Hilla University College Journal For Medical Science

Manuscript 1117

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HILLA UNIV COLL J MED SCI 2026;4:74–80

ORIGINAL STUDY

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Malondialdehyde and Ceruloplasmin: A More Sensitive and Specific Marker than Other Obesity Markers for Diagnosis of Obese Individuals

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Abstract

Background: Higher levels of lipid peroxidation are associated with obesity. Malondialdehyde (MDA) is a byproduct of this process and acts as a biomarker. Plasma ceruloplasmin (Cp) is also connected to obesity and is considered a risk factor for cardiovascular disease.

Objectives: This study aimed to measure MDA and CP levels in obese people and compare them with levels in people of normal weight, including both men and women.

Methods: Case and control study was conducted on a total (90) participants, consisting of (45) Normal individuals with BMI <25 Kg/m2, (45) Obese individuals with BMI ≥30 Kg/m2 were enrolled in the study. Serum MDA and CP were estimated using the ELISA technique. The present study was carried out from 8th January 2024, to 1st May 2024, at Rizgary Hospital, Cardiac Center, and Cihan University.

Results: Malondialdehyde (MDA) was assessed in both groups. Sensitivity and specificity for MDA were 95.1(0.95) with significance ($p < 0.0001$), for CP 93.33(0.93) with significance ($p < 0.0001$), significant increase in median of MDA from 287.0 ($p < 0.0001$), median CP from 230.1 ($p < 0.0001$) in obese subjects. A positive Spearman's correlation between MDA and BMI ($P < 0.0001$) and between CP with BMI ($P < 0.0001$), indicating that it is an excellent biomarker for diagnosing obesity.

Conclusion: This finding suggests that obesity is an important factor for enhanced MDA oxidative stress and Cp protein, with the highest sensitivity and specificity, in obese individuals. which may be suggested as a marker of obesity.

Keywords: Ceruloplasmin, Malondialdehyde, Obesity, Oxidative stress

1. Introduction

Obesity is a chronic condition marked by excessive adiposity that compromises health. It increases the risk of various metabolic complications, including type 2 diabetes, cardiovascular disease, metabolic dysfunction-associated steatotic liver disease, previously known as non-alcoholic fatty liver disease, and various mechanical consequences, such as osteoarthritis and obstructive sleep apnea [1]. Body mass index (BMI) ≥30 kg/m2 is used for the diagnosis of obesity. Obesity reduces life quality and has a negative economic impact on nations across the world [2]. Reactive oxygen species, or ROS, have been linked to oxidative stress, which occurs in physiological settings and affects many organs [3]. It is associated with several pathogenic mechanisms, including atherogenic processes, diabetes, obesity, and cardiovascular disease [4].

The buildup of lipid oxidation products, such as lipid hydroperoxides and MDA, may increase as a result of the distinction between oxidant and antioxidant pathways. These substances are toxic and raise blood pressure through other lipoproteins that induce arteriosclerosis [5]. Lipid peroxidation also produces malondialdehyde (MDA), which has been investigated as a marker of oxidative stress. Polyunsaturated

Received 10 January 2026; revised 26 April 2026; accepted 28 April 2026.
Available online 20 June 2026

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https://doi.org/10.62445/2958-4515.1117
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fatty acid (PUFA) oxidation produces MDA, which is a useful indicator for identifying peroxidation processes [6]. The liver produces Cp, a 132 kDa α-2-glycoprotein that contains copper. Copper transport, iron metabolism, angiogenesis, and coagulation all depend on Cp, which also functions as an antioxidant or pro-oxidant [7]. Plasma Cp level is positively correlated with obesity and has been suggested as a biomarker for obesity [8]. The objective of the current study was to assess the level of malondialdehyde (MDA) and (CP) and to find out the sensitivity and specificity of MDA and Cp in obese individuals compared with normal individuals.

2. Materials and methods

A study of a case and control. There were 90 patients enrolled. This study's participants were divided into two groups. Group I, or the control group, comprised 45 people with a BMI below 25 kg/m^2. Group II: This group comprised 45 obese people with a BMI of $\geq$30 kg/m^2. (BMI) was measured for all samples by dividing weight kg by height squared m^2.

2.1. Blood sample collection

Ninety samples were collected from forty-five lean and forty-five obese people. The samples were centrifuged at 3500 rpm for 10 minutes. Serum TC, TG, HDL, LDL, and VLDL were quantified using the COBAS technology, while the isolated MDA and CP sera were evaluated using the ELISA approach. Serum tests were stored at –70°C until assessment, whereas kits should be maintained at 2–8°C until testing.

2.2. Requirements for inclusion and exclusion

The criteria for inclusion were: female and male sexes, individuals aged 30–80, obesity, normal weight heart disease, hypertensive, and diabetes. Exclusion criteria included: patients who were underweight, younger than thirty, utilizing hormone or antioxidant supplements, endocrine disorders, cancer, chronic kidney disease, pregnancy, dieting, and hemolyzed blood samples.

2.3. Study timeline

The current study was conducted at Rizgary Hospital, Cardiac Center, and Cihan University in Erbil, Iraq, between January 8, 2024, and May 1, 2024.

2.4. Questionnaire form design

The study employed a pretested face-to-face questionnaire with implemented access changes. Furthermore, sensitive personal information that is personally identifiable and not publicly accessible was utilized in medical records. The questionnaire comprises inquiries about the patient's clinical risk factors, familial history, habitual smoking, and personal details (name, age, gender, residence, time, and date).

2.5. Biochemical assays

Serum MDA and CP were measured by the ELISA technique. The assay was carried out in line with the manufacturer's instructions. Centrifugation was used to separate the serums for 10 minutes at 3500 rpm. MDA (0.29–100 nmol/mL), and CP (20–40 mg/dL).

2.6. Statistical analysis

The statistical software utilized to analyze the data in this study was GraphPad Prism (version 10). MedCalc is a statistical software package for the biomedical sciences. The normality of the data was determined using the Shapiro-Wilk test. Non-parametric tests were employed for data that was not distributed normally. The data were represented as the mean and standard error of the mean (Mean $\pm$ SE). The median rankings in two groups were compared using the Mann-Whitney test. The median rankings of three groups were compared using one-way ANOVA and the Kruskal-Wallis test. The chi-square test was used to check for categorical characteristics between the groups, while Spearman's rank correlation was used to determine correlations. Data was compared by analyzing the difference in mean levels between the two groups through this method. There has been established statistical significance with the p-value level ($p < 0.05$) of significance. The area under the curve (AUC) for diagnostic efficiency in Obese was established as an assessed median. Calculations were done with the ROC curve (Receiver Operating Characteristic) analysis.

3. Results

Ninety sample included in this study. They were separated into two subgroups, where Group 1 had 45 obesities. The study results highlighted a substantial difference in the mean age of (48.33 $\pm$ 1.44), which was about (50%) of the study population. Also, (53.33% male and 46.67% female), and Group 2 consisted of 45 healthy individuals with a mean age of (37.82 $\pm$ 1.13), (84.44% male and 15.56% female) (p-value = 0.002). While the obese involvement levels of BMI were higher means compared to the healthy group ($P < 0.0001$), there was a significant variation ($p < 0.05$) between the two groups for SBP, DBP, and FBS, but non-Signiant for pulse rate ($p > 0.05$) (Table 1).

Table 1. Basic characteristics.

Variables	Obese, N (45) Mean ± SE	Healthy, N (45) Mean ± SE	p-value
Gender N (%)			0.0024
Meal	24(53.33%)	38(84.44%)	
Female	21(46.67%)	7(15.56%)	
Age (years)	48.33 ± 1.44	37.82 ± 1.13	<0.0001
BMI$(Kg/m)^2$	37.70 ± 0.74	22.91 ± 0.25	<0.0001
Systolic BP	12.33 ± 0.2524	13.83 ± 0.2735	0.0001
Diastolic BP	8.687 ± 0.1889	7.938 ± 0.1652	0.0037
Pulse rate	81.44 ± 1.768	80.76 ± 1.987	0.7962
FBS	148.8 ± 8.482	110.8 ± 4.382	0.0001

*By t test., *By Chi-square test.

According to the Chi-square test, all risk factors showed significant differences ($p < 0.05$) between the obese and healthy group (Table 2).

Table 2. Risk factor for obesity.

Risk factors	Obese (n = 45) No. (%)	Healthy (n = 45) No. (%)	p-value
Genetic disease			<0.0001
• Positive	36 (70.59%)	15 (29.41%)	
• Negative	9 (23.08%)	30 (76.92%)	
Smoking			
• Ex-smoker	8 (27.59%)	21 (72.41%)	0.0055
• Non-smoker	37 (58.73%)	26 (41.27%)	
Physical activity			
• Active	32 (43.84%)	41 (56.16%)	
• Non-active	13 (72.22%)	4 (23.53%)	0.0310
Hypertension			0.0002
• Yes	14 (93.33%)	1 (6.67%)	
• No	31 (41.33%)	44 (58.67%)	
Diabetes			<0.0001
Yes	16 (94.12%)	1 (5.88%)	
No	29 (39.73%)	44 (60.27%)	

*By Chi-square test.

In the obese group, there was a significant increase in the median CP from 230.1 (p value < 0.0001), there was a decrease in the median CP in the healthy group. Similarly, in the obese group, there was a significant increase in the median of MD from 287.0 (p value < 0.0001) and a higher decrease in the healthy group, 125.0, (Table 3).

Table 3. Medians of biomarkers between obese and healthy.

	Obese		Healthy		
Biomarker	Median	Range	Median	Range	p-value
CP	231.0	47	140.0	59.5	<0.0001
MD	287.0	117.5	125.0	39.5	<0.0001

Data are mean ±SE and median. *By nonpaired t test., *By Mann-Whitney test.

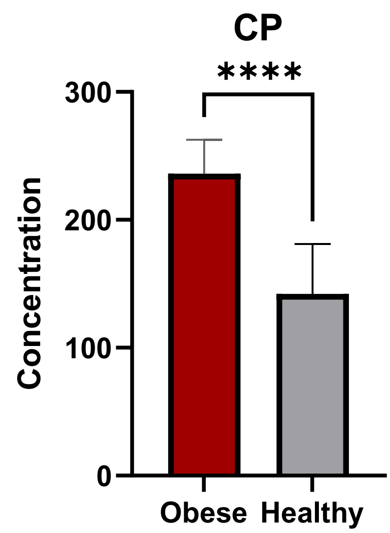

Fig. 1. CP levels between healthy and obese groups.

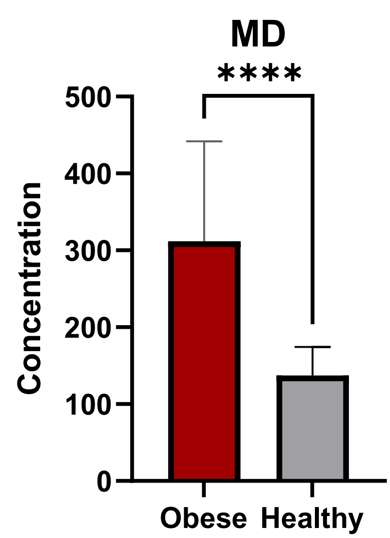

Fig. 2. MD levels between healthy and obese groups.

Table 4. Correlation between CP and MDA with BMI in the studied groups.

GP-BB	Spearman's rank correlation	BMI
CP	r	0.7492
	P value	<0.0001
MDA	r	0.6470
	P value	<0.0001

*By correlation test.

Studied groups have a positive Spearman's correlation between CP and BMI (P-value < 0.0001) as shown. A similar result was studied in a group showing a positive Spearman's correlation between MDA and BMI (P-value < 0.0001) as shown in (Table 4) and (Figs. 3 and 4). Using the (Kruskal-Wallis) test indicated that there was no association between CP and MD with

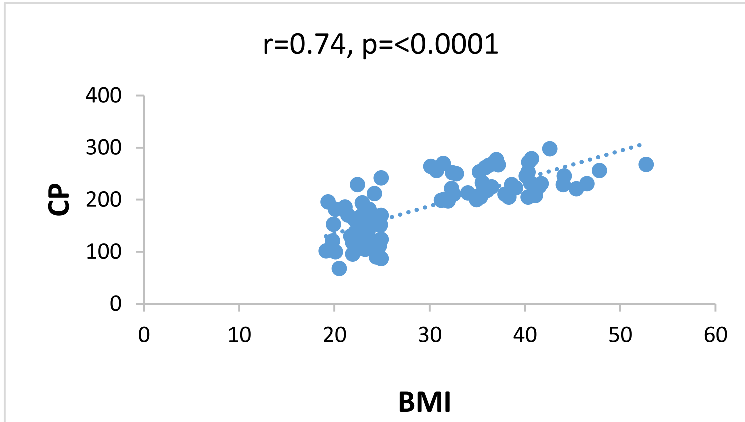

Fig. 3. Correlation between CP and BMI in the studied group.

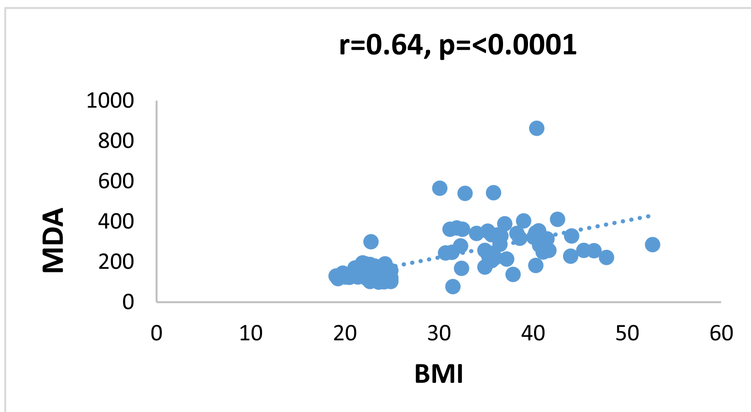

Fig. 4. Correlation between MDA and BMI in the studied group.

Table 5. Association CP and MDA of obesity.

	Obese I, N (13)		Obese II, N (16)		Obese III, N (16)			
Parameters	Median	Range	Median	Range	Median	Range	K test value	p-value
CP	213.0	53.5	228.5	50	239.0	38.2	3.892	0.1428
MDA	279.0	157	323.5	126.7	284.5	89.5	0.00250	0.9987

*By One way ANOVA, *By Kruskal-Walli's test.

subclass obesity (I, II, III), with p-value (0.142, and 0.998) respectively, as shown in (Table 5).

The Area Under the Curve Analysis (ROC) is the preferred method for evaluating the diagnostic accuracy of the CP and MDA tests. The AUC values for serum CP and MDA are both 93.33(0.93) and 95.1(0.95), respectively, with significant p-values of less than 0.0001, indicating that they are excellent biomarkers for diagnosing obesity (Table 6).

4. Discussion

A global health concern, obesity is thought to be the primary risk factor for illness and death [9]. Numerous biochemical alterations are brought about by oxidative stress, which is characterized as an excess of oxidants over antioxidants. These alterations are significant pathogenic mediators in a broad range of human diseases. Elevated blood levels of MDA have been linked to the excessive generation of ROS. MDA is a typical measure of oxidative state and is the principal end product of lipid peroxidation by ROS [10]. Endothelial cell and β-cell dysfunction are frequently caused by oxidative stress, which is a pathogenic factor. A detrimental component that seems to contribute to insulin resistance, β-cell malfunction, poor glucose tolerance, and eventually type-2 diabetes is increased oxidative stress. The connection between systemic oxidative stress and each of these disorders may involve obesity [11].

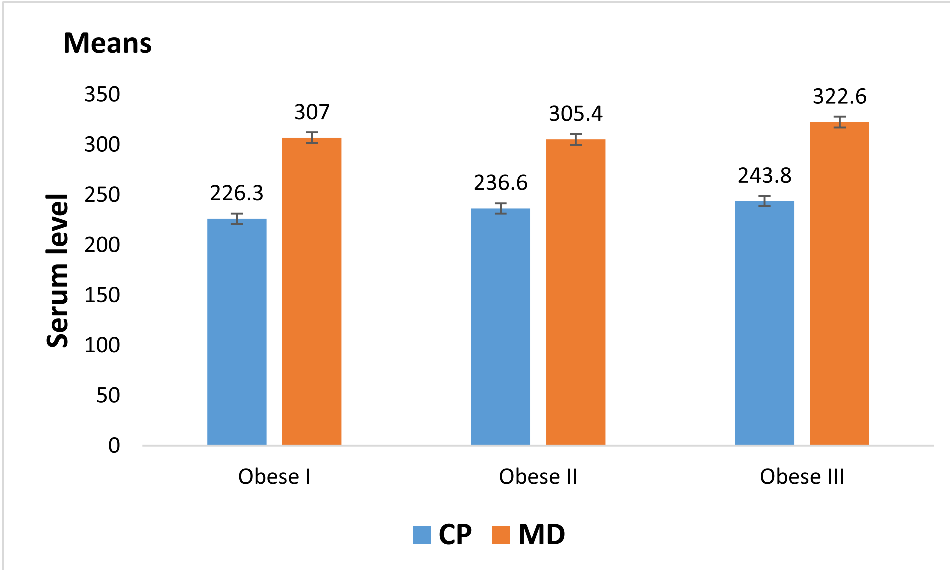

Fig. 5. The means of CP and MDA in subclass obesity.

Table 6. The sensitivity, specificity, PPV and NPV of obese biomarkers.

Biomedical Parameters	Cut off	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)	Accuracy	AUC	p-value
CP	>196	100.00	93.33	93.7	100.0	93.33	97.1	<0.0001
MDA	>195	88.89	97.78	97.6	89.8	86.67	95.1	<0.0001

*By MedCalc statistical software., *By Roc curve analysis.

Lipid peroxidation is the most harmful effect of oxidative stress and has been linked to the etiology of many illnesses, including diabetes, cancer, atherosclerosis, and aging. Polyunsaturated fatty acids undergo oxidative degradation due to a process known as lipid peroxidation, which is triggered by the addition or abstraction of hydrogen by oxygen radicals [12]. The MDA serum test is the most often used approach in clinical practice because of its sensitivity and simplicity. However, various drugs interact with this assay [13]. This study demonstrates a significant increase in the median of MD from 287.0 ($p < 0.0001$) in obese subjects, (Table 3 & Fig. 2). A positive Spearman's correlation between MDA and BMI ($P < 0.0001$) (Table 4 & Fig. 4). Matés et al. represent lipid oxidative damage in vivo. Obesity has also been considered an independent risk factor for plasma lipid peroxidation.

These findings are consistent with reports of a significant rise in MDA levels, a measure of oxidative stress, in obese persons in comparison to non-obese healthy controls [14]. The study's findings are consistent with the research conducted by Vehkala et al. Obesity and type 2 diabetes (T2D) have been linked to elevated oxidative stress. It has been proposed that one of the antigenic epitopes in MDA-LDL accountable for antibody recognition is malondialdehyde acetaldehyde (MAA) adducts [15]. Our results are consistent with a recent study by Adnan et al., which suggested that a possible causal element for the pathophysiology of obesity might be a rise in MDA with a decreased level of antioxidants [16]. Additionally, Monserrat-Mesquida et al. obese people had greater plasma levels of MDA than normal-weight and overweight participants, according to research by Monserrat-Mesquida et al.

Lipid peroxidation's primary byproduct and a commonly used indicator of oxidative damage is MDA. MDA levels can be lowered with weight loss, and they were higher in obese individuals [17]. Cota-Magana et al. observed that MDA is the most widely utilized biomarker for determining lipid peroxidation levels in biological fluids. In this recent study, the researchers assessed it in serum samples since obesity causes increased triglyceride synthesis and reduced serum clearance. This suggests that fatty acids associated with triglycerides are more prone to oxidation. Metabolic markers related to obesity were examined in serum since they were thoroughly studied and linked to the multiple molecular mechanisms involved in the pathophysiology of obesity. [18]. Cp is an α-2 glycoprotein that has seven copper atoms per molecule and makes up about 95% of the copper that is circulating in healthy humans. It contributes to iron homeostasis, coagulation, angiogenesis, and defense against oxidative stress. Serum Cp concentrations have been shown to be a potential independent risk factor for CVD in a number of prospective studies. Diabetes mellitus, obesity, dyslipidemia, hypertension, and other risk factors for CVD are linked to a low-grade inflammation that may be connected to elevated blood Cp concentrations [19]. This study reported a significant increase in median CP from 230.1 ($p < 0.0001$) (Table 3 & Fig. 1). A positive Spearman's correlation between CP and BMI ($P < 0.0001$) was shown in (Table 4 & Fig. 3).

Our study is consistent with studies that have been done by Rajappa et al. hypothesized that the substantial correlation shown between raised plasma Cp levels and higher BMI suggests that ceruloplasmin (Cp) might be used as a biomarker of obesity. Rajappa et al. showed that the predominant cuproprotein in blood is called Cp, and fat individuals have higher Cp levels [20]. Yang et al. and Engström reported that Obese people's elevated ceruloplasmin levels may be a response to oxidative stress [21, 22]. Few research have looked at the relationship between serum MDA and Cp in obesity. (ROC) analysis showed the highest sensitivity and specificity for MDA. The AUC values for serum MDA 95.1(0.95) with significant ($p < 0.0001$), indicating that it is an excellent biomarker for diagnosing obesity (Table 6) and (Fig. 7). Lee et al. and Singh et al. concluded that MDA had a most sensitive and specific in obesity [23, 24]. This is correlated with our results. While Wang et al. and Muhsin et al. showed that (ROC) analysis observed the highest sensitivity and specificity for CP [25, 26]. The AUC values for serum CP were 93.33 (0.93) and were significant ($p < 0.0001$), which is an excellent biomarker for diagnosing obesity in obese persons (Table 6) and

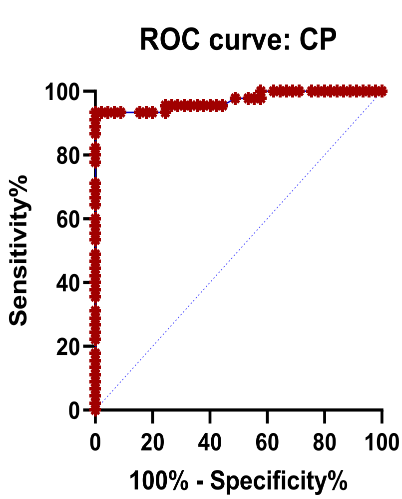

Fig. 6. CP (ROC) curve of obesity.

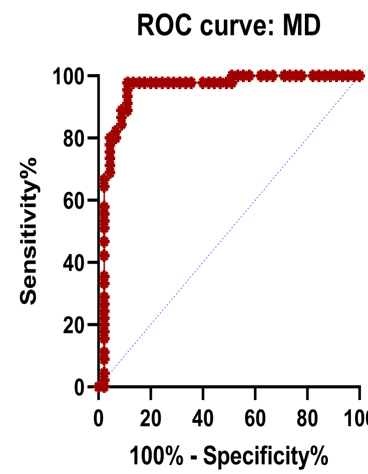

Fig. 7. MDA (ROC) curve of obesity

(Fig. 6). Hassan et al. and Khudhair et al. reported that CP had the most sensitive and specificity in obesity [27, 28].

5. Conclusion

According to the current study's data, obese individuals had significantly higher levels of oxidative stress and protein parameters, such as ceruloplasmin and malondialdehyde, which are the most sensitive and specific indicators of an increased risk of obesity and may be used as a predictor of obesity.

Acknowledgement

The author would like to thank all individuals who supported this work.

Ethical approval

The research procedures were reviewed and approved by the scientific committee of Cihan University's College of Dentistry, as well as the ethical committee of Erbil's General Directorate of Health. The participants in this study also provided their permission. In addition to promises about data security and confidentiality. The purpose and scope of the study were fully explained to each participant. Cihan University-Erbil, Kurdistan Region, IRAQ Scientific Research Ethics Committee, Certification of Research Ethics Committee Approval, Certificate Number: (CUE-REC/2025/07), Approval Date: (2025)

Abbreviations and notation

MASLD: metabolic-dysfunction associated steatotic liver disease, T2D: type 2 diabetes, CP: Ceruloplasmin, MDA: malondialdehyde, OSA:

obstructive sleep apnea, BMI: Body mass index, TC: Total Cholesterol, TG: Triglyceride, HDL: High density lipoprotein, LDL: Low density lipoprotein, VLDL: Very low density lipoprotein, CVD: cardiovascular disease.

Conflicts of interest

The author declares no conflict of interest.

Funding

None.

Author contributions

Sawen Tahseen Taha contributed to the conceptualization, study design, data collection, data analysis, and interpretation of results. The author also wrote the original draft, revised the manuscript critically, and approved the final version for publication.

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