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Insulin Resistance in Association with Metabolic Syndrome in Wasit University Students in Iraq

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Abstract

Metabolic syndrome (MetS) and insulin resistance (IR) are increasing in prevalence worldwide. Each carries risks for the development diabetes mellitus and cardiovascular disease. These risks will be greatly exaggerated if they occur together in the same individual.

This study aimed to find the prevalence of IR in subjects with MetS and the association between these two condition and the potential risk factors for their development in university students.

In this cross-sectional study, participants were selected from Wasit University in Wasit, Iraq. The recruited candidates were apparently healthy aged between 18 and 25 years. The diagnosis of MetS was depending on the IDF/AHA/NHLBI criteria, and HOMA-IR was used to determine IR. Anthropometric measurements and blood pressure were recorded for each participant; along with the measurement of fasting serum levels of insulin, glucose, and lipid profile.

From 124 MetS subjects in this study, the prevalence of IR was (24.2%). The component of MetS that were significantly associated with the risk of insulin resistance is central obesity (OR 2.24) and high blood pressure (BP) with (OR 2.107, 2.41) for systolic and diastolic blood pressure respectively, also found that Body mass index (BMI) ≥25, smoking and consumption of fast food were risk factors for the development of IR (OR 6.107, 2.633, 3.45) respectively.

Insulin resistance is quite common in Iraqi university students with MetS. The more encountered MetS components were central obesity and high BP; moreover, overweight, smoking and the tendency for consumption of fast food are the major risk factors for IR in those individuals.

Keyword: Insulin resistance, Metabolic syndrome, HOMA-IR, University students.

مقاومة الانسولين و ارتباطها بمتلازمة التمثيل الغذائي في طلاب الجامعة العراقيين

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الخلاصة

يتزايد انتشار متلازمة التمثيل الغذائي ومقاومة الأنسولين في جميع أنحاء العالم. كل منها يحمل مخاطر لمرض السكري في المستقبل وتطور أمراض القلب والأوعية الدموية. ستتفاقم هذه المخاطر بشكل كبير إذا حدثت معًا في نفس الفرد.

الغرض من هذه الدراسة هو معرفة انتشار مقاومة الأنسولين في الأشخاص الذين يعانون من متلازمة التمثيل الغذائي ، والارتباط بين هاتين الحالتين وعوامل الخطر المحتملة لتطورهما لدى طلاب الجامعات.

في هذه الدراسة المقطعية ، تم اختيار المشاركين من جامعة واسط في واسط ، العراق. أن المرشحين المعينين هم يبدون أصحاء تتراوح أعمارهم بين ١٨ و ٢٥ عامًا. تم وصف متلازمة التمثيل الغذائي باستخدام معايير IDF / AHA / NHLBI ، وحددت صيغة HOMA-IR مقاومة الأنسولين (IR). تم تسجيل القياسات الأنثروبومترية وضغط الدم لكل مشارك. إلى جانب قياس مستويات الأنسولين والجلوكوز والدهون في مصل الصيام.

من بين ١٢٤ مشاركًا في هذه الدراسة ، كان معدل انتشار مقاومة الانسولين في الأشخاص الذين يعانون من MetS (٢٤,٢ ٪). إن ارتباط مكونات MetS بخطر مقاومة الأنسولين هو السمنة المركزية(OR ٢,٢٤) و ارتفاع ضغط الدم مع (٢,١٠٧، ٢,٢١) على التوالي ، كما وجد أن ارتفاع مؤشر كتلة الجسم (> ٢٥) ، التدخين والاستهلاك المرتفع للوجبات السريعة مرتبط بشدة بحدوث مقاومة الأنسولين(OR٦,١٠٧و ٢,٦٣٣و ٣,٤٥) على التوالي.

مقاومة الأنسولين شائعة جدًا لدى طلاب الجامعات العراقية الذين لديهم متلازمة التمثيل الغذائي. كانت مكونات MetS الأكثر شيوعًا هي السمنة المركزية وارتفاع ضغط الدم. علاوة على ذلك ، فإن زيادة الوزن و التدخين والميل إلى استهلاك الوجبات السريعة هي عوامل الخطر الرئيسية للإصابة بمقاومة الانسولين لدى هؤلاء الأفراد.

الكلمات الرئيسية: مقاومة الانسولين، متلازمة التمثيل الغذائي ، اختبار تحليل مقاومة الانسولين ، طلاب الجامعة.

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Introduction

Insulin resistance (IR), is a state of reduced cellular response to the hormone insulin in insulin-dependent cells such as skeletal muscle, adipose tissue and liver. IR is caused by a combination of genetic and environmental factors. IR is one of the earliest manifestations of a group of human diseases, including type 2 diabetes (T2DM) and cardiovascular disease. These diseases are typically associated with intertwined metabolic abnormalities, including obesity, hyperinsulinemia, hyperglycemia, and hyperlipidemia[(1)] .

Oxidative stress, mitochondrial dysfunction and central obesity that increase with age, increase the risk of the development of IR.[(2)] Obese individuals were reported to have high serum levels of inflammatory cytokines and free fatty acids that were associated with reduced insulin sensitivity and T2DM[(3)] .Gender, physical inactivity and unhealthy food, represent additional risk factors for the development of IR[(4–6)] .

The incidence of insulin resistance differs between nations. According to studies, the prevalence was (15%) for young adults Swedish [(7)] .and, 25.3% for adolescent in Brazil.[(8)], 48.5% for adults in Congo in Africa[(9)] . In meta-analysis for adults in Southeast Asia was 44.3%, with Malaysia having the highest prevalence rate 50.4%, followed by Indonesia 44.2%. [(10)] While in Lebanon, the prevalence of IR among adults was 38%[(11)] . Metabolic syndrome (MetS) represents a cluster of metabolic risk factors, including central obesity, hyperglycemia, hypertension, and hyperlipidemia. However, the exact etiology of MetS is uncertain. IR is largely involved in the pathogenesis of the metabolic abnormalities that represent the components of MetS. Insulin sensitivity is significantly lower in subjects with two or more components of the metabolic syndrome compared to those with none of these components[(12)] .The purpose of this study was to survey the Prevalence of IR in subjects with MetS, and the association of IR with the component of MetS, and studying whether age, gender, physical activity, and smoking and dietary habits are risk factors for the development of IR and MetS in young Iraqi university students.

Subjects and Methods

Subjects.

Three hundred university students participate in this study (165 female and 135 male), 124 were diagnosed as MetS subjects according to IDF/AHA/NHLBI criteria. All participants enrolled in this study were from Wasit University in Wasit, Iraq, and they are apparently healthy aged between 18 and 25 years. Subjects with any condition or on any medication that may interfere with insulin sensitivity or glucose or lipid metabolism were excluded. The study was approved by The Ethics Committee of the College of Pharmacy, University of Baghdad; a verbal consent was taken from each participant after being informed about the purpose of the study and the expected benefits.

Anthropometrics and biochemical measurements.

During the 30-minute in-person interview, the questionnaire was distributed to the students. It included several questions about their age, daily eating of fast food and vegetables (≥ 1-3 times/week) considered eating, physical activity and the smoking status were divided into two groups regular active smokers (smoking at least 10 cigarette/week) and non-smokers, then the anthropometric measurements were done.

The performance of physical activity of moderate intensity for longer than 150 min/week [(13,14)] was recommended as adequate to maintain a good state of health. This was the cut-off point used in our study to differentiate the subjects doing regular physical activity from subjects not doing.

The height was measured with a portable stadiometer (Seca, Germany), the weight with a portable electronic scale, and the waist circumference was measured with a non-stretchable tape. Weight and height were measured with the subjects wearing light clothing and without shoes. The waist circumference was assessed at the midpoint between the lowest rib and the iliac crest at the end of a normal expiration[(15)] .The BMI was calculated by dividing the subject's weight in kilograms by the square of height in meters (kg/m^2) and classified according to the World Health Organization (WHO) recommendation[(15)] .Blood pressure (BP) was taken using a standardized mercury sphygmomanometer. The average of two recorded values was recorded**.**

Fasting blood sample was collected from each participant and was used for obtaining serum. Serum levels of fasting blood glucose (FBG), high-density lipoprotein cholesterol (HDL-C), total cholesterol (TC), and triglycerides (TG) were measured colorimetrically using the corresponding kits: glucose kit (LINEAR CHEMICALS, SPAIN), HDL-C kit (LINEAR CHEMICALS, SPAIN), cholesterol kit (Biolabo SAS, France), and TG kit (LINEAR CHEMICALS, SPAIN). Fasting insulin was measured using a commercially available enzyme-linked immunosorbent assay (ELISA) kit: (DEMEDITEC Insulin ELISA kit, Germany).

Insulin resistance was calculated using the homeostasis model assessment of insulin resistance (HOMA-IR); where HOMA-IR= [(FBG (mg/dL) × fasting insulin (μU/mL))/405].[(16)] HOMA-IR values above or equal to 2.0 were the diagnostic value in distinguishing individuals with IR[(17)] .

The metabolic syndrome (MetS) was diagnosed using the IDF/AHA/NHLBI criteria([18]) .The diagnosis was based on the presence of three or more of the following components: FBG level ≥ 100 mg/dL, TG levels ≥ 150 mg/dL, systolic and/or diastolic BP of ≥130 and ≥ 85 mmHg, respectively, waist circumference (WC) with ethnic-specific values for Middle East (Arab) population's ≥ 37 inches (94cm) for male and ≥ 31.5 inches (80cm) for female and HDL-C level < 40 and < 50 mg/dl for male and female, respectively.

Statistical analyses

Statistical analysis of the present study was conducted with the Windows version 25 of the Statistical Package for the Social Sciences (SPSS). The Shapiro-Wilk test was applied to assess the assumption of normality for the data. Continuous variable expressed as median (interquartile rang), and difference between groups were checked using Mann-Whitney tests. And categorical variables were expressed as percentages, And difference between groups were checked using Chi-square test. The differences between the two groups with and without IR were compared using Mann-Whitney U tests. An odds ratio (OR) is used to measure the association between IR group and MetS components and to study whether age, gender, physical activity, smoking, and dietary habits are risk factors for the development of IR in a population with MetS. Observed associations were expressed as odds ratios (OR) with 95% confidence intervals (CI). A P value of less than 0.05 was considered statistically significant.

Results

The present study showed that 124 (41.3%) of participants were having MetS; the prevalence was higher in females 83(67%) than in males 41(33%). Metabolic syndrome subjects showed that 122 subjects (98.4%) were having FBG level >100 mg/dL; 109 (88%) were having WC ≥ 37 inches for male and ≥ 31.5 inches for female; 106 subjects (85.5%) were having HDL-C lower than 50mg/dl for females and 40mg/dl for males; 53 subjects (42.7%) were having high systolic blood pressure (≥130 mmHg); 50 subjects (40%) were having high diastolic blood pressure ≥85 mmHg; and 30 subjects (24.2%) were having insulin resistance (HOMA-IR ≥2), most of participants non-smokers (80%). (Table-1 and 2)

Table 1. Biochemical measurements of participants with MetS as Median (Interquartile Range).

	Female (83)	**Male (41)**	**p-value**
FBG (mg/dl)	112 (7)	116(12)	0.059
Insulin (µU/ml)	5.4 (1.4)	6.3(2.3)	0.012*
HDL-C (mg/dl)	42 (11)	38(15)	0.987
TG (mg/dl)	107 (5)	110(5)	0.015*

Table2. Anthropometric, biochemical, and lifestyle characteristics of participants with MetS.

	Total (124) *n* (%)	**Female** (83) *n* (%)	**Male** (41) *n* (%)	**p-value**
Age (year)				
18-21	54(43.5)	42 (50.6)	12(29)	0.02*
22-25	70(56.5)	41 (49.4)	29(71)	
BMI (Kg/m²)				
Underweight < 18.5	1 (0.8)	0 (0)	1 (2.5)	
Normal-weight 18.5-24.9	40 (32.2)	34 (41)	6 (14.7)	0.008*
Overweight 25 - 29.9	64 (51.6)	36 (43.4)	28(68.1)	
Obese ≥30	19 (15.4)	13 (15.6)	6 (14.7)	
WC (inch)				
Normal <31.5(female) <37(male)	15 (12)	6 (7)	9 (22)	0.01*
High ≥31.5(female) ≥37(male)	109 (88)	77 (93)	32 (78)	
BP (mmHg)				
SBP < 130	71 (57.3)	62 (75)	9 (22)	<0.001*
≥130	53(42.7)	21 (25)	32 (78)	
DBP <85	74 (60)	60 (72)	14 (34)	<0.001*
≥85	50 (40)	23 (28)	27 (66)	
FBG (mg/dl)				
<100	2 (1.6)	1 (1)	1 (2.5)	0.06
≥100	122(98.4)	82 (99)	40 (97.5)	

Counited table 2.

Insulin (µU/ml) Normal 2-25	124 (100)	83 (100)	41 (100)	_
HDL-C (mg/dl) Normal ≥ 50 (female) ≥ 40(male) Low <50(female) < 40(male)	18 (14.5) 106 (85.5)	3 (3.6) 80 (96.4)	15 (36.5) 26 (63.5)	<0.001*
TG (mg/dl) <150 ≥150	124(100) 0 (0)	83 (100) 0 (0)	41 (100) 0 (0)	_
HOMA-IR <2 ≥2	94 (75.8) 30 (24.2)	70 (84.3) 13 (15.7)	24 (58.5) 17 (41.5)	0.002*
Smoking Yes No	25 (20) 99 (80)	2 (2.4) 81(97.6)	23 (56) 18 (44)	<0.001*
fast-food-eating Yes No	113(91) 11 (9)	75(90.4) 8 (9.6)	38 (93) 3 (7)	0.066
Physical activity **Yes** **No**	0 (0) 124(100)	0 (0) 83 (100)	0 (0) 41 (100)	_

BMI: body mass index; WC: waist circumference; SBP: systolic blood pressure; DBP: diastolic blood pressure; FBG: fasting blood glucose; HDL-C: High-density lipoprotein cholesterol; TG: triglycerides; HOMA-IR: homeostasis model assessment of insulin resistance; Smoking: NO: nonsmoker; Fast-food-eating: No: Never ate fast food; physical activity: No: less than 150 min/week; *: significant difference.

Based on the results of the HOMA-IR, participants were divided into two groups: Insulin-resistant and non-insulin-resistant. (Table 3) Subjects with IR had higher BMI, WC, FBG levels, fasting insulin levels, triacylglycerol levels, and low HDL-C levels than subjects without IR. Statistics showed that this difference was significant ($p < 0.05$). While SBP and DBP variables were not significantly differentiated between the two groups.. The following clinical factors were shown to be highly linked with IR (Table 4): central obesity (WC male > 37, female > 31.5 inch) with the odds ratio 2.24, and high blood pressure (BP > 130/85 mmHg) although it was not statistically significant for SBP, also exhibited a connection with IR.

Table 3 . A comparison of individuals with and without insulin resistance.

variable	Insulin Resistance		p-value
	Yes (*n*= 30)	No (*n*= 94)	
Age (year)	22 (3)	22(3)	0.642
BMI (Kg/m²)	28.4(3.29)	25.8(4.19)	0.002*
WC (inch)	39 (3)	35 (6)	0.003*
BP mmHg SBP DBP	130 (19) 85 (12)	125 (22) 81 (15)	0.104 0.290
FBG (mg/dl)	122.5(10)	112 (6)	<0.001*
Insulin (µU/ml)	7.8 (2.1)	5.2 (1.01)	<0.001*
HDL-C (mg/dl)	46 (12)	38 (12)	0.001*
TG (mg/dl)	111 (3)	107 (4)	<0.001*

Data are Median (Interquartile Range), *significant difference.

Table 4 . Metabolic syndrome components as risk factors for Insulin resistance.

MetS component	Insulin resistance *n* (%)		OR	95% CI
	Yes (*n*=30)	**No** (*n*=94)		
FBG (mg/dl) <100 ≥100	 0 (0) 30(100)	 2 (2.1) 92 (97.9)	1	1 - 1.45
HDL-C (mg/dl) Normal Low	 13 (43.3) 17 (56.7)	 5 (5.3) 89 (94.7)	0.073	0.02-0.23
SBP (mmHg) <130 ≥130	 13 (43.3) 17 (56.7)	 58 (61.7) 36 (38.3)	2.107	0.91-4.84
DBP (mmHg) <85 ≥85	 13 (43.3) 17 (56.7)	 61 (64.9) 33 (35.1)	2.41	1.04-5.58
TG (mg/dl) <150 ≥150	 30 (100) 0 (0)	 94 (100) 0 (0)	–	–
WC (inch) Normal High	 2 (6.7) 28 (93.3)	 13 (13.8) 81 (86.2)	2.24	0.47-10.5

Low: HDL-C <40 for the male, <50 for the female. High: WC ≥37 inch for male, ≥31.5 for the female. Normal HDL-C ≥40 for male, ≥50mg/dl for female. Normal WC <37 inch for male, < 31.5 inch for female; OR: odd ratio; CI: confidence intervals.

According to the results in (Table 5) IR is more related with high BMI (>25) with OR 6.107 (95%CI: 1.7- 21.5), smoker subjects with OR 2.633(95%CI: 1.03- 6.73) and consumption of fasting food with OR 3.45 (95%CL: 0.42-28.1). All subjects with insulin resistance had a low level of physical activity. (Table 5).

Table 5 .The association of risk factor and IR.

	Insulin Resistance *n* (%)		OR	95% CI
	Yes (*n*= 30)	**No (***n*= 94)		
Age 18-21 22-25	 13(43.3) 17(56.7)	 41(43.6) 53(56.4)	1.01	0.44- 2.3
Gender Female Male	 13(43.3) 17(56.7)	 70(74.5) 24(25.5)	0.262	0.11- 0.618
BMI <25 ≥25	 3 (10) 27(90)	 38(40.4) 56(59.6)	6.107	1.7- 21.5
Smoking Yes No	 10(33.3) 20 (66.7)	 15(16) 79(84)	2.633	1.03- 6.73
Physical activity Yes No	 0 (0) 30(100)	 0 (0) 94(100)	–	–
fast-food-eating Yes No	 29(96.7) 1 (3.3)	 84(89.4) 10(10.6)	3.45	0.42- 28.1

Discussion

This research looked at the relationship between IR and MetS in healthy young adults in Iraq between 18 – 25 years. The results of the current study point to a significant association between subjects with IR and irregularities in each component of MetS in Iraqi university students, with a higher prevalence among males (56.7 %) than females. Insulin resistance was highly correlated with consumption of fast-food, high BMI, regular smoking and physical inactivity.

Insulin resistance prevalence in our study was 24.2% among124 subjects with MetS. Naja *et al.* reported (44.6%) IR prevalence among randomly selected 308 Lebanese adults (19). Globally, there was insufficient studies about university students, insulin resistance prevalence among adults differ by country , in Venezuela it was 46.5 % [20]. Other study for young Asian populations like a study among Malaysians were 44.5% [21], Korean 47.1% [22], and Chinese 44.3% [23].

In our study, only two of five MetS components have association with IR. The two components are high blood pressure and increased waist circumference with odds ratio (2.107 SBP, 2.41 DBP) and (2.24 WC), this finding is parallel and consistent with analysis by Juan Salazar *et al.* suggesting that obesity is the most common cause of IR in adult [20].

Insulin resistance (IR) is associated with obesity due to lipolytic effects of adipocytes leading to large amounts of free fatty acids and impaired secretion of adipokines and both involved in the modulation of insulin sensitivity [24].

Our study found that obesity is associated with insulin resistance (odds ratio 2.24). Through various productions in released metabolites, hormones and adipocytokines, adipose tissue plays a major role in the mechanism for developing insulin resistance [25]. The production of non-esterified fatty acids by adipocytes prevent carbohydrate metabolism through substrate competition and impaired intracellular insulin signaling. The most common cytokines secreted by adipose tissue is adiponectin. Adiposity is inversely correlated with adiponectin. Adiponectin possesses remarkable insulin-sensitizing effect hence the increase of adipose tissue causing reduced levels of adiponectin associated with insulin resistance and metabolic syndrome[26].

Insulin and obesity have a strong correlation. Obesity reduces the ability of the cell to respond to insulin, leading to insulin resistance and raising insulin levels. Conversely, elevated insulin levels promote weight gain [27]. By changing an organism's oxidative status, obesity contributes to the development of MetS. Through the control of several mechanisms, oxidative stress is closely linked to the rise in blood pressure [28]. More significantly, it is linked to the emergence of insulin resistance. Insulin resistance is brought on by oxidative stress in a number of cells that control the body's response to insulin: muscle, liver, and adipocytes. It has been established that oxidative stress is principally responsible for the developing of insulin resistance [29].

This study found a substantial correlation between IR and high blood pressure but not significant for SBP. It is well known that IR is linked to higher BP in most people, [30]. Insulin resistance contributes to increase BP through several mechanisms, among which are the enhanced tissue angiotensin II (AngII) and aldosterone activities, the increased sympathetic nervous system activity (SNS) through elevating norepinephrine levels, and oxidative stress. However, emerging evidence indicate that endothelial dysfunction may represent the upstream event preceding peripheral reduction of insulin sensitivity, due to impairment of peripheral tissue blood flow. [31]

In this study, the most risk factors that affected the incidence of IR were high BMI, regular active smoking and dietary consumption of fasting food. Being overweight may be an essential connection between IR and MetS in young. Likewise, Juarez-L ´opez *et al.* [32] demonstrated that (irrespective of age and gender), the severity of IR was strongly linked with the prevalence of disorders of the MetS component in obese children in Mexico. All of these results point to the possibility that IR may be crucial in grouping MetS risk factors, especially in young, healthy students.

Smoking increase the risk of insulin resistance with OR (2.633) in our study. This finding may be due to hormonal changes associated with smoking. Moreover, smoking may induce insulin resistance directly, owing to its effect on abdominal obesity, which may partly occur due to nicotine absorption during smoking [33]. Another possible mechanism involves the smoking-triggered secretion of hormones such as cortisol, catecholamines, and growth hormones, which oppose the effects of insulin. These hormones increase lipolysis, subsequently increasing free fatty acid release and impairing endothelial function, which may contribute to insulin resistance [34]. Finally, smoking is negatively associated with adiponectin levels in a dose–response manner [35]. Smoking cessation may protect against insulin resistance.[36].

There are a few hypothesized ways that eating fast food contributes to the emergence of insulin resistance and metabolic syndrome[37]. The most significant sign of them is low-quality nutrition caused by frequent fast-food consumption; which is identified by more eating of lipids, saturated fats, cholesterol, and salt, in addition to lesser eating of carbohydrates, fiber, calcium, and antioxidant vitamins, which are regarded to be the chief dietary risk factors for fat gain, metabolic syndrome, and insulin resistance [38] . Fast food's high fats, cholesterol, and saturated and trans fatty acids are also seen as risk factors for developing postprandial metabolic disorders, such as dyslipidemia, oxidative stress, and subclinical inflammations [39], as well as long-term abdominal fat gain, insulin resistance, and hypertension [37].

A study by Tucker *et al*. Found that 272 middle-aged non-diabetic American women had an increase in insulin resistance with high dairy intake [40]. And Z Bahadoran *et al.* showed that after three

years of follow-up in Iranian individuals, increased fast food intake had bad effects on metabolic syndrome [37]. A fifteen year follow-up of US women showed strong positive associations between fast food consumption with weight gain and insulin resistance [41] .

All subjects in our study had physical inactivity; a study by Vighnesh *et al*. [42] discovered that acute exercise significantly increased insulin sensitivity.

Metabolic syndrome is the key risk factor to monitor to forecast the development of cardiovascular disease (CVD) and type 2 diabetes (T2DM) accurately [43]. Additionally, insulin resistance is one of the most vital markers for assessing metabolic syndrome [12]. Thus, investigating the IR in an early stage is crucial for trying to stop the disease's progression into a chronic disease.

Conclusions

The current research shows a considerable percentage of IR among metabolic syndrome subjects. Central adiposity and high blood pressure are the two most significant MetS factors linked to IR. High BMI, smoking and fast food consumption also affect IR in these subjects. Consequently, the early detection of insulin resistance and MetS is essential. Effective interventions for improving insulin sensitivity, blood pressure and reducing abdominal obesity primarily by weight reduction, smoking cessation and physical activity may help in preventing type 2 diabetes and cardiovascular disease. Clinical trials in the future are required to validate our results.

Conflicts of Interest

None.

Funding

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Ethics statements

This work was performed according to the the Ethics Committee of the College of Pharmacy, University of Baghdad. Before their acceptance to participate in the trial, all participants were told about the study's goal and predicted benefits.

Author contribution

First author contribution: Collected the data, performed the analysis, statistical analysis and writing of the manuscript.

Second author contribution: Have made a substantial contribution to the concept and the design of the article, conceived and designed the analysis, revised it critically and approved the version to be published.

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