
	61 – 70	335.29 ± 6.74	263.67 ± 6.26	<0.001
BMI (kg/m^2)	41 – 50	38.43 ± 4.94	21.48 ± 1.76	<0.001
	51 – 60	38.01 ± 4.98	21.09 ± 1.70	<0.001
	61 – 70	38.62 ± 4.74	21.37 ± 1.74	<0.001

p<0.05(Significant), p<0.001(Highly significant)

Table 01 presents the comparison of metabolic parameters between cases and controls across three age groups (41–50, 51–60, and 61–70 years).

Across all age categories, cases showed significantly higher levels of fasting blood glucose (FBG) and HbA1c compared to controls ($p < 0.001$), indicating poor glycemic control. Lipid parameters (TC, TG, LDL, VLDL) were significantly elevated in cases, while HDL levels were lower ($p < 0.001$), reflecting dyslipidemia.

Serum insulin and HOMA-IR were markedly higher in cases, suggesting substantial insulin resistance ($p < 0.001$). Fetuin-A levels were significantly elevated in cases across all age groups, supporting its role as a potential marker of metabolic dysfunction.

BMI was significantly higher among cases (mean ~38 kg/m^2) compared to controls (~21 kg/m^2) across all age groups ($p < 0.001$), highlighting the presence of obesity in the affected group.

These differences were statistically significant across all age groups, indicating a consistent pattern of metabolic derangement in cases.

Table 2: Gender-wise Comparison of Biomarker Levels Between Subjects and Controls

Biochemical Parameters	Gender	Obese type 2 DM (Mean±SD)	Healthy (Mean±SD)	p-value
FBG (mg/dl)	Male	227.38 ± 67.35	89.03 ± 9.22	<0.001
	Female	185.02 ± 51.17	89.86 ± 9.48	<0.001
HbA1c (%)	Male	9.05 ± 1.22	5.26 ± 0.17	<0.001
	Female	8.60 ± 1.26	5.29 ± 0.13	<0.001
TC (mg/dl)	Male	185.55 ± 50.54	130.22 ± 28.85	<0.001
	Female	170.85 ± 31.07	138.75 ± 18.17	<0.001
TG (mg/dl)	Male	260.16 ± 88.30	98.65 ± 19.01	<0.001
	Female	277.41 ± 88.69	110.71 ± 23.12	<0.001
HDL (mg/dl)	Male	38.83 ± 10.87	46.61 ± 4.74	<0.001
	Female	35.48 ± 11.45	56.02 ± 2.75	<0.001
LDL (mg/dl)	Male	109.34 ± 44.76	84.98 ± 22.57	<0.001
	Female	92.47 ± 35.93	96.76 ± 17.19	0.137
VLDL (mg/dl)	Male	51.75 ± 21.39	20.06 ± 4.10	<0.001
	Female	55.78 ± 26.20	18.85 ± 4.04	<0.001
Insulin (mμ/l)	Male	31.77 ± 2.59	11.45 ± 3.28	<0.001
	Female	28.84 ± 2.72	10.68 ± 3.15	<0.001
HOMA-IR	Male	58.43 ± 25.30	11.87 ± 4.13	<0.001
	Female	54.52 ± 23.10	16.32 ± 3.03	<0.001
Fetuin-A (μg/ml)	Male	334.83 ± 7.23	264 ± 6.21	<0.001
	Female	335.57 ± 5.56	263.73 ± 6.36	<0.001
BMI (kg/m^2)	Male	37.75 ± 5.04	21.39 ± 1.50	<0.001
	Female	38.71 ± 4.78	21.20 ± 1.97	<0.001

Table 02 presents a gender-wise comparison of biochemical parameters between obese individuals with type 2 diabetes mellitus (T2DM) and healthy controls.

Both male and female diabetics showed significantly higher levels of FBG and HbA1c compared to controls ($p < 0.001$), indicating poor glycemic control. Lipid parameters (TC, TG, VLDL, and LDL) were elevated in diabetics, while HDL was significantly lower in both sexes ($p < 0.001$). The difference in LDL among females was not statistically significant ($p = 0.137$).

Insulin levels and HOMA-IR were markedly increased in both male and female diabetics ($p < 0.001$), suggesting notable insulin resistance. Fetuin-A levels were significantly elevated in the diabetic group across both genders ($p < 0.001$).

BMI was also significantly higher in diabetic males and females compared to controls ($p < 0.001$), confirming the presence of obesity.

These findings highlight consistent metabolic disturbances in obese T2DM patients irrespective of gender.

Table 3: Distribution of Biochemical Parameters in Controls and Subjects

	Group	N	Mean ± SD	Test - Value	P value
FBG mg/dl	Obese type 2 DM	100	196.6 ± 59.94	12.798	**<0.001**
	Healthy	100	86.28 ± 9.29		
HbA1c %	Obese type 2 DM	100	8.74 ± 1.35	17.976	**<0.001**
	Healthy	100	5.27 ± 0.14		
TC mg/dl	Obese type 2 DM	100	170.16 ± 33.81	7.427	**<0.001**
	Healthy	100	129.2 ± 20.98		
TG mg/dl	Obese type 2 DM	100	263.31 ± 86.68	12.645	**<0.001**
	Healthy	100	102.26 ± 21.82		
HDL mg/dl	Obese type 2 DM	100	37.01 ± 11.98	-8.348	**<0.001**
	Healthy	100	51.82 ± 5.8		
LDL mg/dl	Obese type 2 DM	100	97.64 ± 33.99	0.828	0.412
	Healthy	100	92.86 ± 20.46		
VLDL mg/dl	Obese type 2 DM	100	54.26 ± 26.13	9.564	**<0.001**
	Healthy	100	19.24 ± 4.21		
Insulin mμ/l	Obese type 2 DM	100	29.52 ± 3.02	28.423	**<0.001**
	Healthy	100	11.1 ± 3.19		
HOMA-IR	Obese type 2 DM	100	160.16 ± 32.81	29.435	**<0.001**
	Healthy	100	119.2 ± 20.88		
FetuinA μg/ml	Obese type 2 DM	100	334.69 ± 6.29	54.607	**<0.001**
	Healthy	100	263.07 ± 6.06		
BMI Kg/m^2	Obese type 2 DM	100	39.22 ± 4.43		**<0.001**
	Healthy	100	21.2 ± 1.61		

p<0.05(Significant), p<0.001(Highly significant)

Table 03 shows the distribution of biochemical parameters in obese type 2 diabetic subjects and healthy controls. Significant differences were observed in the mean values of FBG, HbA1c, insulin, HOMA-IR, TC, TG, LDL, VLDL, HDL, BMI, and Fetuin-A. All parameters—except HDL, which was

significantly lower—were significantly higher in obese type 2 diabetic subjects compared to healthy controls.

DISCUSSION

Fetuin-A is a multifunctional glycoprotein that plays a key role in metabolic regulation. It is abundantly synthesized during fetal development by multiple tissues and predominantly secreted by the liver in adults. In the context of type 2 diabetes mellitus (T2DM), Fetuin-A has been shown to impair insulin signaling by inhibiting the autophosphorylation of the insulin receptor, thereby inducing insulin resistance and contributing to hyperglycemia and hyperinsulinemia [Joachim et al., 2006; Stefan et al., 2008].

In our study, the mean plasma levels of Fetuin-A were significantly higher in T2DM patients compared to healthy controls ($p < 0.0001$). This elevation was accompanied by significant increases in fasting blood glucose (FBG), HbA1c, insulin, HOMA-IR, total cholesterol (TC), triglycerides (TG), low-density lipoprotein (LDL), very low-density lipoprotein (VLDL), and body mass index (BMI), along with reduced levels of high-density lipoprotein (HDL). These findings strongly support the role of Fetuin-A in the development of both insulin resistance and dyslipidemia in obese T2DM patients.

Hypercholesterolemia, hypertriglyceridemia, and hyperinsulinemia, as observed in our study population, may stimulate hepatic synthesis of Fetuin-A. Our results align with previous research by Wojtysiak-Duma et al. (2010), Ishibashi et al. (2010), Song et al. (2011), Sun et al. (2013), Ahmed et al. (2014), and Moustafa (2016), all of whom reported elevated Fetuin-A levels in individuals with insulin resistance or obesity-related T2DM.

A 2020 meta-analysis by Liu S et al. confirmed that elevated circulating Fetuin-A is a consistent biomarker of metabolic dysfunction in individuals with impaired glucose regulation. Furthermore, a recent study by Mostafa EA et al. (2022) in the Indian population emphasized the potential of Fetuin-A as a predictive marker for cardiovascular and hepatic complications in diabetes.

Fetuin-A has also been implicated in cardiovascular complications of diabetes. Studies have identified it as a marker of coronary atherosclerosis and vascular calcification in T2DM [Lin YH et al., 2024]. Elevated Fetuin-A levels are thought to mediate endothelial dysfunction, inflammation, and lipid accumulation, all of which contribute to atherogenesis.

Beyond its metabolic and cardiovascular effects, Fetuin-A may also impact pancreatic beta-cell function. Impaired beta-cell insulin secretion is a hallmark of fasting hyperglycemia in T2DM. Fetuin-A–induced insulin resistance may exacerbate this dysfunction, leading to further disruption of glucose homeostasis. As such, Fetuin-A is not only a marker of insulin resistance but also a potential contributor to disease progression in individuals with compromised beta-cell function [Mostafa EA et al., 2022].

Collectively, our findings support the hypothesis that Fetuin-A serves as an independent and multifaceted marker of obese type 2 diabetes. Its involvement in insulin resistance, lipid metabolism, beta-cell function, and cardiovascular risk underlines its potential as a biomarker for disease screening and progression monitoring. Routine measurement of plasma Fetuin-A could therefore be particularly valuable in identifying individuals at high risk for developing T2DM and its associated complications.

CONCLUSION

Our findings confirm that elevated Fetuin A in obese T2DM patients correlates with insulin resistance, dyslipidemia, NAFLD, and cardiovascular risk. Supported by genetic, mechanistic, and epidemiological data, Fetuin A emerges as a key mediator and potential marker in T2DM pathogenesis. Plasma Fetuin A measurement may thus be essential for stratifying risk in individuals predisposed to obesity associated T2DM.

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Author Contributions

Dr. Shailja Kurele: Conceptualization, Methodology, Investigation, Writing – original draft, Formal analysis.

Dr. Sapna Singh: Writing – review & editing, Visualization.

Dr. Jusmita Dutta: Supervision, Project administration, Writing – review & editing.

All authors: Validation, Critical review, and approval of the final manuscript

REFERENCES

1. American Association of Clinical Endocrinologists. AACE Comprehensive Diabetes Algorithm 2013 Consensus Statement. Endocr Pract Suppl. 2015;21(Suppl 1):1–87.
2. Purcell K, Sumithran P, Prendergast LA, Bouniu CJ, Delbridge E, Proietto J. The effect of rate of weight loss on long-term weight management: a randomised controlled trial. Lancet Diabetes Endocrinol. 2014;2(12):954–62.
3. Bonora E, Dauriz M, Rinaldi E, Mantovani A, Boscari F, Mazzuccato M, et al. Chronic complications in diabetes: a comprehensive update. J Endocrinol Invest. 2021;44(1):75. doi:10.3847/s40618-020-01270-0.
4. Oguntibeju OO. Type 2 diabetes mellitus, oxidative stress and inflammation: examining the links. Int J Physiol Pathophysiol Pharmacol. 2019;11(3):45–63.

5. Galicia-Garcia U, Benito-Vicente A, Jebari S, Larrea-Sebal A, Siddiqi H, Uribe KB, et al. Pathophysiology of type 2 diabetes mellitus. Int J Mol Sci. 2020;21(17):6275.
6. Sami W, Ansari T, Butt NS, Hamid MRA. Effect of diet on type 2 diabetes mellitus: A review. Int J Health Sci (Qassim). 2017;11(2):65–71.
7. Li S, Zhang E, Li M, Pan W. Research on the effectiveness of application of diabetes management APP. China Med Dev. 2015;30(08).
8. Zheng Y, Ley SH, Hu FB. Global aetiology and epidemiology of type 2 diabetes mellitus and its complications. Nat Rev Endocrinol. 2018;14(2):88–98.
9. Marín-Peñalver JJ, Martín-Timón I, Sevillano-Collantes C, Del Cañizo-Gómez FJ. Update on the treatment of type 2 diabetes mellitus. World J Diabetes. 2016;7(17):354–95.
10. Chattopadhyay M, Khemka VK, Chatterjee G, et al. Enhanced ROS production and oxidative damage in subcutaneous white adipose tissue mitochondria in obese and type 2 diabetes subjects. Mol Cell Biochem. 2015;399(1-2):95–103.
11. Ebrahimi ZS, Nasli-Esfahani E, Nadjarzade A, et al. Effect of symbiotic supplementation on glycemic control, lipid profiles and microalbuminuria in patients with non-obese type 2 diabetes: a randomized, double-blind, clinical trial. J Diabetes Metab Disord. 2017;16:23.
12. Redel JM, DiFrancesco M, Lee GR, et al. Cerebral blood flow is lower in youth with type 2 diabetes compared to obese controls: A pilot study. Pediatr Diabetes. 2018;23(3):291–300. doi:10.1111/pedi.13313.
13. Yılmaz NG, Renders CM, Nicolaou M, Vrijkotte TGM. The explanatory role of maternal feeding practices: do they explain ethnic differences in body weight of preadolescents? Appetite. 2019;142:104354.
14. Bilecik T. Metabolic Effects of Sleeve Gastrectomy with Transit Bipartition in Obese Females with Type 2 Diabetes Mellitus: Results After 1-Year Follow-up. Obes Surg. 2019;29(3):805–10.
15. Erion KA, Corkey BE. Hyperinsulinemia: a cause of obesity? Curr Obes Rep. 2017;6(2):178–86.
16. Pan X, Wen SW, Bestman PL, Kaminga AC, Acheampong K, Liu A. Fetuin-A in Metabolic syndrome: A systematic review and meta-analysis. PLoS One. 2020;15(3):e0229776.
17. Birukov A, Polemiti E, Jäger S, et al. Fetuin-A and risk of diabetes-related vascular complications: a prospective study. Cardiovasc Diabetol. 2022;21(1):6.
18. Guo VY, Cao B, Cai C, et al. Fetuin-A levels and risk of type 2 diabetes mellitus: a systematic review and meta-analysis. Acta Diabetol. 2018;55(1):87–98.
19. Öner-İyidoğan Y, Koçak H, Seyidhanoğlu M, et al. Curcumin prevents liver fat accumulation and serum fetuin-A increase in rats fed a high-fat diet. J Physiol Biochem. 2013;69(4):677–86.
20. Stefan N, Sun Q, Fritsche A, Machann J. Impact of the adipokine adiponectin and the hepatokine fetuin-A on the development of type 2 diabetes: cross-sectional studies. PLoS One. 2014;9(3):e92238.
21. Wojtysiak-Duma B, Malecha-Jedraszek A, Burska A, Duma D, Donica H. Serum fetuin-A level in patients with type 2 diabetes mellitus. Ann Univ Mariae Curie Sklodowska Med. 2010;23(2):14.
22. Gunduz FO, Yildirmak ST, Temizel M, et al. Serum visfatin and fetuin-a levels and glycemic control in patients with obese type 2 diabetes mellitus. Diabetes Metab J. 2011;35(5):523–8.
23. Lorant PD, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia. 2015;28:4467–89.
24. Öner-İyidoğan Y, Koçak H. Interaction of fetuin-A with obesity related insulin resistance and diabetes mellitus. Turkish Journal of Biochemistry. 2025 Apr 30;50(2):170-82.
25. Liu S, Xiao J, Zhao Z, Wang M, Wang Y, Xin Y. Systematic review and meta-analysis of circulating fetuin-A levels in nonalcoholic fatty liver disease. Journal of Clinical and Translational Hepatology. 2020 Dec 9;9(1):3.
26. Huang Y, Huang X, Ding L, Wang P, Peng K, Chen Y, Dai M, Zhang D, Xu M, Bi Y, Wang W. Serum fetuin-A associated with fatty liver index, early indicator of nonalcoholic fatty liver disease: a strobe-compliant article. Medicine. 2015 Sep 1;94(39):e1517.
27. Lin YH, Lin MH, Shi CS, Lin YS, Lin CL, Yang YH, Liao YS, Chen MY, Tsai MH, Lin MS. The impact of fetuin-A on predicting aortic arch calcification: secondary analysis of a community-based survey. Frontiers in Cardiovascular Medicine. 2024 Jul 8;11:1415438.
28. Mostafa EA, AlShaarawy BA, Abd El-Hamid SM, Gamal AM. Evaluation of Fetuin-A level in diabetic retinopathy. The Egyptian Journal of Internal Medicine. 2022 Dec;34(1):62.