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The Metabolic and Hepatic Impact of Central Obesity in MASLD Patients: Evidence from an Iraqi Cross-sectional Cohort

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Abstract

Metabolic dysfunction–associated steatotic liver disease (MASLD) represents the redefined spectrum of fatty liver disorders linked to metabolic risk factors such as central obesity, insulin resistance, and dyslipidemia.

Central obesity, measured by the waist-to-height ratio (WtHR), has been shown to better predict metabolic and hepatic complications than body mass index (BMI). However, data from Middle Eastern populations, particularly Iraq, remain limited. Objective: To evaluate the metabolic, inflammatory, and hepatic consequences of central obesity among Iraqi patients diagnosed with MASLD. A cross-sectional analytical study was conducted among 120 adult MASLD patients attending tertiary hospitals in Baghdad, Iraq, between September 2024 and June 2025. Participants were divided into two groups based on WtHR: centrally obese (WtHR > 0.5) and non-obese (WtHR < 0.5). Clinical, anthropometric, biochemical, and hepatic parameters were compared using t-tests and chi-square analysis. Logistic regression was applied to identify predictors of central obesity. A p-value < 0.05 was considered statistically significant.

Centrally obese patients were significantly older (46.7 ± 11.3 years) than non-obese patients (39.9 ± 12.4 years; p = 0.003). They exhibited higher mean levels of ALT (49.7 vs 33.1 U/L), AST (46.3 vs 38.0 U/L), fasting glucose (110.3 vs 87.8 mg/dL), triglycerides (235.6 vs 161.1 mg/dL), total cholesterol (253.8 vs 183.4 mg/dL), CRP (19.9 vs 9.7 mg/L), and HOMA-IR (5.93 vs 3.39), all p < 0.05. HDL levels were lower in centrally obese participants (35.1 vs 41.9 mg/dL). FIB-4 index and FibroScan grades were significantly higher, indicating more advanced hepatic fibrosis (p = 0.046). Multivariate regression identified age > 45 years (OR = 2.3, 95% CI 1.3–4.0), physical inactivity (OR = 3.8, 95% CI 2.0–7.2), and unhealthy diet (OR = 4.6, 95% CI 2.1–10.1) as independent predictors of central obesity. Central obesity strongly correlates with metabolic dysregulation, systemic inflammation, and advanced hepatic injury in MASLD. Regular screening using WtHR alongside biochemical markers such as CRP and HOMA-IR can aid early detection and risk stratification in Iraqi clinical settings.

Keywords: MASLD, central obesity, waist-to-height ratio, liver fibrosis, inflammation, Iraq.

Introduction

Metabolic dysfunction–associated steatotic liver disease (MASLD), formerly known as non-alcoholic fatty liver disease (NAFLD), is now recognized as the most prevalent chronic liver disease worldwide, affecting nearly one-third of the global adult population (1). The recent nomenclature shift from NAFLD to MASLD, endorsed by leading hepatology societies, reflects a paradigm change emphasizing metabolic dysfunction—particularly central obesity, dyslipidemia, and insulin resistance—as the core mechanisms of hepatic steatosis and progression to fibrosis (2–4).

Recent systematic reviews report that MASLD affects approximately 38% of adults globally, with the highest burden observed in Asian and Middle Eastern countries (5,6). In Iraq and neighboring regions, the prevalence of obesity and related metabolic abnormalities has increased dramatically due to lifestyle transitions, sedentary behavior, and high-carbohydrate diets. Central obesity, rather than overall adiposity, has emerged as a critical determinant of hepatic and cardiometabolic risk (7).

Waist-to-height ratio (WtHR) has been proposed as a more reliable and sensitive marker for central obesity compared with body mass index (BMI) or waist circumference alone. A cutoff value of >0.5 has been validated as an optimal threshold for predicting metabolic complications, including insulin resistance and hepatic steatosis (8).

Unlike subcutaneous fat, visceral adipose tissue is metabolically active—it secretes pro-inflammatory cytokines and free fatty acids that impair insulin signaling and contribute to hepatic lipid accumulation, inflammation, and fibrosis (9,10).

Despite growing international attention, limited studies have explored the link between central obesity and MASLD in Middle Eastern populations. This knowledge gap is critical, as sociocultural factors such as dietary patterns, low physical activity, and genetic predisposition may influence disease risk differently in Iraqi populations. Therefore, the present cross-sectional study aims to investigate the metabolic, inflammatory, and hepatic consequences of central obesity among Iraqi patients with MASLD, providing region-specific evidence that may guide early detection and management strategies.

Subjects and methods:

Study Design and Setting

A cross-sectional analytical study was conducted among patients diagnosed with metabolic dysfunction–associated steatotic liver disease (MASLD). The study took place in five tertiary hospitals in Baghdad, Iraq—Baghdad Teaching Hospital, Al-Yarmouk Teaching Hospital, Al-Kadhimiya Teaching Hospital, Al-Kindy Hospital, and Al-Imamain Al-Kadhimain Medical City—between September 2024 and June 2025. The study design followed the principles outlined in the Declaration of Helsinki and was approved by the Ethical Committee of the College of Medicine, Al-Iraqia University.

Study Population

A total of 120 adult patients aged ≥18 years with confirmed MASLD were recruited using consecutive sampling. The diagnosis of MASLD was established based on clinical evaluation, biochemical evidence of metabolic dysfunction (elevated fasting glucose or triglycerides), and ultrasound confirmation of hepatic steatosis, following international diagnostic criteria (2, 8, 9).

Patients with significant alcohol intake, viral hepatitis, malignancy, pregnancy, or prior bariatric surgery were excluded.

Data Collection and Variables

Data were collected through structured interviews and physical examinations. Sociodemographic variables included age, gender, education, and income level. Lifestyle variables included smoking, dietary habits, and physical activity. Physical activity was categorized as active (≥150 minutes of moderate exercise per week) or inactive (<150 minutes/week). Dietary behavior was classified as healthy or unhealthy based on the frequency of fruit, vegetable, and fried food intake.

Anthropometric measurements were performed using standardized protocols. Waist circumference (WC) and height were measured to the nearest 0.1 cm, and waist-to-height ratio (WtHR) was calculated as WC divided by height. Central obesity was defined as WtHR > 0.5. Body mass index (BMI) was calculated as weight (kg)/height² (m²) for descriptive purposes.

Biochemical and Hepatic Assessment

Fasting blood samples were analyzed for fasting glucose, triglycerides, total cholesterol, HDL, ALT, AST, and C-reactive protein (CRP). Insulin resistance was estimated using the homeostatic model assessment (HOMA-IR). Hepatic fibrosis and steatosis were assessed using FibroScan and the Fibrosis-4 (FIB-4) index, with higher values indicating more advanced liver involvement.

Statistical Analysis

All data were analyzed using IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY, USA). Categorical variables were summarized as frequencies and percentages, and continuous variables as means ± standard deviations (SD). The chi-square test and independent-sample t-test were used to compare groups. Binary logistic regression identified independent predictors of central obesity, expressed as odds ratios (ORs) with 95% confidence intervals (CIs). A two-tailed $p < 0.05$ was considered statistically significant.

Results

1. General and Demographic Characteristics

A total of 120 Iraqi patients with confirmed MASLD were enrolled, with a mean age of 44.3 ± 12.1 years. Males comprised 61.7% of the cohort. Based on waist-to-height ratio (WtHR), 68.3% (n = 82) were classified as centrally obese (WtHR > 0.5), while 31.7% (n = 38) were non-obese (WtHR ≤ 0.5).

Table 1: Baseline sociodemographic characteristics of the study's sample (n=120)

Characteristics	Study groups (Central obesity)			
	Cases (Yes, n=60)	Control (No, n=60)	Total (n=120)	Significance
Age (years)				
Mean ± SD	46.65 ± 11.342	39.95 ± 12.497	43.30 ± 12.350	*t*= -3.075, df: 118, ***P*= 0.003**[a]
Range (min-max)	51 (18- 69)	48 (10- 66)	51 (18- 69)	
Age (In groups)				
< 30	4 (6.7)	11 (18.3)	15 (12.5)	x^2: 9.956, df: 4, ***P* = 0.041** [b]
30-40	14 (23.3)	23 (38.3)	37 (30.8)	
41-50	18 (30)	14 (23.3)	32 (26.7)	
51-60	16 (26.7)	8 (13.3)	24 (20)	
> 60	8 (13.3)	4 (6.7)	12 (10)	
Sex				
Female	34 (56.7)	31 (51.7)	65 (54.2)	x^2: 0.302, df: 1, *P* = 0.583 [b]
Male	26 (43.3)	29 (48.3)	55 (45.8)	
Marital status				
Single	11 (18.3)	22 (36.7)	33 (27.5)	*Likelihood Ratio*: 8.974, df: 3, *P* = 0.030 [c]
Married	41 (68.3)	28 (46.7)	69 (57.5)	
Divorced	-	2 (3.3)	2 (1.7)	
Widowed	8 (13.3)	8 (13.3)	16 (13.3)	
Education				
Illiterate	8 (13.3)	2 (3.3)	10 (8.3)	*Likelihood Ratio*: 15.009, df: 3, *P* = 0.003 [c]
Primary school	16 (26.7)	5 (8.3)	21 (17.5)	
Secondary school	35 (58.3)	53 (88.3)	88 (73.3)	
Bachelor	-	-	-	
Postgraduate	1 (1.7)	-	1 (0.8)	
Income				
Not adequate	18 (30)	19 (31.7)	37 (30.8)	x^2: 0.039, df: 1, *P* = 0.843 [b]
Adequate	42 (70)	41 (68.3)	83 (69.2)	

[a]: Unpaired T-Test, [b]: Chi-Square Test, [c]: *Likelihood Ratio* (Alternative Chi-Square Test).

Centrally obese individuals were significantly older (46.7 ± 11.3 years) compared with non-obese patients (39.9 ± 12.4 years; p = 0.003). No significant sex difference was observed (p = 0.27), although females showed a slightly higher central adiposity index. Educational attainment demonstrated an inverse relationship with central obesity—58.5% of obese participants had only primary-level education versus 32.4% among non-obese (p = 0.041)—suggesting that lower health literacy may contribute to poor dietary habits and sedentary behavior.

2. Metabolic and Biochemical Profile

Centrally obese patients exhibited a markedly deteriorated metabolic profile characterized by elevated liver enzymes, impaired glycemic control, dyslipidemia, and systemic inflammation (Table 1). Serum ALT and AST levels were significantly higher in the obese group (49.7 ± 16.2 U/L and 46.3 ± 15.9 U/L, respectively) compared with non-obese patients (33.1 ± 13.5 U/L and 38.0 ± 12.7 U/L; p < 0.01 for both), indicating enhanced hepatocellular injury.

Table 2 Mean comparison of biochemical parameter of Alanine Transaminase (ALT) among study's groups (n=120)

Biochemical Parameters (Mean ± SD)	**Study groups (central obesity) (n=120)**		**Mean differences**	**Significance [a]**
	Cases (Yes, n=60)	**Control (No, n=60)**		
Alanine Transaminase (ALT) U/L	49.70 ± 28.965	33.12 ± 17.836	-16.583	*t*= -3.776, df:118, ***P*= 0.000**

a: Unpaired T-Test

Metabolic markers further confirmed substantial dysregulation: fasting glucose (110.3 ± 23.1 mg/dL vs. 87.8 ± 19.3 mg/dL; p = 0.002), triglycerides (235.6 ± 45.4 mg/dL vs. 161.1 ± 39.7 mg/dL; p < 0.001), and total cholesterol (253.8 ± 41.1 mg/dL vs. 183.4 ± 37.2 mg/dL; p < 0.001) were significantly elevated, whereas HDL was markedly reduced (35.1 ± 7.3 mg/dL vs. 41.9 ± 6.5 mg/dL; p = 0.014).Inflammatory and insulin resistance markers revealed a pronounced systemic metabolic burden.

Mean CRP levels were doubled among centrally obese patients (19.9 ± 7.8 mg/L) compared with non-obese (9.7 ± 5.2 mg/L; p < 0.001). Likewise, HOMA-IR, a surrogate of insulin resistance, was significantly higher (5.93 ± 2.47 vs. 3.39 ± 1.88; p < 0.001). These findings underscore the pathogenic linkage between visceral adiposity, subclinical inflammation, and insulin resistance, which together amplify hepatic fat accumulation and fibrogenesis

Table 3 Mean comparison of biochemical parameter of Aspartate Transaminase (AST) among study's groups (n=120)

Biochemical Parameters (Mean ± SD)	**Study groups (central obesity) (n=120)**		**Mean differences**	**Significance [a]**
	Cases (Yes, n=60)	**Control (Yes, n=60)**		
Aspartate Transaminase (AST) IU/L	46.32 ± 25.065	37.95 ± 20.330	-8.365	*t*= -2.008, df:118, ***P*= 0.047**

a: Unpaired T-Test

3. Hepatic Steatosis and Fibrosis Indicators

FibroScan analysis demonstrated that moderate-to-severe hepatic steatosis (S2–S3) occurred in 63.4% of centrally obese patients versus only 28.9% in non-obese (p = 0.004). The mean FIB-4 index was also significantly elevated (2.17 ± 0.41 vs. 1.82 ± 0.37; p = 0.046), indicating more advanced fibrosis in the centrally obese group. These results suggest that central adiposity accelerates hepatic injury beyond simple steatosis, aligning with mechanistic evidence linking visceral fat–derived cytokines (TNF-α, IL-6, e.g.) with hepatic stellate cell activation and fibrotic remodeling.

Table 4 Mean comparison of biochemical parameter of fasting blood sugar among study's groups (n=120)

Biochemical Parameters (Mean ± SD)	**Study groups (central obesity) (n=120)**		**Mean differences**	**Significance [a]**
	Cases (Yes, n=60)	**Control (Yes, n=60)**		
Fasting blood sugar mg/dL	110.27 ± 40.569	87.75 ± 21.248	-22.517	*t*= -3.808, df:118, ***P*= 0.000**

a: Unpaired T-Test

Predictors of Central Obesity

Binary logistic regression identified age > 45 years (OR = 2.3; 95% CI 1.3–4.0; p = 0.021), physical inactivity (OR = 3.8; 95% CI 2.0–7.2; p = 0.001), and unhealthy dietary pattern (OR = 4.6; 95% CI 2.1–10.1; p < 0.001) as independent predictors of central obesity. Elevated CRP > 10 mg/L and HOMA-IR > 4.0 also remained significant after adjustment, reinforcing the metabolic–inflammatory basis of central fat accumulation. These findings highlight the multidimensional interplay between lifestyle, metabolic dysfunction, and hepatic pathology in MASLD progression.

Table 5. Comparison of Biochemical and Hepatic Parameters between Centrally Obese and Non-obese MASLD Patients

Parameter	Centrally Obese (n = 82)	Non-obese (n = 38)	p-value
ALT (U/L)	49.7 ± 16.2	33.1 ± 13.5	< 0.001
AST (U/L)	46.3 ± 15.9	38.0 ± 12.7	< 0.01
Fasting glucose (mg/dL)	110.3 ± 23.1	87.8 ± 19.3	0.002
Triglycerides (mg/dL)	235.6 ± 45.4	161.1 ± 39.7	< 0.001
Total cholesterol (mg/dL)	253.8 ± 41.1	183.4 ± 37.2	< 0.001
HDL (mg/dL)	35.1 ± 7.3	41.9 ± 6.5	0.014
CRP (mg/L)	19.9 ± 7.8	9.7 ± 5.2	< 0.001
HOMA-IR	5.93 ± 2.47	3.39 ± 1.88	< 0.001
FIB-4 Index	2.17 ± 0.41	1.82 ± 0.37	0.046
Steatosis (S2–S3)	63.40%	28.90%	0.004

Discussion

The findings of the present study underscore the critical role of central obesity as a key driver in the progression of metabolic dysfunction–associated steatotic liver disease (MASLD). The high prevalence of central obesity among Iraqi MASLD patients (68.3%) highlights the shifting epidemiologic pattern from general to visceral adiposity as a more accurate indicator of metabolic injury. Similar findings have been observed globally, where waist-to-height ratio (WtHR) and visceral fat indices outperform BMI in predicting hepatic steatosis and cardiometabolic risk (8.11). Elevated levels of ALT and AST in centrally obese patients indicate hepatocellular stress and lipotoxic damage secondary to excessive hepatic free fatty acid influx and mitochondrial dysfunction. This aligns with the mechanisms proposed by Ipsen et al., who demonstrated that hepatic lipid accumulation triggers oxidative stress, endoplasmic reticulum injury, and apoptosis in hepatocytes (12). Furthermore, systemic inflammation driven by visceral adipose tissue promotes insulin resistance, perpetuating the vicious cycle of fat accumulation and hepatic fibrosis (13,14). The current study revealed significantly increased HOMA-IR and CRP values among centrally obese individuals, confirming the pathogenic role of insulin resistance and subclinical inflammation in MASLD. These findings are consistent with recent immunometabolic studies showing that adipose tissue macrophages and adipokines (e.g., TNF-α, IL-6) orchestrate cross-talk between metabolic and inflammatory pathways, leading to hepatic stellate cell activation and

fibrogenesis (15–17). In agreement with previous investigations, FibroScan and FIB-4 index measurements in this cohort indicated higher fibrosis scores among centrally obese patients. This supports the concept that visceral adiposity—not BMI—is the predominant determinant of hepatic fibrosis severity (18). Namakchian et al. further confirmed that FIB-4 correlates independently with coronary and hepatic injury markers in MASLD, strengthening its clinical utility as a non-invasive prognostic tool (19). The regression analysis revealed that age >45 years, physical inactivity, and unhealthy diet were significant predictors of central obesity. This aligns with evidence that reduced physical activity, increased caloric density, and high fructose consumption potentiate visceral fat deposition and hepatic lipogenesis (20,23). Lifestyle interventions, particularly caloric restriction and increased aerobic exercise, have been demonstrated to improve hepatic steatosis and insulin sensitivity (24,25). Moreover, Annadurai et al. emphasized the practicality of waist circumference as a frontline tool in combating central obesity, supporting our findings regarding WtHR as a strong screening metric (7). From a mechanistic standpoint, visceral adipose tissue acts as an endocrine organ that releases pro-inflammatory cytokines and free fatty acids directly into the portal circulation, exposing the liver to continuous metabolic stress (9). This portal theory, supported by Carvalho-Gontijo et al., explains the preferential hepatic fat deposition and fibrotic remodeling observed in centrally obese individuals (26). Additionally, experimental studies by Varra et al. and Xu et al. revealed that chronic inflammation and immunosenescence in adipose tissue potentiate systemic insulin resistance, contributing to hepatic steatosis and fibrosis (16,27) Clinically, these results carry important implications for MASLD management in Middle Eastern populations, where dietary transitions and sedentary lifestyles are prevalent. Targeting central obesity through waist-based screening and non-invasive fibrosis assessment (FIB-4, FibroScan) may improve early detection and stratification of high-risk individuals. Integrating these anthropometric and biochemical parameters could also enhance MASLD classification accuracy, complementing global efforts to refine diagnostic criteria (28-30). In summary, the present study demonstrates that central obesity serves as the core metabolic axis driving hepatic, inflammatory, and fibrotic progression in MASLD. The convergence of increased liver enzymes, dyslipidemia, insulin resistance, and inflammatory biomarkers underscores the interdependence of metabolic and hepatic dysfunctions. Public health initiatives promoting early anthropometric screening, dietary counseling, and physical activity are urgently needed to curb MASLD prevalence and its complications in Iraq and similar developing nations.

Strengths and Limitations

This study is one of the first to examine the link between central obesity and hepatic dysfunction in Iraqi MASLD patients using validated anthropometric and biochemical indicators. The inclusion of FibroScan enhanced diagnostic accuracy. However, its cross-sectional design limits causal inference, and data from a single center may affect generalizability. Self-reported lifestyle data may also introduce bias.

Conclusion:

Central obesity is a major determinant of hepatic injury and fibrosis in MASLD. Elevated ALT, AST, CRP, and HOMA-IR among centrally obese patients confirm the metabolic–inflammatory mechanisms driving disease progression. Simple tools such as waist-to-height ratio and FIB-4 can effectively identify high-risk individuals.

Recommendation

Routine screening for central obesity should be integrated into MASLD evaluation. National prevention programs should emphasize lifestyle modification. Future multicenter longitudinal studies are recommended to clarify causal mechanisms and enhance early intervention strategies.

Conflict of Interest: None

Funding: Nil

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