(ISSN Online: 3006-1113) Full Paper

Libyan Journal of Medical and Applied Sciences

Journal homepage: https://ljmas.com/index.php/journal/index

A RESEARCH ARTICLE

PREVALENCE OF OBESITY AMONG ADULTS IN SINJAR DISTRICT, IRAQ: ITS ASSOCIATION WITH COMMON CHRONIC DISEASES AND SOME INFLAMMATORY BIOMARKERS

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Article Information

Article history:
Received on: 11/Mar/2026
Revised on: 20/Apr/2026
Accepted on:10/May/2026
Published on: 06/Apr/2026

Keywords:
Obesity;
Sinjar district;
Chronic diseases;
Inflammatory state;
Interleukin 6

Abstract

Obesity is defined as excessive accumulation of body fat resulting from an imbalance between calories consumed and calories burned. This study aimed to determine the prevalence of obesity and overweight among adults in Sinjar District, Iraq, compared to other Iraqi cities. It also sought to confirm the relationship between the accumulation of adipose tissue, elevated levels of inflammatry markers, and the development of certain chronic diseases. It is worth noting that the Yazidi community in Sinjar differs ethnically, culturally, and religiously from the Arab majority in the country. This cross-sectional study included 240 adults aged 18–80 years, randomly selected between February and April 2025. Serum glucose, lipids, interleukin-6 (IL-6), and C-reactive protein (CRP) levels were measured to assess biomarkers associated with obesity. The results showed that, compared to other Iraqi cities, Sinjar recorded lower levels of obesity, but its overweight figures remained consistent with the general averages observed in previous research. A strong association was observed between body mass index (BMI) and the development of diabetes and cardiovascular disease, with significant increases ($p < 0.05$) in glucose, low-density lipoprotein (LDL), Triglyceride (TG), and inflammatory markers among overweight and obese individuals. This study concluded that the ethnic and cultural distinctiveness of the Yazidi community in the Sinjar district may have contributed to recording relatively low obesity rates compared to other Iraqi cities. The study also confirms a close relationship between increased fat mass and elevated inflammatory markers and metabolic disorders, reinforcing the role of obesity as a major risk factor for the spread of chronic diseases in the region.

1. Introduction

Obesity is an excessive or irregular buildup of body fat. It is a complex condition resulting from multiple factors, including genetics, nutrition, lifestyle, and environmental influences [1]. The prevalence of obesity has increased dramatically over the past four decades in almost all countries, and it is associated with the global problem of chronic disease and disability [2]. A recent report by the World Health Organization (WHO) states that the body mass index (BMI) for adults aged 20 and older is classified into several categories: underweight below 18.5, normal weight 18.5–24.9, pre-obesity 25.0–29.9, obesity class I 30.0–34.9, obesity class II 35.0–39.9, and obesity class III, 40.0 and above [3].

Diabetes mellitus (DM) results from hyperglycemia due to impaired insulin action/secretion or both. It includes two major categories: Type 1 DM (T1DM) and Type 2 DM (T2DM) [4]. According to reports from the International Diabetes Federation (IDF), DM affected 366 million adults in 2011, with 552 million cases projected by 2030. Over 90 % of cases are accounted for by T2DM [5], which is caused by impaired insulin receptor activity due to genetic and environmental factors [4]. A higher risk of this latter type is believed to be associated with BMI, and more than 80 % of individuals with T2DM are also obese. The reason for this link is unknown; however, some studies suggest that genetic factors may offer protection [6]. Poor glycemic control is often associated with widespread metabolic changes, including abnormal plasma insulin levels and impaired beta-cell function, which may be linked to the development of a range of diseases such as dyslipidemia, cardiovascular disease, and kidney disease [5].

DOI: https://doi.org/10.64943/ljmas.2026.040202

Studies indicate that obesity is a significant risk factor for dyslipidemia [7]. Conversely, another study reported that lower body weight is linked to improved lipid profiles [8]. In addition, obesity is believed to be closely associated with inflammation through the secretion of pro-inflammatory adipokines like leptin, interleukin-6 (IL-6), and tumour necrosis factor-α from adipose tissue [9]. However, studies have demonstrated mixed results regarding the relationship between obesity and inflammation, some depicting chronic low-grade and others severe inflammation. These contradictory results highlight the multifaceted nature of the association between obesity and inflammation and the importance of further studies.

The study aims at determining the incidence of obesity in the adult population living in the Sinjar District, the association of family history and physical activity with obesity, and the relationship between fat accumulation and the risk of developing chronic disorders, such as DM, hypertension, dyslipidemia, and heart disease, and the inflammatory process, after eliminating influencing factors.

2. Materials and Methods

2.1. Study design and subject selection

This study used a community-based cross-sectional approach to determine the obesity prevalence in northwest Iraq's Sinjar District. Additionally, biochemical and anthropometric markers between obese and non-obese people were compared using a case-control analytical method. Data were collected between February and April 2025. In order to guarantee proportionate representation of every neighborhood in the district, 240 adult volunteers were chosen for the study using stratified random sampling at the household level. To ensure data independence, one participant was chosen at random from each household. Anthropometric measures and structured interviews were conducted with the participants. Additionally, blood samples were taken for biochemical examination.

2.2. Exclusion criteria

In this study, individuals at risk for inflammation or weight gain due to health conditions, habits, or medications were excluded. These included those with cancer, chronic inflammatory conditions, pregnant women, those with hormonal imbalances, or those taking medications such as antidepressants, steroids, anti-inflammatory herbs and antibiotics.

2.3. Sample Collection

Each participant provided 5 ml of blood in single-use gel tubes. After 20 min of clotting, samples were centrifuged at 4,000 rpm for 10 min. Serums were then stored at -20°C for biochemical analyses. The longest storage time was 65 days for the initial 15 samples, and the rest were stored for less time based on their sampling dates within a total collection period of about two months.

2.4. Serum determination of glucose and lipid profile

Fasting blood sugar (FBS) and lipid levels, including total cholesterol (TC), triglycerides (TG), and high-density lipoprotein (HDL), were measured using BIOLABO enzyme analysis kits. Determination of FBS and TC was performed by oxidation using the Trinder reaction [10], while TG was determined through a hydrolysis reaction that was subsequently concluded with the Trinder reaction [11,10]. Following the method described by Mohsen Ibrahim et al. (2013), HDL was determined, and cholesterol was measured in the upper liquid after centrifugation using a TC reagent [12]. The manufacturer's instructions for the reagent solutions were followed, and measurements were performed using a UV-Vis spectrophotometer at a wavelength of 500 nm. Low-density lipoprotein (LDL) was calculated using the Friedewald method exclusively for samples with TG levels ≤ 400 mg/dL, and in other cases using direct quantitative enzymatic assays [13].

2.5. Serum determination of human IL-6

Measurement of IL-6 concentrations was performed using the ELK Biotechnology human ELISA kit, based on the sandwich enzyme immunoassay principle. Samples and reagents were added to microtiter plates pre-coated with anti-IL-6 antibodies. After processing, absorbance was measured at 450 nm. A standard curve was constructed using the standard solution concentrations and their absorbances. The sample concentrations were determined by comparing their optical densities to the curve, as shown in Figure 1.

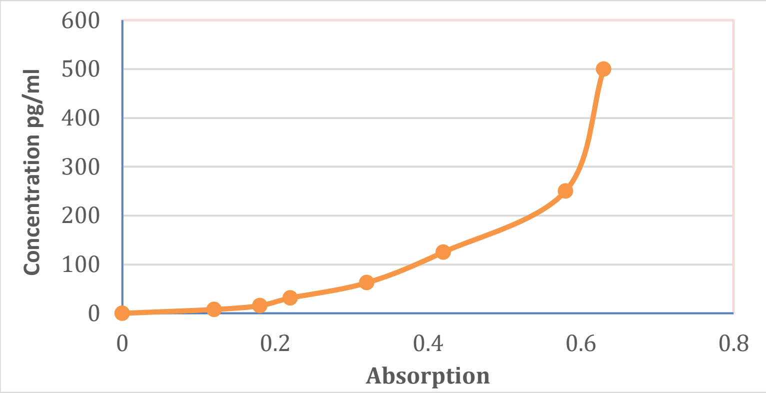

Figure 1. Standard curve of IL-6

2.6. Serum determination of C-reactive protein (CRP)

The ichroma™ CRP kit (Boditech Med, South Korea) was used to measure CRP levels using a sandwich immunoassay. The method analyses fluorescence intensity from antigen–antibody complexes bound to the detecting antibody, which is then processed by the ichroma™ device [14].

2.7. Statistical analysis

Data were presented as means with standard deviation (SD). Group comparisons were performed using ANOVA, with $p < 0.05$ considered significant. Data analysis was conducted using Microsoft Excel and SPSS (version 25.0, 2021).

3. Results and Discussion

3.1. Distribution of age groups and their relationship to weight

Figure 2 shows the distribution of participants across BMI categories within three age groups (18–35, 36–50, and over 50 years). In the normal weight group, the majority were young adults (18–35), whereas the overweight and obese groups showed a higher representation of middle-aged and older adults, indicating an increasing prevalence of higher BMI with age, indicating a growing prevalence for higher BMI with age. However, Table 1 shows no significant difference in mean age between groups (p = 0.056), allowing biochemical comparisons without age stratification.

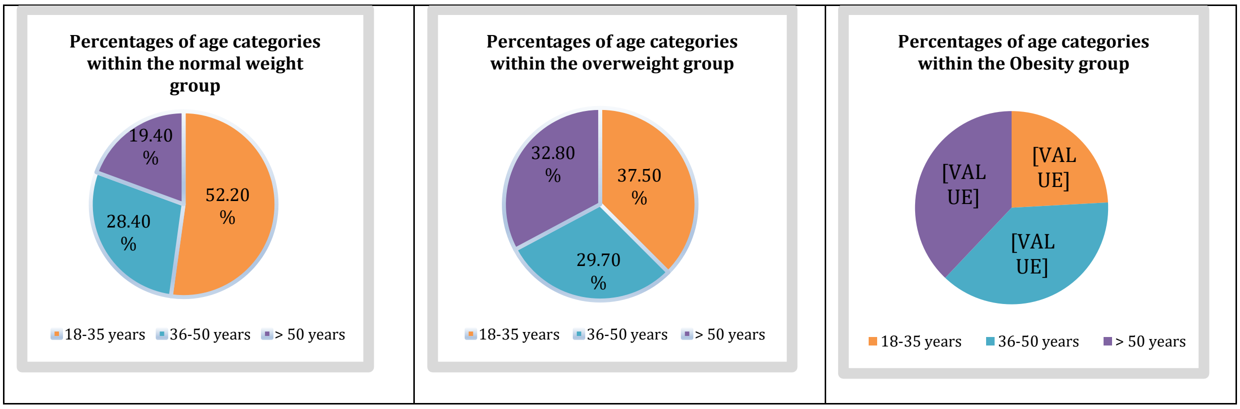

Figure 2. Distribution of samples according to age groups

Table 1. Comparison of the mean ages between the three study groups

Age group	Normal weight	Overweight	Obesity
Mean ± SD	35 ± 10.3	38.4 ± 10.4	40.7 ± 8.9
p-value		0.056	

Results are interpreted as mean ± SD, and the p-value corresponds to the ANOVA test.A statistical test was done after considering the p-value = 0.05

This direct relationship between age and weight gain is consistent with numerous studies worldwide. For example, in Mansoura, Egypt, the prevalence reached 33.3 % [15], while in Amirshahr, Iran, it was 44.2 % [16]. Similarly, in the United States, this rate has recently doubled from 22 % to 40 %, whereas China has reported even higher rates [17]. The etiology of these trends is multifactorial, as obesity in older adults results from a combination of genetic, biological, and societal factors, including ageing, hormones, immunity, and lifestyle [18]. Furthermore, retirement is associated with fat gain due to positive energy balance [19]. Muscle loss contributes to Insulin resistance (IR), also linked to fat accumulation [20]. Additionally, lower sex hormone levels affect fat distribution by correlating with fat uptake and lipoprotein lipase (LPL) activity [21].

3.2. Prevalence of obesity and overweight

The primary objective of this study was to determine the prevalence of obesity, overweight, and normal weight in the Sinjar district (northwestern Iraq). As shown in Table 2 and Figure 3, which include the results of our study alongside the results of all studies that addressed obesity and overweight rates in Iraqi cities with an Arab majority, namely Baghdad [22], a national study [23], Basra [24], Babil [25], and Karbala [26]. The obesity rate in Sinjar (20.4 %) is clearly lower than in other cities, especially Baghdad (40.7 %) and Babil (40 %). In contrast, the overweight rate in Sinjar (35.8 %) falls within the average range compared to other studies. In the same table, our results' gender comparisons show that obesity is more prevalent among females than males, consistent with national studies and research from Basra and Karbala, whereas the Baghdad study reported higher rates among males.

Table 2. Prevalence of obesity, overweight, and normal weight in Sinjar compared to other Iraqi cities

Study	Category	Obesity (%)	Overweight (%)	Normal Weight (%)
Current study Sinjar (2025)	Overall	20.4	35.8	43.8
	Males	15.7	30.3	53.9
	Females	26.8	42.2	31.0
Baghdad (2022)	Overall	40.7	26.4	32.9
	Males	50.1	25.0	36.0
	Females	37.1	27.0	25.0
Nationwide in Iraq (2021)	Overall	33.9	31.8	30.8
	Males	25.9	33.1	36.2
	Females	43.0	30.3	24.5
Basrah (2012)	Overall	23.8	31.3	44.9
	Males	38.9	50.2	54.7
	Females	61.1	49.8	45.3
Babil (2015)	Overall	40.0	37.6	22.4
	Males	N/A	N/A	N/A
	Females	N/A	N/A	N/A
Karbala (2008)	Overall	29.3	31.4	33.6
	Males	22.2	N/A	N/A
	Females	37.2	N/A	N/A

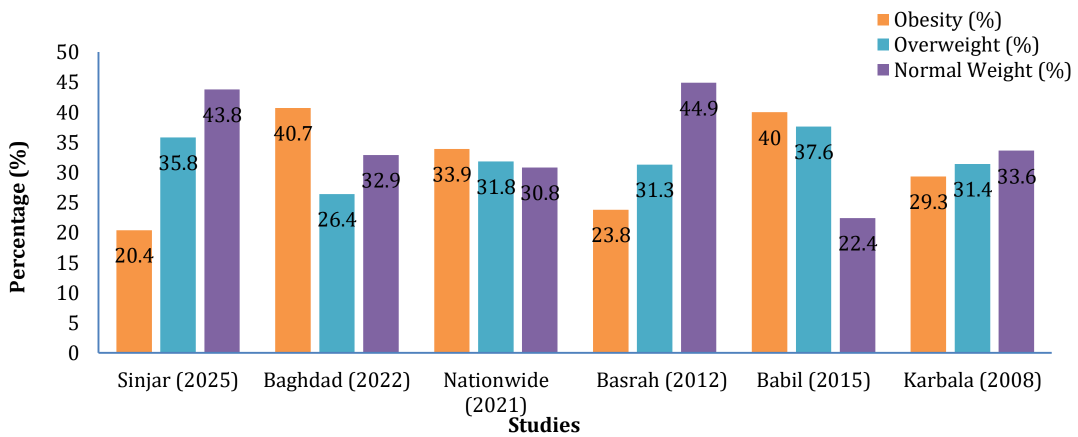

Figure 3. General comparison of obesity, overweight, and normal weight rates in Sinjar and some Iraqi regions

We were encouraged to choose the region because of its Yazidi majority population. This group is ethnically, socially, and culturally distinct from the Arab majority, and they were exposed to harsh conditions during the ISIS terrorist invasion. Accordingly, the difference between our results and those from other Iraqi cities may be attributed to behavioural, environmental, genetic, and lifestyle factors [27]. Nutritionally, healthy eating habits are often more prevalent in remote areas [28]. Indeed, a survey we conducted on the Sinjar district's residents showed that they consume whole grains, legumes, vegetables, seasonal fruits, moderate amounts of meat and plant proteins, and use local olive oil, reducing trans-fat intake, which is potentially associated with lower obesity rates. Genetically, fat mass and obesity-associated gene (FTO) mutations are linked to early and severe obesity across all ages; also, their variants, like rs9939609, increase calorie intake and preference for high-fat, high-carb foods [27]. Additional genes, including the leptin (LEP) and the melanocortin 4 receptor (MC4R), also affect the balance of energy and hunger. Polymorphic variations of MC4R (e.g., rs17782313) influence food intake, and LEP variations influence energy regulation [29]. In relation to the gender gap, women tend to be more obese than men, and this is in line with most of the local studies above; this may be due to biological and hormonal changes such as menopause and low estrogen levels, which predispose to visceral fat [30]. Social aspects are also linked to this trend, such as emotional eating because of stress [31]. This is also reflected in the sedentary lifestyle of women, such as being a housewife or working in jobs that require low physical activity [16]. The latter can be applied especially to women in the Sinjar community.

3.3. The relationship of the three study groups to physical inactivity, family history, and some common diseases

Table 3 provides a summary of lifestyle factors, family history, and chronic diseases by BMI. There is a significant difference ($p < 0.05$) in the obese group regarding physical inactivity and a family history of obesity. Likewise, obese participants had significantly more chronic diseases, such as hypertension, heart disease, lipid disorders, and T2DM, than the overweight or normal-weight groups. Such results indicate a significant relationship between high BMI and high metabolic and cardiovascular risk. The results in Table 4, also shown in Figure 4, which compares fasting blood glucose and blood lipid levels among the three BMI groups, are consistent with the previous table regarding the proportion of individuals with diabetes and dyslipidemia. Thus, Table 4 indicated that there were significant differences in the levels of these biomarkers among the three study groups, all of which were statistically significant ($p < 0.05$), as follows: FBS levels increased progressively from normal weight to overweight and peaked in obese participants, reflecting a clear relationship between elevated BMI and glucose dysregulation. Similarly, the lipid profile showed BMI-dependent alterations: TC, LDL, and TG levels increased, whereas HDL levels declined progressively from the normal weight to the overweight and obese groups. These findings underscore the combined impact of excess body weight on glycemic control and lipid metabolism.

Table 3. The relationship of overweight and obesity with some variables and chronic diseases

Variables and Chronic diseases No. (%)	Normal weight (n= 105)	Overweight (n= 86)	Obesity (n= 49)	*p*-value
Physical inactivity	64 (60.9 %)	55 (63.9 %)	41 (83.6 %)	**0.017**
Family history	16 (15.2 %)	17 (19.7 %)	25 (51.0 %)	**0.0001**
Hypertension	5 (4.7 %)	15 (17.4 %)	30 (61.2 %)	**0.0001**
Heart disease	5 (4.7 %)	0 (0 %)	5 (10.2 %)	**0.016**
Hyperlipidemia	6 (5.7 %)	11 (12.8 %)	10 (20.4 %)	**0.023**
T2DM	0 (0 %)	0 (0 %)	17 (34.7 %)	**0.0001**

Results are interpreted using the Chi-Square test; A statistical test was done after considering the p-value = 0.05

Table 4. Comparison of FBS and lipid levels between the three study groups

Parameters	Normal weight Mean ± SD	Overweight Mean ± SD	Obesity Mean ± SD	*p*-value
FBS (mg/dl)	98.3 ± 2.4	104.3 ± 2.7	181.7 ± 11.4	**0.0001**
TC (mg/dl)	178.7 ± 4.1	209.0 ± 7.4	218.2 ± 13.9	**0.003**
HDL (mg/dl)	63.2 ± 1.1	54.8 ± 1.2	47.2 ± 2.4	**0.0001**
LDL (mg/dl)	81.7 ± 3.3	129.5 ± 3.7	149.2 ± 7.2	**0.0001**
TG (mg/dl)	134.3 ± 6.6	182 ± 17.8	228.9 ± 26.7	**0.001**

Results are interpreted as mean ± SD, and the p-value corresponds to the ANOVA test; A statistical test was done after considering the p-value = 0.05

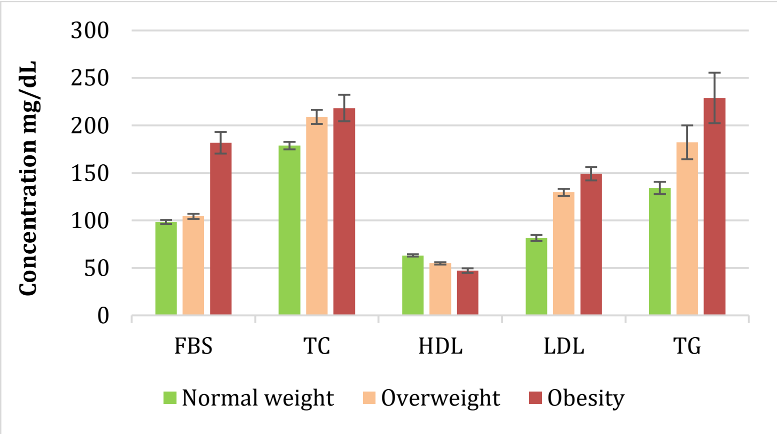

Figure 4. A graph showing FBS and lipid levels among the three study groups

Our findings are consistent with some studies. For instance, a cross-sectional survey among 646 volunteers in Pakistan found that weight, waist-to-hip ratio and daily exercise had associations [32]. Similarly, Cleven et al. (2020) conducted a systematic review that revealed that greater physical activity is associated with reduced risk of obesity [33]. Therefore, exercise should be encouraged at an early age to promote healthy growth. On the topic of the family history of obesity, research shows that genetics could be a key factor in weight gain, commonly associated with mutations in the *LEP* and *MC4R* genes [34]. According to this, research conducted in Italy revealed that family history of obesity, DM, and heart disease can predict early and late onset of obesity [35]. Notably, while genetics are influential, a recent study has shown that environmental and lifestyle factors can substantially reduce obesity risk even in individuals with high genetic predisposition [36].

As for the emergence of chronic diseases, some studies show that 65 % – 75 % of essential hypertension risk is due to increased visceral fat [37]. Mechanistically, increased body fat mass elevates blood volume through the renin-angiotensin-aldosterone system (RAAS). Angiotensin II and aldosterone inhibit insulin signalling by stimulating serine kinases and enhancing serine phosphorylation of insulin receptors, which reduces arterial relaxation, and contributing to hypertension [38].

In addition, studies indicate that up to 85 % of adults with T2DM are overweight or obese [39]. Pathophysiologically, several sequential mechanisms contribute to the development of T2DM in obese individuals, including decreased secretion of hormones such as adiponectin, while the release of pro-inflammatory proteins increases due to abdominal obesity, reducing insulin response. This cascade is often associated with elevated free fatty acids (FFAs) secretion from visceral fat, which in turn reduces the activity of carbohydrate pathway enzymes and impairs β-cell function by disrupting insulin secretion pathways [40]. Despite the above, the exact mechanism by which fat contributes to the development of T2DM remains unclear, as not all obese people develop it. In this regard, geneticists have found that 48 nucleotides may be responsible for this association [6]. Consequently, it suggests that although visceral adiposity is a powerful metabolic stressor via the pro-inflammatory and FFA pathways, it may not be an independent cause of T2DM. It is suggested that the phenomenon of obesity transforming into clinical T2DM is probably regulated by a certain metabolic threshold, which is predetermined by the unique genetic environment of an individual.

Our study on the relationship between BMI and blood lipid levels is consistent with Mishra et al. (2023), who reported an association between obesity and elevated TC and LDL levels among young adults in Delhi [41]. Similarly, an Italian study found that individuals with obesity exhibited reduced HDL alongside elevated LDL and TG levels [42]. However, these findings are contradicted by an Indonesian study conducted by Setyawati and Lasroha (2021), which revealed that the majority of obese patients maintained normal levels of TC, LDL, and TG [43]. Such results can be explained by the obesity paradox or the particular distribution of the visceral fat as opposed to the total body mass. This is in agreement with the findings of Hussain et al. (2019), who indicated that genetic factors in some South Asian populations cause visceral obesity and lipid disorders despite having a low BMI, thereby highlighting the limitations of BMI as a global predictor of lipid disorders [44].

The underlying basis for our findings is twofold. Biochemically, IR in obesity increases FFAs in the blood, which stimulates the liver to produce very-low-density lipoprotein (VLDL), resulting in elevated TG and LDL levels and decreased HDL levels. Also, the decline in LPL activity is associated with slow clearance of TG from plasma [45]. Genetically, mutations in the *PCSK9* gene contribute to its increased activity, which is linked to the degradation of LDL receptors and consequently, increased levels of it [46].

3.4. The difference in IL-6 and CRP levels between the three groups studied (Normal weight, Overweight, and Obese)

Finally, regarding the levels of inflammatory markers, the data in Table 5 and shown in Figure 5 indicate a significant increase (p = 0.0001) in IL-6 and CRP levels across the study groups, as they increased gradually from normal weight to overweight to obese.

Table 5. Comparison of IL-6 and CRP levels between the three study groups

Parameters	Normal weight Mean ± SD	Overweight Mean ± SD	Obesity Mean ± SD	*p*-value
IL-6 (pg/mL)	8.7 ± 0.44	12.1 ± 1.07	23.1 ± 1.90	0.0001
CRP (mg/L)	3.1 ± 0.27	6.9 ± 0.56	15.1 ± 2.04	0.0001

Results are interpreted as mean ± SD, and the p-value corresponds to the ANOVA test; A statistical test was done after considering the p-value = 0.05

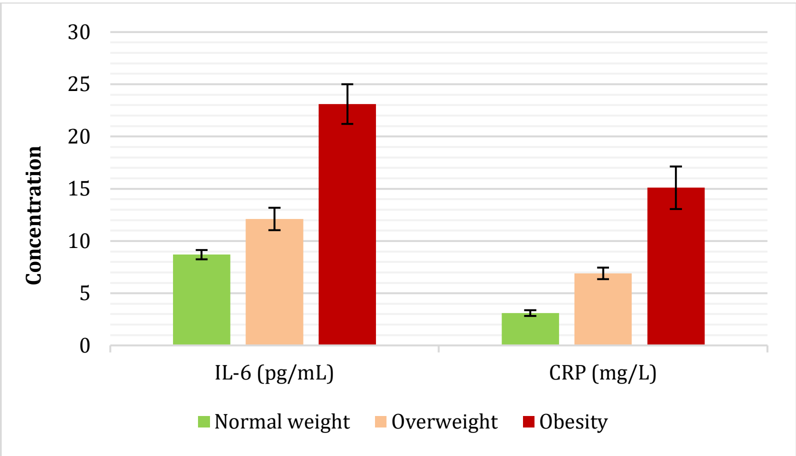

Figure 5. A graph showing IL-6 and CRP levels among the three study groups

Our findings align with some previous studies, while others reported no significant relationship. For instance, a British study found a strong association between IL-6 concentration and BMI in older adults [47]. Similarly, another study linked CRP levels to BMI, noting that its levels were significantly higher in overweight women in Montenegro [48]. It is hypothesized that hypoxia in deep adipose tissue is associated with macrophage invasion, promoting the generation of inflammatory mediators that induce local inflammation. Furthermore, excess FFAs in obesity activate a proinflammatory serine kinase cascade, triggering IL-6 secretion from fat cells and stimulating liver cells to produce CRP [9].

Ultimately, the severity of inflammation associated with obesity is influenced by multiple factors, including genetic predisposition and the ratio of visceral fat [49].

4. Limitation

Although our study has been useful in providing insights into obesity in the Sinjar district, we have to admit that it has some practical limitations. To begin with, we employed a cross-sectional design that helped us to set a baseline on prevalence as well as correlations within this region; yet, we cannot ascertain a direct cause-and-effect relationship. Secondly, our study sample was limited to 240 participants. This was a compromise to ensure the accuracy of the biochemical tests, given the technical and logistical challenges we faced in the fieldwork. Furthermore, a limitation of this study is the lack of renal function assessment (e.g., creatinine and urea). However, potential confounding effects were minimized by excluding participants with known chronic inflammatory or systemic diseases, such as chronic kidney disease (CKD), which could independently elevate IL-6 and CRP levels. Finally, we used structured interviews to collect lifestyle information because they were not available in the region due to the lack of electronic health records. We understand that this technique relies on the memory of the participants and that might bring about recall bias."

5. Conclusions

This research has found that being overweight was more common in Sinjar as compared to other parts of Iraq, with obesity being lower. There was a significant role of physical inactivity and positive family history in the development of obesity, and a direct correlation between the increase in the BMI and the inflammatory conditions and chronic diseases such as hyperlipidemia, T2DM and cardiovascular disease.

Author Contribution

This study was designed, and its biochemical measurements were performed by Dr Ashraf R Salem Al-Safar. Scientific and linguistic review and statistical analysis were conducted by Mr Mohanad Mowfik Sehree. Both authors read and approved the final version of the manuscript.

Acknowledgments

We extend our sincere thanks to all the Sinjar families who volunteered to provide information and blood samples for their families. We also express our appreciation to the University of Mosul Research Centre for facilitating laboratory measurements for this research project.

Competing Interests

The authors declare that there is no conflict of interest.

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انتشار السمنة بين البالغين في قضاء سنجار، العراق: علاقتها بالأمراض المزمنة الشائعة وبعض المؤشرات الحيوية الالتهابية

الملخص

تُعرف السمنة بأنها تراكم مفرط للدهون في الجسم ناتج عن اختلال التوازن بين السعرات الحرارية المتناولة والمستهلكة. هدفت هذه الدراسة إلى تحديد معدل انتشار السمنة وزيادة الوزن بين البالغين في قضاء سنجار – العراق، ومقارنة هذه المعدلات بنظيراتها في مدن عراقية أخرى. كما سعت إلى التحقق من العلاقة بين تراكم النسيج الشحمي وارتفاع مؤشرات الالتهاب الحيوية وتطور بعض الأمراض المزمنة. ويُذكر أن المجتمع الإيزيدي في سنجار يتميز بخصوصية عِرقية وثقافية ودينية تختلف عن الغالبية العربية في البلاد.

أُجريت هذه الدراسة المقطعية على عينة عشوائية ضمت 240 بالغاً تتراوح أعمارهم بين 18 و80 عاماً، وذلك خلال الفترة من شباط إلى نيسان 2025. جرى قياس مستويات الغلوكوز والدهون في مصل الدم، إضافة إلى الإنترلوكين-6 (IL-6) والبروتين التفاعلي C (CRP)، بوصفها مؤشرات حيوية مرتبطة بالسمنة.

أظهرت النتائج أن معدلات السمنة في سنجار كانت أقل مقارنةً بمدن عراقية أخرى، في حين ظلت نسب زيادة الوزن متقاربة مع المتوسطات العامة المسجلة في الدراسات السابقة. كما تبين وجود ارتباط قوي بين مؤشر كتلة الجسم (BMI) وتطور داء السكري وأمراض القلب والأوعية الدموية، مع تسجيل زيادات ذات دلالة إحصائية ($p < 0.05$) في مستويات الغلوكوز، والبروتين الدهني منخفض الكثافة (LDL) ، والدهون الثلاثية (TG) ، ومؤشرات الالتهاب لدى الأفراد المصنفين ضمن فئتي زيادة الوزن والسمنة.

وخلصت الدراسة إلى أن الخصوصية العرقية والثقافية للمجتمع الإيزيدي في قضاء سنجار قد تكون أسهمت في تسجيل معدلات سمنة أقل نسبياً مقارنةً بمدن عراقية أخرى. كما تؤكد النتائج وجود علاقة وثيقة بين زيادة الكتلة الدهنية وارتفاع مؤشرات الالتهاب والاضطرابات الاستقلابية، مما يعزز اعتبار السمنة عاملاً خطراً رئيساً في تفشي الأمراض المزمنة في المنطقة.

الكلمات المفتاحية: السمنة، قضاء سنجار، الأمراض المزمنة، حالة التهابية، إنترلوكين 6