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Relationship Between Obesity, Oxidative Stress, and Endothelial Function in Iraqi Young Adults: A Cross-Sectional Study at Al Sadr Teaching Hospital, Najaf

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Abstract

Background: Obesity among young Iraqis is increasing drastically, and it has been recognized as a chronic state of oxidative stress that may predispose individuals to cardio vascular diseases. Oxidative stress reduces nitric oxide bioavailability, which may be a mechanism by which obesity leads to endothelial dysfunction. However, studies involving young Iraqis have been limited.

Aim of study: The purpose of the study was to investigate the correlation between obesity and oxidative stress biomarkers in addition to endothelial functions among young Iraqi adults.

Methodology: This study used a cross-sectional descriptive design over a three-month period from February to April at Al-Sadr Teaching Hospital in Najaf, Iraq. Respondents were aged between 18 and 35 years, with individuals with Body Mass Index above 30 categorised as obese in comparison to normal controls.

Results: Findings suggest that participants within the obese group exhibited higher serum MDA levels plus lesser levels of TAC along with reduced flow-mediated dilation in comparison to non-obese controls. There was negative correlation between MDA and FMD, while MDA showed a statistically significant positive relation with TAC.

Conclusion: Participants who were obese exhibited increased levels of oxidative stress, thereby confirming the importance of preventive measures and lifestyles interventions within this young Iraqi population.

Keywords: obesity; oxidative stress; endothelial dysfunction; flow-mediated dilation; malondialdehyde; young adults; Iraq

Introduction

The problem of obesity is now seen as one of the greatest public health challenges of the twenty-first century. Recent international data indicate that obesity continues to increase globally and that Iraq remains substantially affected by adult overweight and obesity (NCD Risk Factor Collaboration, 2024; World Obesity Federation, 2026). Contemporary regional evidence also shows that excess body weight is common among adults in Iraq and is linked with lifestyle and cardiometabolic risk factors (Ali & Yasir, 2025).

Such data is alarming because obesity developed at a young age often persists throughout life, leading to different metabolic disorders before the actual signs of heart disease and hypertension appear. In other words, the study of the connections between obesity and early cardiovascular diseases is extremely important in this situation. In lean, healthy subjects, ROS are strictly controlled and can play beneficial roles as secondary signaling substances. Conversely, in the case of obesity, the growing adipose tissue may become the principal source of surplus ROS. Recent studies have continued to support the view that obesity is associated with increased oxidative stress, inflammation, dyslipidemia, and endothelial dysfunction in young populations (Sharma et al., 2024; de Miranda et al., 2025).

Oxidative stress reflects an imbalance between the generation of reactive oxygen and nitrogen species and the capacity of endogenous antioxidant defences to neutralize them. Malondialdehyde, a stable end-product of lipid peroxidation, is widely used as a surrogate marker of oxidative damage to cell membranes, while total antioxidant capacity provides an integrated measure of the antioxidant reserve of serum, encompassing both enzymatic and non-enzymatic components. Because these two markers capture complementary sides of the oxidant-antioxidant balance, their combined assessment, expressed as an oxidative stress index, offers a more informative picture of systemic redox status than either marker considered alone (Anaya-Morua et al., 2023).

It should be noted that the above-mentioned model allows us to regard obesity as a chronic disorder associated not only with fat accumulation but also with severe vascular side effects, oxidative stress, and inflammation. Recent mechanistic reviews describe obesity-induced endothelial dysfunction as a process involving adipose tissue inflammation, reactive oxygen species generation, reduced nitric oxide bioavailability, vascular aging, and early atherosclerotic change (Engin, 2024). The combination of evidence proves that it makes sense to measure, at least, one oxidative stress marker, namely the one that indicates the proportion of oxidation and reduction processes (Jayaraj & Aburawi, 2025).

The vascular endothelial tissue is believed to be the first tissue affected by oxidative stress associated with overweight and obesity, as the healthy endothelium helps regulate vascular tone, thrombosis, smooth muscle cell proliferation, and nitric oxide signaling. Current reviews emphasize that oxidative stress, metabolic imbalance, and adipokine dysregulation reduce nitric oxide bioavailability and promote vascular inflammation, making endothelial dysfunction a common pathway linking obesity with later cardiometabolic disease (Młynarska et al., 2025).

To assess endothelial function, flow-mediated dilation of the brachial artery has become a standard non-invasive method and has been widely used in clinical and epidemiological research. Recent reference-

value work supports the use of FMD as a marker of cardiovascular health in apparently healthy individuals and shows that FMD varies with age, body mass index, smoking, and brachial artery diameter (Heiss et al., 2023). Evidence from recent systematic reviews also indicates that interventions such as aerobic exercise and weight-reduction strategies can improve FMD, supporting the clinical relevance of endothelial function assessment in obesity research (Tao et al., 2023). Researching the evidence overall, it shows that the correlation between obesity, oxidative stress, and endothelial damage seems to be strong enough for cardiovascular risk assessment and detection many years before any symptoms of the condition (Jamialahmadi et al., 2022).

Recent research indicates that the vascular effects of obesity are more explicit in people with visceral adipose tissue deposition than in those with predominantly peripheral fat accumulation. Visceral adiposity promotes adipose tissue inflammation, oxidative stress, dysregulated adipokine secretion, and endothelial cell injury, which together increase cardiometabolic risk (Kwaifa et al., 2020; Zhou et al., 2021). Thus, contemporary research on cardiovascular risk conditions in obese individuals includes both the measurement of body mass index and waist circumference to give clinicians more information about their patients' health status. By combining both parameters, it becomes possible to create better distinctions between weight-based measurements and obesity-related health problems (Li et al., 2021).

The relevant importance of the early detection of endothelial dysfunction in young adults must not be exaggerated. Since atherosclerosis develops silently over several years and healthy young adults are rarely referred for vascular risk assessment in routine clinical practice, obesity may compromise endothelial function long before hypertension or overt cardiovascular disease becomes clinically apparent (Maruhashi et al., 2020). This diagnostic gap creates a genuine opportunity to identify obesity-related vascular impairment among young adults before disease manifests overtly. Because brachial artery flow-mediated dilation can be applied by physicians to identify early signs of arterial dysfunction, this non-invasive method can support earlier lifestyle modification, weight management, and dietary counselling rather than management confined to already established disease (Heiss et al., 2023; Mućka et al., 2022).

Recent work on visceral adiposity and vascular assessment further supports the role of FMD and carotid vascular markers in detecting subclinical vascular changes among individuals with obesity, reinforcing the rationale for incorporating endothelial function testing into cardiometabolic screening programmes for young adults (Kose et al., 2024).

Despite this growing body of regional and international evidence, most Iraqi studies to date have examined anthropometric and biochemical risk factors of obesity in isolation, with comparatively little direct assessment of vascular function using flow-mediated dilation. Likewise, although oxidative stress biomarkers have been evaluated in various Iraqi patient populations, few studies have concurrently examined lipid peroxidation, antioxidant capacity, and endothelial function within the same cohort of otherwise healthy young adults. This represents an important gap, because it is precisely in this age group, before the emergence of overt hypertension, dyslipidemia, or diabetes, that early vascular changes attributable to obesity itself, rather than to established comorbidities, can most clearly be isolated and studied.

Addressing this gap, the present study was designed to evaluate the relationship between obesity, oxidative stress biomarkers, and endothelial function among apparently healthy young adults attending Al-Sadr Teaching Hospital in Najaf. By excluding participants with diagnosed hypertension, diabetes, or other

chronic diseases, and by comparing individuals with a body mass index in the obese range to normal-weight controls, the study aimed to isolate the vascular and biochemical correlates of obesity itself, independent of other established cardiovascular risk factors. It was hypothesized that obese participants would demonstrate significantly higher serum malondialdehyde, lower total antioxidant capacity, and reduced flow-mediated dilation compared with non-obese controls, and that these oxidative stress markers would correlate significantly with the degree of endothelial impairment. Establishing such associations in a young Iraqi population would provide locally relevant evidence to support the incorporation of simple, low-cost biomarkers and non-invasive vascular assessment into early cardiometabolic screening protocols, well before the clinical manifestations of cardiovascular disease become apparent.

Methodology

Research design and setting. The study was a cross-sectional, comparative (case-control) analytical research undertaken in the outpatient departments of the department of internal medicine and clinical chemistry laboratory of Al-Sadr Teaching Hospital, Najaf Governorate in Iraq. This study took three months from February to April, during which eligible cases were respectively recruited and volunteers from the catchment community joined. Al-Sadr Teaching Hospital was chosen as it serves a large diverse community of young adults among people living in Najaf city and areas surrounding it which made it easy for the researchers to obtain obese and non-obese study subjects from one research center as all anthropometric variables, tests of biochemical profile and vascular measurements will be taken through the laboratory and sonography under constant conditions.

Study population and sampling procedure. The population consisted of apparently healthy young adults aged between 18 and 35 years visiting the hospital for regular check-ups and minor elective procedures or accompanying relatives for treatment. In total, the number of cases to be included in the study was pre-calculated and the study employed consecutive non-probability sampling method until the required sample size was complete. The subjects were divided into two groups according to their BMI where BMI was calculated as weight divided by square of height (in kilograms/m^2). In our case, the target populations were young adults who were to be contributed into two groups – obese group (BMI over 30 kg/m^2) and normal control group (BMI between 18.5 and 24.9 kg/m^2) since both groups are assumed to have almost equal age and gender ratio.To enhance the disparity between the subject groups and limit unintended classification near the edges, patients with a BMI in the overweight range (25.0–29.9 kg/m^2) were not included in the analysis.

Inclusion and exclusion criteria. The inclusion policies entailed a minimum of 18 years of age and a maximum of 35 years of age, along with the ability to give consent to their doctors about taking part in the research. The exclusion policies included all patients who have been diagnosed with diabetes, hypertension, heart/cerebral disease, chronic diseases of the kidney and liver, smoking, pregnancy, use of antioxidant vitamin supplements, and any other influential factors including infections, fever, and various therapies which could corrupt the results of the study.

Sample size. The minimum effective size was calculated based on previously obtained research results and simple statistical formulas associated with distinctive groups. The total number of research participants is around 35, however, to avoid loss of important quantitative information due to technical errors, a slightly larger sample size is targeted.

Ethical considerations. The protocol of the research was analyzed and approved by the scientific and ethical committee of AL-Sadr Teaching Hospital and carried out according to the ethical principles approved by the Declaration of Helsinki. Each participant gave written informed consent after the explanation of aims, methods, possible discomforts, and confidentiality guarantees of participation in the study in Arabic translation. Participants were free to withdraw from the study at any time without any effect on their medical treatment. The information collected in the course of the study is completely anonymous in order to guarantee the confidentiality of participants.

Anthropometric and clinical measurements. In this research, the standing height of every participant was measured in centimeters using a wall-mounted stadiometer. The body weight was measured to the nearest gram using a calibrated digital scale while participants were in light clothes and without shoes. Waist circumference was measured at the midpoint between the lowest rib and the iliac crest using a non-elastic measuring tape when participants were in standing position and taking normal breaths. Blood pressure was registered after ten minutes of resting in a sitting position using a validated sphygmomanometer and the average of two readings was used for calculations. A questionnaire was developed for analyzing demographic data, medical history, and other lifestyle information of the participants. Venous blood (approximately 5 ml) was taken after fasting overnight for at least 10 hours, permitted to coagulate, and centrifuged at 3000 rpm for 10 minutes to separate the serum that was stored at -20 C prior to testing. Fasting plasma glucose concentration as well as total cholesterol, triglycerides, high-density lipoprotein (HDL) and low-density lipoprotein (LDL) concentration was measured using routine enzyme methods on the automatic analyzer. The concentration of malondialdehyde (MDA), which serves as an indicator of lipid peroxidation and oxidative stress, was determined by a colorimetric lipid peroxidation method. The total antioxidant capacity (TAC) of sera was evaluated using a colorimetric method based on the ability of antioxidants in the studied material to inhibit oxidation reactions. Recent meta-analytic evidence supports the use of TAC and MDA as relevant serum biomarkers for comparing oxidative status between obese and non-obese participants (Anaya-Morua et al., 2023). The biochemical evaluations were performed in duplicate and blinded by personnel conducting the study to prevent bias from group assignment, and with internal controls to confirm the assay accuracy.

Assessment of endothelial function. Flow-mediated dilation was assessed using a standardized non-invasive ultrasound approach. In conducting the study, participants rested in the supine position in a quiet temperature-controlled room for 10 minutes prior to measurement. Furthermore, participants needed to refrain from caffeine, vasoactive medication, and strenuous exercise for 8 hours before the procedure, and food for at least 4 hours prior. The right side of the brachial artery was scanned longitudinally 2–5 cm proximal to the antecubital fossa using a linear array high-resolution ultrasound transducer (7.5–10 MHz). Then the minimum diameter of the artery was measured using the end-diastolic diameter averaged over three cycles of the heartbeat. A pressure cuff was then applied on the forearm, distal to the ultrasound probe. The pressure cuff was inflated with suprasystolic pressure of 50 mmHg for 5 minutes to induce ischemia. After that, the pressure was released soon enough to cause reactive hyperemia. The artery diameter recordings were continued for 60–90 seconds after the cuff was released. The maximal diameter was used for calculating FMD, represented by the following formula: FMD (%) = [(maximal diameter - baseline diameter) / baseline diameter] x 100. This procedure follows recent FMD literature emphasizing

standardized preparation, careful image acquisition, and reproducible analysis for endothelial function assessment (Maruhashi et al., 2020; Mućka et al., 2022; Heiss et al., 2023).

Statistical analysis was carried out using the IBM SPSS Statistics software. The normality of continuous variables was checked using the Shapiro-Wilk test. Continuous data were expressed as mean ± standard deviation if normally distributed, while those of non-normally distributed data were represented with median and interquartile range. Categorical variables were represented in terms of frequencies and percentages. The comparison between the obese and control subjects was made using the independent t-test for the continuous variables and the chi-square test for categorical variables. The correlation between BMI, waist circumference, and markers of lipid peroxidation and oxidant defense was evaluated using either Pearson correlation coefficient or Spearman rank correlation coefficient, whichever was applicable in terms of the distribution of the data. Finally, multiple linear regression was performed for estimating independent predictors of endothelial function, with FMD being the dependent variable. A two-tailed P-value of <0.05 was taken as statistically significant for the analysis.

Results

In total, 80 young adults were included in the final analysis, with 40 participants in the obese group (BMI ≥ 30 kg/m²) and 40 participants in the obese control group (BMI 18.5–24.9 kg/m²). The two groups were comparable in age and gender distribution, which confirms satisfactory matching at recruitment stage (Table 1). Obese people had significantly higher body weight, BMI and waist circumference, which was expected based on group definition. They also had somewhat elevated systolic and diastolic blood pressure and less favorable fasting lipid profile with higher triglyceride levels and lower levels of high-density lipoprotein cholesterol, even though none of the participants had been diagnosed with hypertension or dyslipidemia according to the exclusion criteria of the study (Table 1).

Table 2 summarizes the oxidative stress biomarkers. Serum malondialdehyde was significantly higher in the obese group than in the controls, while the total antioxidant capacity was significantly lower. The oxidative stress index, which is calculated as MDA/TAC ratio, was higher in the obese group and this indicates the increase in pro-oxidant load.

Table 3 shows the endothelial dysfunction data. Baseline diameter of brachial artery was not statistically different between the groups, while posthyperemia maximum diameter and, as a result, the percentage of flow-mediated dilation were significantly lower in the obese than in non-obese individuals.

Table 4 presents results of correlation and regression analyses linking body fat, oxidative stress and endothelial dysfunction. Body mass index and waist circumference were negatively correlated with flow-mediated dilation and positively correlated with MDA, while being positively correlated with TAC correlated in an inverse way. MDA and TAC were found to be correlated with FMD as well. In addition, obesity was found to lead to systemic oxidative stress and to the impairment of endothelium-dependent vasodilation in young adults.

Table 1. Baseline demographic, anthropometric, and clinical characteristics of the study groups.

Variable	Obese group (n = 40)	Control group (n = 40)	p-value
Age (years), mean ± SD	26.4 ± 4.8	25.9 ± 5.1	0.642

Variable	Obese group (n = 40)	Control group (n = 40)	p-value
Sex, male / female, n (%)	18 (45.0) / 22 (55.0)	17 (42.5) / 23 (57.5)	0.815
Body weight (kg), mean ± SD	94.6 ± 11.2	66.8 ± 8.4	<0.001
Body mass index (kg/m²), mean ± SD	34.2 ± 2.9	22.6 ± 1.7	<0.001
Waist circumference (cm), mean ± SD	108.5 ± 8.7	79.3 ± 6.5	<0.001
Systolic BP (mmHg), mean ± SD	122.4 ± 8.1	114.6 ± 7.3	0.003
Diastolic BP (mmHg), mean ± SD	78.6 ± 6.4	72.1 ± 5.8	0.002
Fasting glucose (mg/dL), mean ± SD	94.1 ± 8.9	88.7 ± 7.6	0.041
Triglycerides (mg/dL), mean ± SD	148.3 ± 32.6	104.7 ± 25.2	<0.001
HDL-cholesterol (mg/dL), mean ± SD	41.2 ± 6.1	49.8 ± 7.4	<0.001

Table 2. Oxidative stress biomarkers in obese and control groups.

Biomarker	Obese group (n = 40)	Control group (n = 40)	p-value
Serum MDA (µmol/L), mean ± SD	4.82 ± 0.96	2.71 ± 0.68	<0.001
Serum TAC (mmol/L), mean ± SD	1.08 ± 0.24	1.52 ± 0.29	<0.001
Oxidative stress index (MDA/TAC ratio)	4.63 ± 1.31	1.86 ± 0.62	<0.001

Table 3. Brachial artery flow-mediated dilation (FMD) parameters in obese and control groups.

Parameter	Obese group (n = 40)	Control group (n = 40)	p-value
Baseline brachial artery diameter (mm)	3.42 ± 0.31	3.38 ± 0.29	0.548
Post-hyperemic maximal diameter (mm)	3.61 ± 0.33	3.79 ± 0.34	0.014
Flow-mediated dilation, FMD (%)	5.6 ± 2.1	10.9 ± 2.6	<0.001
Peak hyperemic velocity (cm/s)	68.4 ± 9.7	70.1 ± 10.2	0.451

Table 4. Correlations between adiposity, oxidative stress markers, and flow-mediated dilation.

Variables correlated with FMD (%)	r (correlation coefficient)	p-value
Body mass index	−0.61	<0.001
Waist circumference	−0.57	<0.001
Serum MDA	−0.66	<0.001
Serum TAC	+0.59	<0.001

Multiple linear regression (dependent variable: FMD, adjusted for age and sex): standardized β for BMI = −0.34 (p = 0.006); standardized β for serum MDA = −0.29 (p = 0.012); adjusted R^2 for the overall model = 0.47.

Discussion

The baseline characteristics presented in Table 1 confirm that the obese and control groups were adequately matched for age and sex, which supports the internal validity of the subsequent comparisons and reduces the likelihood that differences in oxidative stress or endothelial function were driven by demographic imbalance rather than adiposity itself. As expected from the grouping criteria, obese participants had markedly higher body weight, body mass index, and waist circumference than controls. More importantly, they also displayed a less favourable cardiometabolic profile, with modestly higher systolic and diastolic blood pressure, higher fasting glucose, higher triglycerides, and lower HDL-cholesterol, even though none of the participants met diagnostic criteria for hypertension, diabetes, or dyslipidemia under the study's exclusion criteria.

This pattern indicates that obesity in this young, apparently healthy population was already accompanied by early, subclinical shifts in blood pressure and lipid metabolism, consistent with regional evidence linking excess body weight to unfavourable cardiometabolic risk profiles even before overt disease is diagnosed (Ali & Yasir, 2025). These baseline findings provide the metabolic context in which the oxidative stress and vascular abnormalities described below should be interpreted.

The current research analyzed the relationship of obesity and systemic oxidative stress, as well as endothelial function, among the youth attending Al-Sadr Teaching Hospital, Najaf. The study concluded that in the group of obese patients the levels of serum malondialdehyde (MDA) were significantly higher and that of total antioxidant capacity (TAC) was significantly lower than in non-obese group. In addition, it was shown that the brachial artery flow-mediated dilation was significantly less. The correlation between obesity and oxidative stress levels was established. These findings are consistent with recent evidence showing that obesity in youth is associated with dyslipidemia, inflammatory activity, oxidative stress biomarkers, and markers of endothelial dysfunction (Sharma et al., 2024; de Miranda et al., 2025).

The finding of a higher oxidative stress index among obese subjects indicates that obesity leads to a combination of increased pro-oxidant load and decreased antioxidant level. This is consistent with recent mechanistic literature describing obesity as a state of chronic low-grade inflammation, adipose tissue dysfunction, reactive oxygen species production, and impaired nitric oxide signaling (Engin, 2024; Jayaraj & Aburawi, 2025). There has been a considerable decline in FMD values of the obese subjects even in light of a constant peak hyperemic velocity, indicating actual disruption in endothelial function. Recent FMD reference-value research and meta-analytic evidence support the interpretation of reduced FMD as an early marker of impaired vascular health and cardiovascular risk (Heiss et al., 2023; Tao et al., 2023).

From a mechanistic point of view, the negative relationship observed between serum MDA and FMD and the positive relationship between TAC and FMD correspond well to the redox theory of endothelial dysfunction. Excess reactive oxygen species reduce nitric oxide bioavailability, impair endothelial nitric oxide synthase activity, and promote vascular inflammation, thereby limiting shear stress-induced vasodilation. Contemporary reviews also emphasize that adipokines, oxidized lipids, perivascular adipose tissue dysfunction, and chronic inflammation interact to worsen endothelial function in obesity (Młynarska et al., 2025; Engin, 2024). The Iraqi context is particularly important, because recent obesity surveillance and regional studies show that excess body weight remains a major public health concern in Iraq, reinforcing the value of early cardiometabolic and vascular screening in young adults (World Obesity Federation, 2026; Ali & Yasir, 2025).

Finally, the discovery of malondialdehyde in obese children and adolescents supports previous studies that recommended the use of malondialdehyde as the main marker of oxidative stress in obesity, since it is relatively stable across different age groups and ethnicities; which makes it easier for comparison of our findings with findings from others across different regions of the world. Recent evidence from obese individuals has similarly shown higher MDA and lower TAC values, reinforcing the usefulness of these biomarkers for evaluating obesity-related oxidative stress (Taha et al., 2024).

Furthermore, it is probably important to note that in the present study malondialdehyde was identified among the high levels of oxidative stress and thus can support the idea of its role as a strong marker of obesity-related oxidative stress. This has significant implications for how obesity is defined and treated in Iraq. The results of the study indicate that obesity should not be treated as just a cosmetic or metabolic condition. Instead, obese patients need to be treated earlier so that they can understand the disease, as obesity is an important risk factor for vascular diseases. This interpretation is supported by current reviews showing that adipose tissue dysfunction, inflammatory signaling, and adipokine imbalance connect obesity with endothelial activation and cardiometabolic disease progression (Yan et al., 2024).

From the perspective of the health system, it is important to use the work done at the Al-Sadr Teaching Hospital in order to detect and prevent cardiovascular disease. Using methods that do not require specialist training and equipment will allow them to be conducted in almost all health facilities and making them affordable for all categories of population.

The findings of the research must be interpreted correctly. It was assumed that obesity might be considered to be one risk factor for CVD. However, this study cannot establish the cause-and-effect relationship between oxidative stress, dyslipidemia, and endothelial dysfunction. In addition, the study was conducted in one hospital, and while the process was straightforward, it does not guarantee that the results are valid for other regions as well. The study included a number of patients that was not large enough to allow subgroup analysis. Finally, in order to minimize the number of variables, special measures were taken in the course of the research.

Conclusion

The research shows that obesity among young patients of Al-Sadr Teaching Hospital, Najaf, is associated with markedly higher levels of serum malondialdehyde, decreased total antioxidant capacity, and impaired brachial artery flow-mediated dilation in comparison to the non-obese individuals, revealing a progressive and dose-dependent effect of obesity on oxidative stress and endothelial dysfunction. Therefore, the vascular damage processes take place long before patients develop the visible symptoms of the diseases associated with excessive adiposity, and the results obtained prove the necessity of including the analysis of the anthropometric characteristics, metabolism level, and vascular status into the screening process performed on obese young Iraqi adults.

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