ORIGINAL ARTICLE

Evaluation of Serum Omentin-1 Levels in Diabetes Type-II with and without Diabetic Nephropathy

Somaya S. Eissa [a], Eman M. Nasef [a], Nagwa A. Mohamed [b], Nada K. M. Elkassify [a,*]

[a] Department of Internal Medicine, Faculty of Medicine for Girls, Al-Azhar University, Cairo, Egypt
[b] Department of Clinical and Chemical Pathology, National Research Center, Cairo, Egypt

Abstract

Background: ***Type II diabetes mellitus (DM) is a disorder that is becoming increasingly prevalent worldwide. Diabetes causes many microvascular and macrovascular complications. Diabetic nephropathy (DN) is among the most dangerous complications of DM. Pathways involved in the progression of DN include oxidative stress, angiotensin II, and inflammatory processes. Omentin-1 improves insulin sensitivity by increasing insulin-dependent glucose absorption in adipose tissue cells in the visceral, subcutaneous, and liver muscles.***

Objective: ***To measure the serum omentin-1 level in type-II diabetic patients with diabetic nephropathy or without nephropathy.***

Subjects and Methods: ***This case-control involved 90 participants: 60 are type-II diabetics and 30 healthy control group. 30 of the patients have DN, and 30 have not. Each group has 15 male individuals and 15 female individuals.***

Results: ***Omentin-1 level is found to be lower in type II diabetic patients with DN than in diabetic patients without DN.***

Conclusion: ***Omentin-1 is significantly lower in type II diabetic patients with DN than in type II diabetic patients without DN, which is lower than that of healthy controls.***

Keywords: Adipocytokines; Diabetic nephropathy; Diabetes mellitus; Omentin-1

1. Introduction

Recently, diabetes mellitus (DM) has become more prevalent worldwide, making it one of the most prevalent medical conditions. Diabetes mellitus (DM) is related to many complications, including macrovascular complications and microvascular complications.[1]

The "Diabetic kidney disease" term can be defined as the existence of albuminuria, declining estimated glomerular filtration rate (eGFR), or both in diabetic patients. Albuminuria ranges between fair or moderate increase albuminuria, "previously termed microalbuminuria," and excessive or severe increase albuminuria, “previously termed macroalbuminuria" .[2,3]

Hyperglycemia is considered the leading etiological factor that leads to the incidence of diabetic kidney disease. Once hyperglycemia occurs, multiple pathophysiological events disturb tubuloglomerular feedback and cause renal hypoxia, leading to progressive glomerular sclerosis and a glomerular filtration rate (GFR) decrease.[4]

The inflammatory processes in DN and chronic kidney disease (CKD) have been predicted using a wide variety of biomolecules.[5]

Omentin-1 (OMT-1) is an adipokine which is synthesized by omental adipose tissue and has insulin-sensitizing effects. Omentin-1 is closely related to diabetes mellites and its hazards.[6]

Omentin is involved in glucose homeostasis as it improves insulin-dependent glucose uptake in human adipocytes by activating protein kinase signaling pathways. Omentin-1 expression decreased in obesity, insulin resistance, and type II DM .[7]

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* Corresponding author at: Internal Medicine, Faculty of Medicine for Girls, Al-Azhar University, Cairo, Egypt.
E-mail address: n.k.kassify@gmail.com (N. K. M. Elkassify).

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Omentin causes vasodilation and promotes inflammatory resolution.[8] Omentin-1 plays a role in regulating multiple metabolic and biological events in an endocrine manner, such as inflammation, lipid metabolism, insulin sensitivity, and immunity .[9]

Therefore, our goal is to determine if type-II diabetes individuals with or without diabetic nephropathy have elevated serum omentin-1 levels.

2. Patients and methods

Our work is a case-control study, conducted on 6 October for a health insurance hospital in collaboration with Al-Zahraa Teaching Hospital and the National Research Center during the period from November 2021 to January 2023. The thesis was conducted on 90 subjects. They were categorized into 30 type II diabetic patients with diabetic nephropathy and 30 type II diabetic patients without diabetic nephropathy. 30 healthy subjects matched in sex and age.

Patients with age above 18 and type-II DM were included in the study. Meanwhile, patients with Type-I DM, active infectious disease, pregnancy, polycystic kidney disease, malignancy, collagen vascular diseases, history of radiocontrast material or nephrotoxic drug use in last 30 days, heart failure, ischemic cardiovascular disease, or chronic obstructive pulmonary disease were excluded.

Patients' complete medical histories were reviewed, paying particular attention to the length of their diabetes, any history of acute renal damage, and any signs of chronic kidney disease. Clinical examinations include systemic and general examinations, as well as the calculation of body mass index.

Laboratory investigations included: Complete blood picture (CBC), ESR, and CRP level to exclude active inflammatory disease. Serum creatinine and blood urea, eGFR, Uric acid, Sodium concentration level, Potassium Concentration level. Lipid profile including cholesterol and triglyceride, LDL, and HDL. The Coulter Counter T890 was used to determine the CBC. CRP level, FBG, two hours Postprandial Blood glucose, HbA1C, Kidney function tests, and Lipid profile were performed on a Hitachi 912 auto-analyzer by colorimetric techniques. Urine analysis to evaluate albuminuria. Albumin/ creatinine ratio. 24-hour urinary protein. Serum Omentin-1 level is measured using enzyme-linked immunosorbent assay kits. Omentin-1 has physiological levels between 100 and 800 ng/mL.[10]

Serum omentin levels were measured using a quantitative sandwich enzyme immunoassay method provided by Cusabio (Wuhan Hi-tech Medical Devices Park, China) .[11]

Imaging: abdominal ultrasonography to exclude other causes of nephropathy or CKD.

Statistical Analysis: Version 27 of the Statistical Package for the Social Sciences (IBM SPSS) was used. For parametric data, the mean, standard deviations, and ranges were presented; for non-parametric data, the median and interquartile range (IQR) were presented. Qualitative variables were also presented as percentages and numbers. After that, appropriate statistical analyses were used. A 95% confidence interval and a 5% acceptable margin of error were established.

3. Results

Thirteen females and thirteen males patients with age groups ranging between 24 – 85 years old were recruited in the studied diseased groups (group I and group II) (Table 1).

Table 2 implements a statistically significant variations between the studied groups as the level of OMT-1 (pg/ml), the level was found remarkably higher in group III (79.17±5.82) than in group II (44.98±7.97) which was remarkebly higher also than group I (24.99±6.53) with p-value <0.001.

Table 3 shows a statistically significant increase in FBS, PP and HbA1c levels in group I and group II than in group III but with no statistically significant contrast between group I and group II.

A statistically significant increase in creatinine level in group I [4.2 (2.7-5.2)] than group II [0.8 (0.7-1.0)] and group III [0.8 (0.7-0.9)] was found with no statistically significant change between group II and group III. Additionally, the table indicates that there was a significant difference in the three groups under study with respect to the level of eGFR, with the highest level in group III and the lowest level in group I, with a p-value <0.001, and a statistically significant difference in the level of urea, with the highest level in group I and the lowest level in group III. The level of UA also showed significant difference between the three studied groups; the highest level was found in group I and lowest level was found in group II (Table 4).

Table 4 shows that, although there was no statistically significant difference between groups II and III, the level of A/CR was substantially greater in group I [82.75 (50.5 – 165)] than in groups II [26 (23 – 49)] and III [24.4 (20.3 – 28)] with a p-value <0.001. Additionally, the table demonstrates that group I had a significantly higher amount of 24-hour urine protein (mg) [335 (200–950)] than groups II and III [115 (80–160)] and 95 (80–110)], although there was no statistically significant difference between the

two groups.

Table 5 shows that among all diabetes mellitus patients (group I + group II); there was statistically significant negative correlation found between the level of OMT-1 level (pg/ml) and BMI, CRP, creat, urea, UA, A/C R and 24 hr urinary protein (mg) and also significant positive correlation between OMT-1 level (pg/ml) and hemoglobin and eGFR levels. OMT-1 level (pg/ml) and BMI and LDL levels were also shown to be statistically significantly correlated negatively in group I, however OMT-1 level (pg/ml) and BMI were found to be statistically correlated negatively in group II, although CRP level was positively correlated.

Table 6 shows a statistically significant contrast between the studied groups regarding ESR 1st hour which was found significantly higher in group I [35 (20-45)] than group II [21 (15-25)] and also significantly rised in group II than group III [11 (9-18)]. Also, the level of CRP showed significant changes between groups was found significantly higher in group I [62.5 (10-99)] than group II [12 (4-24)] and group III [8 (5-15)]; but with no statistically significant contrast between group II and group III.

Table 1. Shows the study demographic data and characteristics of the patients.

		TOTAL NO. PATIENTS = 60
GENDER	Female	30 (50.0%)
	Male	30 (50.0%)
AGE	Mean ± SD	62.12 ± 10.58
	Range	24 – 85
BMI	Mean ± SD	29.35 ± 4.78
	Range	19 – 44.5
DURATION OF DM (YRS)	Median (IQR)	10 (6 – 15)
	Range	0.17 – 35
SMOKING	No	44 (73.3%)
	Yes	16 (26.7%)
HTN	NO	29 (48.3%)
	YES	31 (51.7%)

Table 2. Shows a OMT-1 level comparison among the studied groups

OMT-1 (PG/ML)	GROUP I	GROUP II	GROUP III	ONE WAY ANOVA TEST	P-VALUE	SIG.
	No. = 30	No. = 30	No. = 30			
MEAN ± SD	24.99 ± 6.53	44.98 ± 7.97	79.17 ± 5.82	482.323•	<0.001	HS
RANGE	15.5 – 37.2	30.2 – 58	66.8 – 90			
POST HOC ANALYSIS						
GROUP I VS GROUP II		Group I Vs group III		GROUP II VS GROUP III		
<0.001		<0.001		<0.001		

P-value > 0.05: Non significant; P-value < 0.05: Significant; P-value < 0.01: Highly significant

Table 3. Shows blood glucose parameters comparison among the studied groups

BLOOD GLUCOSE		GROUP I	GROUP II	GROUP III	TEST VALUE	P-VALUE	SIG.
		No. = 30	No. = 30	No. = 30			
FBS	Median(IQR)	178.5 (140–210)	170.5(130–223)	86.5 (77–96)	43.778 ≠	0.000	HS
	Range	70 – 370	73 – 380	70 – 102			
PP	Median(IQR)	230 (170 – 277)	228.5(145–\289)	118.5 (95–133)	34.545 ≠	0.000	HS
	Range	84 – 404	89 – 407	77 – 141			
HBA1C	Mean ± SD	7.30 ± 1.77	7.52 ± 2.01	4.88 ± 0.46	26.212 •	0.000	HS
	Range	4.8 – 12.3	4.7 – 11.9	4 – 5.7			
POST HOC ANALYSIS							
		Group I Vs group II		Group I Vs group III		GROUP II VS GROUP III	
FBS		0.865		<0.001		<0.001	
PP		0.953		<0.001		<0.001	
HBA1C		0.583		<0.001		<0.001	

•: One Way ANOVA test; ≠: Kruskal-Wallis test

Table 4. Shows a comparison between the studied groups regarding kidney function tests, A/CR and 24 hr urinary protein (mg)

KIDNEY FUNCTIONS		GROUP I	GROUP II	GROUP III	TEST VALUE	P-VALUE	SIG.
		No. = 30	No. = 30	No. = 30			
CREAT	Median(IQR)	4.2 (2.7 – 5.2)	0.8 (0.7 – 1)	0.8 (0.7 – 0.9)	60.672≠	0.000	HS
	Range	1.6 – 8.2	0.6 – 1.2	0.6 – 1			
UREA	Median(IQR)	104 (90 – 147)	33.5 (29 – 39)	25 (23 – 32)	65.048≠	0.000	HS
	Range	60 – 254	21 – 69	15 – 32			
EGFR	Median(IQR)	15.55(10.7–24.5)	90.15(79–102)	102.55(93.4–115.6)	62.478≠	0.000	HS
	Range	6.3 – 47	58.7 – 125	57.6 – 156			
UA	Mean ± SD	8.86 ± 1.65	5.04 ± 1.56	6.02 ± 1.22	53.300•	0.000	HS
	Range	6 – 11.5	2.6 – 9.6	3.5 – 8.2			
A/C R	Median(IQR)	82.75(50.5–165)	26 (23 – 49)	24.4 (20.3 – 28)	45.	0.	H

	Range	28 – 887.5	16 – 240	15.9 – 30	997≠	000	S
24 HR URINARY PTN (MG)	Median(IQR)	335 (200–950)	115 (80 – 160)	95 (80 – 110)			H
	Range	95 – 2654	30 – 1200	50 – 160	41. 355≠	0. 000	S

POST HOC ANALYSIS

	Group I Vs group II	Group I Vs group III	GROUP II VS GROUP III
CREAT	<0.001	<0.001	0.282
UREA	<0.001	<0.001	<0.001
EGFR	<0.001	<0.001	0.008
UA	<0.001	<0.001	0.013
A/C R	<0.001	<0.001	0.064
24 HR URINARY PTN (MG)	<0.001	<0.001	0.092

•: One Way ANOVA test; ≠: Kruskal-Wallis test

Table 5. Shows correlation between OMT-1 level (pg/ml) and the other studied parameters among all DM II patients, group I and group II.

	OMT-1 (PG/ML)					
	All DM patients		Group I		GROUP II	
	r	P-value	r	P-value	r	P-VALUE
AGE	-0.050	0.703	0.125	0.510	0.298	0.110
BMI	-0.463**	0.000	-0.834**	0.000	-0.979**	0.000
DURATION OF DM	-0.110	0.405	0.035	0.854	-0.063	0.739
TLC	-0.252	0.052	-0.132	0.487	-0.043	0.822
HB	0.467**	0.000	-0.028	0.881	-0.245	0.192
PLATELETS	-0.141	0.282	-0.147	0.439	-0.199	0.291
ESR 1ST H	-0.144	0.271	0.184	0.331	0.284	0.129
ESR 2ND H	-0.185	0.157	0.018	0.926	0.175	0.356
CRP	-0.300*	0.020	-0.110	0.564	0.390*	0.033
FBS	-0.114	0.388	-0.286	0.126	-0.041	0.831
PP	-0.056	0.671	-0.205	0.277	-0.056	0.769
HBA1C	-0.005	0.973	0.072	0.704	-0.182	0.335
CREAT	-0.709**	0.000	-0.128	0.500	0.153	0.419
UREA	-0.659**	0.000	0.262	0.162	0.121	0.523
EGFR	0.681**	0.000	-0.016	0.931	-0.200	0.289
UA	-0.679**	0.000	-0.071	0.708	-0.122	0.522
NA	0.105	0.427	0.107	0.573	0.181	0.339
K	0.044	0.741	0.078	0.683	-0.032	0.865
A/C R	-0.498**	0.000	-0.158	0.404	0.201	0.287
24 HR URINARY PTN (MG)	-0.520**	0.000	-0.050	0.792	-0.044	0.819
CHOLESTEROL	0.195	0.135	-0.002	0.991	-0.159	0.400
TGS	0.211	0.105	0.180	0.341	-0.324	0.081
HDL	0.228	0.080	0.015	0.937	0.098	0.607
LDL	0.009	0.945	-0.449*	0.013	0.027	0.887

Spearman correlation coefficient.

Table 6. ESR (1st hour) and CRP levels comparison among the studied groups regarding ESR (1st hour) and CRP levels

		GROUP I	GROUP II	GROUP III	KRUSKAL-WALLIS TEST	P-VALUE	SIG.
		No. = 30	No. = 30	No. = 30			
ESR 1ST H	Median (IQR) Range	35(20 – 45) 10 – 70	21(15 – 25) 10 – 66	11(9 – 18) 7 – 29	29.178	<0.001	HS
CRP	Median (IQR) Range	62.5(10 – 99) 2.3 – 293	12(4 – 27) 2.3 – 132	8(5 – 15) 2.5 – 20	19.796	<0.001	HS

POST HOC ANALYSIS

	Group I Vs group II	Group I Vs group III	GROUP II VS GROUP III
ESR 1ST H	0.021	<0.001	<0.001
CRP	0.002	<0.001	0.138

4. Discussion

This work showed a difference between the studied groups in terms of the level of OMT-1 (pg/ml). The level was found significantly lower in group I (24.99±6.53) than in group II (44.98±7.97), which was lower than group III (79.17±5.82), with a p-value <0.001.

This result supported the findings of Latif et al.[12], who noted that plasma omentin-1 levels were significantly lower in type II diabetic patients than in the control group. Devi et al.[13] also discovered that, in contrast to the DM type II group without DN, Omentin 1 levels were lower in the DN group. Also, according to Tekce et al.[14], CKD patients' plasma Omentin-1 levels were considerably lower than those of healthy controls.

Although Hayashi et al.[15]; disagreed with our result as he found serum Omentin-1 levels were higher in type-II diabetic patients than in non-diabetic controls.

Based on our findings, the ESR 1st hour differences across the study groups were statistically significant. Group I had a significantly higher ESR [35 (20-45)] than group II [21 (15-25)], and group II had a significantly higher ESR than group III [11 (9-18)] with a p-value <0.001. Additionally, there was a significant difference in the CRP levels between the three groups, with group I having a significantly higher level [62.5 (10-99)] than group II [12 (4-24)] and group III [8 (5-15)] (p-value <0.001).

These findings concurred with those of Zhang et al.[16], who found that DKD patients had significantly higher ESR and CRP values than patients with DM type II who did not have DKD. A study by Fang et al.[17], found that CRP is increased in patients with urinary albuminuria. Additionally, a meta-analysis by Stanimirovic et al.[18] verified that DM type II patients with DN had higher CRP levels than both healthy subjects and DM type II patients without DN.

According to our findings, there was a p-value <0.001 difference in the uric acid (UA) levels of the three groups under investigation; group I had the highest level, and group II had the lowest.

According to Bombelli et al.[19], a significant rise in uric acid was linked to a higher risk of impaired fasting glucose. Individuals with higher median uric acid levels may also develop metabolic syndrome and diabetes. However, Haque et al.[20] discovered that the serum uric acid level was lower in prediabetic and diabetic individuals than in healthy individuals.

According to Lin et al.[21], there was no correlation between elevated uric acid levels and a higher risk of type II diabetes. This study was conducted on a larger sample than our work.

The present work showed that the level of albumin/ creatinine ratio (A/CR) was significantly higher in group I [82.75 (50.5 – 165)] than in group II [26 (23 – 49)] and group III [24.4 (20.3 – 28)] with p-value <0.001.

The present work showed that the level of albumin/ creatinine ratio (A/CR) was significantly higher in group I [82.75 (50.5 – 165)] than in group II [26 (23 – 49)] and group III [24.4 (20.3 – 28)] with p-value <0.001. Our findings also revealed that group I had a significantly higher level of 24-hour urine protein (mg) [335 (200–950)] than groups II and III [115 (80–160)] and 95 (80–110)] with a p-value <0.001.

The findings of Yao et al.[22] corroborated our findings. They found that DM type II is linked to the development of kidney disease and albuminuria. A study by Andruszko et al.[23], found that albuminuria is highly associated with diabetes complications, especially diabetic nephropathy.

Our work found a statistically significant negative correlation between BMI and OMT-1 level (pg/ml).

Arab et al.[24] described that plasma omentin concentration is significantly lower in overweight subjects. Lis et al.[25] agreed with our results about the negative correlation between the level of OMT-1 level (pg/ml) and BMI.

Our findings demonstrated a negative relationship between Omentin-1 levels and CRP.

Karampela et al. [26] studied the relationship between some inflammatory markers and Omentin and agreed with our results that there was a negative correlation between the level of OMT-1 and CRP. Can Sandikci et al.[27] studied the relationship between Omentin-1 and acute phase reactants and found negative correlations between CRP and ESR and the level of Omentin. These results suggest the anti-inflammatory effect of Omentin-1.

Chaudhari et al.[28], however, did not discover any meaningful association between omentin-1 and CRP.

According to the current findings, OMT-1 level and eGFR were positively correlated, while urea and creatinine, and OMT-1 levels were negatively correlated.

Our results were similar to those of Chaudhari et al.[28] regarding the correlation between kidney functions and Omentin level. They found that omentin-1 was significantly lower than in controls. And found that omentin-1 had a notable positive correlation with eGFR. This finding clarifies that a significant correlation was found between Omentin and renal damage. In their investigation to assess serum Omentin in hypertension patients, Çelik et al.[29] concurred with our findings and discovered that OMT-1 decreases as renal function increases.

4. Conclusion

Omentin-1 is significantly lower in type II diabetic patients with DN than in type II diabetic patients without DN, which is lower than that of healthy controls.

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References

1. R Tomic D, Shaw JE, Magliano DJ. The burden and risks of emerging complications of diabetes mellitus. Nat Rev Endocrinol. 2022;18(9):525–39.
2. Samsu N. Diabetic Nephropathy: Challenges in Pathogenesis, Diagnosis, and Treatment. Bellini MI, editor. BioMed Res Int. 2021; 2021:1–17.
3. García-Carro C, Vergara A, Bermejo S, Azancot MA, Sánchez-Fructuoso AI, Sánchez de la Nieta MD, et al. How to Assess Diabetic Kidney Disease Progression? From Albuminuria to GFR. J Clin Med. 2021; 10(11):2505.
4. DeFronzo RA, Reeves WB, Awad AS. Pathophysiology of diabetic kidney disease: impact of SGLT2 inhibitors. Nat Rev Nephrol. 2021; 17(5):319–34.
5. Pelle MC, Provenzano M, Busutti M, Porcu CV, Zaffina I, Stanga L, et al. Up-Date on Diabetic Nephropathy. Life. 2022; 12(8):1202.
6. Xu J, Li M, Jiang X, Wang Y, Ma H, Zhou Y, et al. Omentin-1 and diabetes: more evidence but far from enough. Arch Physiol Biochem. 2023;0(0):1–7.
7. Chait A, den Hartigh LJ. Adipose Tissue Distribution, Inflammation and Its Metabolic Consequences, Including Diabetes and Cardiovascular Disease. Front Cardiovasc Med. 2020; 7:22.
8. Luo J, He Z, Li Q, Lv M, Cai Y, Ke W, et al. Adipokines in atherosclerosis: unraveling complex roles. Front Cardiovasc Med [Internet]. 2023 [cited 2023 Nov 4];10. Available from: https://www.frontiersin.org/articles/10.3389/fcvm.2023.1235953
9. Dong Q, Xing W, Li K, Zhou X, Wang S, Zhang H. Tetrahydroxystilbene glycoside improves endothelial dysfunction and hypertension in obese rats: The role of omentin-1. Biochem Pharmacol. 2021; 186:114489.
10. Atukeren P, Uzun H. Biomarkers in Medicine. Bentham Science Publishers, 2022.
11. Yan P, Xu Y, Zhang Z, Zhu J, Miao Y, Gao C, Wan Q. Association of Circulating Omentin-1 with Osteoporosis in a Chinese Type 2 Diabetic Population. Mediators of inflammation. 2020;2020(1):9389720.
12. Latif AH, Anwar S, Gautham KS, Kadurei F, Ojo RO, Hafizyar F, Haroon DM, Rakesh FN, Talpur AS. Association of plasma omentin-1 levels with diabetes and its complications. Cureus. 2021 Sep;13(9).
13. Devi RV, Subramaniam V, Adole PS, Senthilkumar GP, Mehalingam V. Cardiovascular autonomic neuropathy in patients with type 2 diabetes mellitus and its association with serum omentin and leptin. Journal of family medicine and primary care. 2020; 9(6):2926-30.
14. Tekce H, Tekçe BK, Aktas G, Alçelik A, Sengul E. Serum omentin-1 levels in diabetic and nondiabetic patients with chronic kidney disease. Experiment Clin Endocrinol Diabetes. 2014; 11:451-6.
15. Hayashi M, Morioka T, Hatamori M, Kakutani Y, Yamazaki Y, Kurajoh M, et al. Plasma omentin levels are associated with vascular endothelial function in patients with type 2 diabetes at elevated cardiovascular risk. Diabetes Res Clin Practice 2019; 148:160-8.
16. Zhang GH, Ye ZH, Guan HJ, Guo M, Zhou XX, Xu YY. Impact of serum omentin-1 concentrations on functional outcome among acute intracerebral hemorrhage patients. Clinica Chimica Acta. 2020; 503:169-74.
17. Fang Y, Wang B, Pang B, Zhou Z, Xing Y, Pang P, et al. Exploring the relations of NLR, hsCRP and MCP-1 with type 2 diabetic kidney disease: a cross-sectional study. Sci Rep. 2024; 14(1):3211.
18. Stanimirovic J, Radovanovic J, Banjac K, Obradovic M, Essack M, Zafirovic S, et al. Role of C-Reactive Protein in Diabetic Inflammation. Mediators Inflamm. 2022; 2022:3706508.
19. Bombelli M, Quarti-Trevano F, Tadic M, Facchetti R, Cuspidi C, Mancia G, et al. Uric acid and risk of new-onset metabolic syndrome, impaired fasting glucose and diabetes mellitus in a general Italian population: data from the Pressioni Arteriose Monitorate E Loro Associazioni study. J Hypertens. 2018; 36(7):1492-8.
20. Haque T, Rahman S, Islam S, Molla NH, Ali N. Assessment of the relationship between serum uric acid and glucose levels in healthy, prediabetic and diabetic individuals. Diabetol Metab Syndrome. 2019; 11:1-8.
21. Lin S, Li X, Zhang J, Zhang Y. Omentin-1: Protective impact on ischemic stroke via ameliorating atherosclerosis. Clinica Chimica Acta. 2021; 517:31-40.
22. Yao C, Ou J, Zhou L, Liu J. Correlation of serum omentin-1 level with clinical features and major adverse cardiac and cerebral events in patients undergoing continuous ambulatory peritoneal dialysis. Scandinavian J Clin Lab Invest. 2023; 83(2):119-24.
23. Andruszko A, Szydłowski J, Grabarek BO, Mazur K, Sirek T, Ossowski P, et al. Impact of Nutritional Status of Patients with Head and Neck Squamous Cell Carcinoma on the Expression Profile of Ghrelin, Irisin, and Titin. Cancers. 2024; 16(2):437.
24. Arab A, Moosavian SP, Hadi A, Karimi E, Nasirian M. The association between serum omentin level and bodyweight: A systematic review and meta-analysis of observational studies. Clin Nutr ESPEN. 2020; 39:22-9.
25. Lis I, Pelczyńska M, Miller-Kasprzak E, Kujawska-Łuczak M, Mądry E, Bogdański P. Association of serum omentin concentration with anthropometric, physiological, and biochemical parameters in obese individuals. Nutrition. 2020; 79:110866.
26. Karampela I, Vallianou NG, Tsilingiris D, Christodoulatos GS, Antonakos G, Marinou I, et al. Diagnostic and Prognostic Value of Serum Omentin-1 in Sepsis: A Prospective Study in Critically Ill Patients. Medicina; 59(5):833.
27. Can Sandikci S, Omma A, Yucel C, Omma T. Is there a relationship between serum omentin level and acute phase response in patients with familial Mediterranean fever?. Clin Rheumatol. 2021; 40:669-74.
28. Chaudhari MJ, Vasanthan MV, Vinodhini VV, Ranen PR, Roy B, Choudhary SA, et al. Omentin-1: A Potential Biomarker and therapeutic agent for Diabetic Chronic Kidney Disease. 2024, PREPRINT (Version 1) available at Research Square [https://doi.org/10.21203/rs.3.rs-3838879/v1]
29. Çelik M, Nar R, Nar G, Sökmen E, Günver G. Serum omentin-1 levels in hypertensive patients. J Hum Hypertens. 2021; 35(3):290-5.