Original Article

Impact of Central Obesity on the Clinical and Biochemical Profile of Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD)

Central Obesity Associated Steatotic Liver Disease

Ahmed M Shaker and Bilal Natiq Nuaman

ABSTRACT

Objective: To assess the association between central obesity (waist-to-height ratio >0.5) and liver fibrosis, steatosis, and inflammatory biomarkers in metabolic dysfunction-associated steatotic liver disease patients.

Study Design: A case-control study

Place and Duration of Study: This study was conducted at the Department of Medicine, College of Medicine, AL-Iraqia of University, Iraq from 15th March 2024 to 30th May 2025.

Methods: A case-control study was conducted on 120 adults diagnosed with metabolic dysfunction-associated steatotic liver disease in Baghdad. Participants were divided into centrally obese (n=60) and non-obese (n=60) groups. Clinical and biochemical parameters including liver enzymes (alanine aminotransferase, aspartate aminotransferase), C-reactive protein, homeostatic model assessment for insulin resistance and Fibro scan-based fibrosis staging were compared.

Results: Centrally obese patients showed significantly elevated alanine aminotransferase (49.7 vs. 33.1 U/L, $p<0.001$), C-reactive protein (19.9 vs. 9.7 mg/L, $p<0.001$), and homeostatic model assessment for insulin resistance (5.93 vs. 3.39, $p<0.001$). Fibrosis grades ≥F2 and steatosis stages ≥ S2 were significantly more frequent among the obese group ($p<0.01$).

Conclusion: Central obesity is significantly associated with worsened liver fibrosis and metabolic derangement in metabolic dysfunction-associated steatotic liver disease patients. These findings support the utility of waist to hip ratio as a clinical screening tool in metabolic dysfunction-associated steatotic liver disease management.

Key Words: Central obesity, Metabolic dysfunction-associated steatotic liver disease, Liver fibrosis, C-reactive protein, Homeostatic model assessment for insulin resistance, Fibro Scan

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INTRODUCTION

Metabolic dysfunction–associated steatotic liver disease (MASLD) is a redefined clinical entity that replaces the historical term non-alcoholic fatty liver disease (NAFLD), aiming for semantic clarity and pathophysiologic precision. NAFLD, once the most prevalent chronic liver disease worldwide, affects over one-third of adults and is closely linked to metabolic syndrome, central obesity, insulin resistance, and type 2 diabetes mellitus.[1-3]

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However, the term NAFLD has been criticized for its exclusionary definition based on alcohol intake and for failing to highlight metabolic risk factors, especially in lean individuals.[2,3]

In 2023, the Liver Disease Nomenclature Consensus Group introduced MASLD to more accurately describe hepatic steatosis associated with metabolic dysfunction.[2,4] The diagnosis requires hepatic steatosis plus at least one cardiometabolic risk factor such as obesity, dyslipidemia, hypertension, or type 2 diabetes.[5,6] Although MASLD overlaps with NAFLD in up to 99% of cases, it provides a clearer, mechanism-based framework emphasizing metabolic risk and cardiovascular relevance.[7-9]

The global prevalence of MASLD is rapidly increasing due to aging, sedentary lifestyle, poor diet, and obesity.[10] Among individuals with type 2 diabetes, prevalence exceeds 65%, peaking in the Middle East (71.2%) and lowest in Africa (53.1%)..[11] In the United States, MASLD cases rose by 138% between 1988 and 2018, surpassing obesity growth rates[12], reflecting a growing clinical and economic burden.[13]

MASLD is associated with elevated risks of all-cause, cardiovascular, and liver-related mortality.[14] Mortality is further aggravated in those with metabolic alcohol-associated liver disease (Meted).[15] In acute settings, such as myocardial infarction, MASLD correlates with obesity, diabetes, chronic kidney disease, and higher mortality.[16]

Given central obesity's pivotal role, early identification using simple anthropometric measures is crucial. Waist-to-height ratio (WtHR) has emerged as a practical, reliable index for central adiposity and metabolic risk, outperforming BMI and waist circumference [21, 22]. This study investigates the impact of central obesity, assessed via WtHR, on the clinical and biochemical profiles of MASLD patients, and evaluates its predictive value for disease severity and progression.

METHODS

This multicenter, hospital-based case–control study was conducted at in five tertiary hospitals in Baghdad, Iraq (Baghdad Teaching Hospital, Medical City, Gastroenterology & Hepatology Hospital, Al-Yarmouk, and Al-Numen) from 15th March 2024 to 30th May 2025. All centers followed unified diagnostic and laboratory standards. Adults ≥18 years with ultrasonographic evidence of hepatic steatosis were included. Central obesity was classified by waist-to-height ratio (WtHR): cases: WtHR >0.50 and controls WtHR <0.50. Participants with other liver diseases, pregnancy, or excessive alcohol intake (>140–350 g/week for females; >210–420 g/week for males) were excluded. Convenience sampling was used. Power analysis (α = 0.05, 80% power) estimated ≥60 participants per group; 120 subjects were enrolled equally (60/60).

Data were obtained via a standardized form including: Demographics: age, sex, marital status, education, income. Lifestyle: smoking, physical activity, diet quality. Medical History: diabetes, hypertension, malignancy. Anthropometry: waist circumference, height, WtHR. Clinical Sign: neck acanthosis nigricans (insulin resistance marker). Biochemistry: ALT, AST, glucose, triglycerides, HDL-C, CRP, insulin (HOMA-IR=insulin×glucose/405). Fibrosis Indices: FIB-4=(age×AST)/(platelets×√ALT) and FibroScan® stiffness (kPa). Imaging: Ultrasound grading (normal to cirrhosis).

MASLD was defined per 2023 Liver Disease Nomenclature Consensus criteria: hepatic steatosis plus ≥1 metabolic risk factor (obesity, hypertension, dyslipidemia, or diabetes) excluding secondary causes. Cases with excess alcohol intake were classified as MetALD and excluded. Analyses were performed using SPSS-27. Independent *t*-tests and χ^2 or likelihood-ratio tests assessed between-group differences, with $p<0.05$ considered significant.

RESULTS

The mean age 43.3±12.35 years; cases were significantly older (46.65±11.34 vs. 39.95±12.50 years, $p = 0.003$). Age distribution also differed ($\chi^2 = 9.956$, $p = 0.041$). Females predominated (54.2%), with no significant sex difference between groups ($p> 0.05$). Marital status varied significantly (p=0.030): married participants were more frequent in cases (68.3% vs. 46.7%), while single and divorced were higher in controls. Educational level differed (p=0.003); illiterate and primary education were more common among cases, while secondary education predominated in controls. Income adequacy showed no significant difference ($p> 0.05$) [Table 1).

Notable lifestyle differences were evident between groups (Fig. 1). Half of the centrally obese participants reported no physical activity, and 31.7% engaged only in light exercise, whereas 56.7% of controls practiced moderate to vigorous activity, highlighting the protective role of regular exercise. Unhealthy dietary patterns were more prevalent among centrally obese individuals (41.7% vs. 6.7%), while healthy or moderately healthy diets predominated in controls, emphasizing diet quality as a determinant of central adiposity. Smoking rates showed no significant variation between groups, indicating limited influence on central obesity. Clinically, neck hyperpigmentation - a visible marker of insulin resistance was significantly more frequent in centrally obese participants, reflecting higher metabolic burden and its utility as a simple screening indicator.

Clinical signs of insulin resistance particularly neck hyperpigmentation were significantly more prevalent in the central obesity group (61.7%) than in controls (11.7%) (χ^2=32.297, df=1, p<0.001). Conversely, absence of such signs was more common among controls (88.3% vs. 38.3%). Biochemically, ALT levels were significantly elevated in centrally obese participants compared to controls (49.70±28.97 vs. 33.12±17.84; t= –3.776, df=118, p<0.001), indicating greater hepatic involvement in central obesity. The mean level of fasting blood sugar was also significantly higher among central obesity group than that of central obesity free group (110.27±40.569 vs. 87.75±21.248) respectively with significant mean difference of -22.517 (t= -3.808, df:118, P=0.000). The mean level of lipids of triglycerides found to been significantly higher among cases group of study's sample in comparison to that in the controls group (235.63±66.350 vs. 161.07±23.989) respectively with significant difference of -74.567 (t= -8.187, df:118, P= 0.000). However, the mean level of lipids of high HDL was significantly lower among cases group of study's sample in comparison to that in the controls group (35.08±4.073 vs. 41.95±3.039) respectively with significant difference of 6.867 (t= 10.467, df:118, P= 0.000). The mean total cholesterol level was significantly higher in the central obesity group compared to controls (253.78±60.84 vs. 183.42±33.56; t = –7.844, df = 118,

p<0.001). Similarly, the inflammatory marker C-reactive protein (CRP) showed a marked elevation among centrally obese participants (19.90±25.36 vs. 9.70±5.38; t = –3.047, df = 118, p<0.001), reflecting enhanced systemic inflammation associated with central obesity. The mean Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) value was significantly higher in the central obesity group compared to controls (5.93±1.54 vs. 3.39±1.33; t = –9.666, df = 118, p<0.001), confirming a markedly greater degree of insulin resistance among centrally obese participants. The Fibrosis-4 (FIB-4) index calculated from age, AST, ALT, and platelet count was significantly higher in the central obesity group compared to controls (1.75±1.07 vs. 1.40±0.80; t = –2.021, df=118, p = 0.046). This indicates a greater likelihood of hepatic fibrosis among centrally obese participants (Table 2).

Table No.1: Baseline sociodemographic characteristics of the study's sample (n=120)

Characteristics	Cases (Yes, n=60)	Control (No, n=60)	Total	Significancy
Age (years)	46.65±11.342	39.95±12.497	43.30±12.350	*t*= -3.075, df: 118, *P*= 0.003[a]
< 30	4 (6.7%)	11 (18.3%)	15 (12.5%)	χ^2= 9.956, df: 4, P =0.041[b]
30-40	14 (23.3%)	23 (38.3%)	37 (30.85)	
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%)	
Gender				
Female	34 (56.7%)	31 (51.7%)	65 (54.2%)	χ^2= 0.302, df: 1, P=0.583[b]
Male	26 (43.3%)	29 (48.3%)	55 (45.85)	
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: 16.009, df: 3, P=0.003[cc]
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%)	χ^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)

Table No.2: Mean comparison of different biochemical parameters (n=120)

Biochemical Parameters	Study groups (central obesity)		Mean difference	Significance[a]
	Cases (Yes, n=60)	Control (Yes, n=60)		
Alanine Transaminase (U/L)	49.70±28.965	33.12±17.836	-16.583	t= -3.776, df:118, P= 0.000
Fasting blood sugar (mg/dL)	110.27±40.569	87.75±21.248	-22.517	t= -3.808, df:118,P= 0.000
Triglycerides (mg/dL)	235.63±66.350	161.07±23.989	-74.567	t= -8.187, df:118, P= 0.000
High-density lipoproteins (mg/dL)	35.08±4.073	41.95±3.039	6.867	t= 10.467, df:118, P= 0.000
C-reactive protein (mg/L)	19.90±25.363	9.70±5.378	-10.200	t= -3.047, df:118, P= 0.003
HOMA-IR (mg/dL)	5.930±1.5389	3.392±1.3303	-2.5383	t= -9.666, df:118, P= 0.000
Fibrosis-4 (FIB-4)	1.75033±1.067136	1.40183±0.803314	-0.348500	t= -2.021, df:118, P=0.046

[a]Unpaired T-Test

Table No.3: Distribution of study samples according to their level of fibrosis (n= 120)

Fibrosis' levels	Cases (n=60)		(Controls (n=60)		Total
	No.	%	No.	%	
F0 (No fibrosis)	4	6.7	-	-	
F (0-1)	33	55	53	88.3	86
F1 (Portal fibrosis without septa)	5	8.3	1	1.7	6
F2 (Portal fibrosis with few septa)	8	13.3	1	1.7	9
F3 (Portal fibrosis with numerous septa without cirrhosis)	4	6.7	3	5	7
F4 (Cirrhosis)	6	10	2	3.3	8

Likelihood Ratio: 21.584, df: 5, P = 0.001

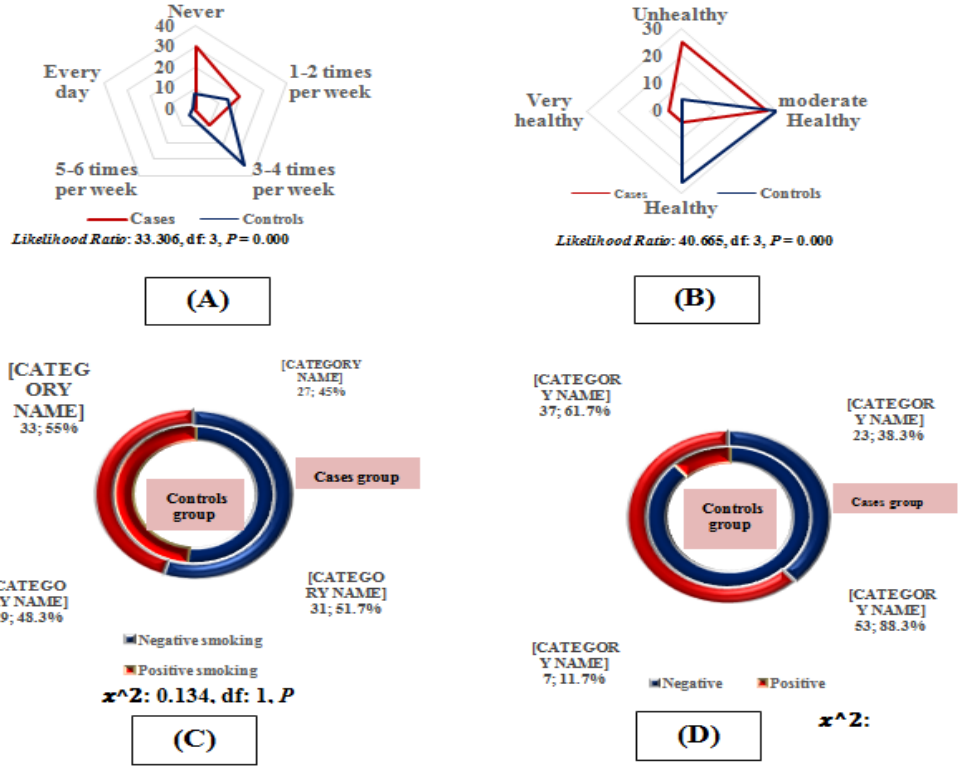

Figure No. 1: Comparison of lifestyle factors and insulin resistance marker between centrally obese and control groups

Table No.4: Distribution of study samples according to their level of steatosis (n= 120)

Fibrosis' levels	Cases (n=60)		(Controls (n=60)		Total
	No.	%	No.	%	
S0 (No steatosis)	31	51.7	50	83.3	81
S1 (5-33%)	9	15	4	6.7	13
S2 (34-66%)	7	11.7	3	5	10
S3 (> 66%)	13	21.7	3	5	16

χ^2 = 14.230, df: 3, P = 0.003

Table No.5: Mean comparison of anthropometric parameter of waist circumference among study's groups (n=120)

Anthropometric Parameter	Study groups (central obesity)		Mean difference	Significance[a]
	Cases (Yes, n=60)	Control (Yes, n=60)		
Waist circumference cm	107.65±12.107	78.63±5.434	-29.017	t= -16.938, df:118, P=0.000

[a]Unpaired T-Test

Table No.6: Mean comparison of anthropometric parameter of waist to height ratio among study's groups (n=120)

Anthropometric Parameter	Study groups (central obesity)		Mean difference	Significance[a]
	Cases (Yes, n=60)	Control (Yes, n=60)		
Waist to height ratio (WtHR)	0.6435±0.07897	0.4815±0.08205	-0.16200	t= -11.019, df:118, P= 0.000

[a]Unpaired T-Test

Other fibrosis and steatosis scores and grading were invesitigated using fibroscan and illustrated that, the higher grading of fibrosis were significantly more among cases groups than that at control group of study samples for F1, F2, F3, and F4 (8.3%, 13.3%, 6.7%, and 10%) vs. (1.7%, 1.7%, 5%, and 3.3%) respectively (Likelihood Ratio 21.584, df: 5, P=0.001) [Table 3].

Likewise, the higher steatosis grading was significantly more among cases groups than that of control group of study samples for S1, S2, and S3 (15%, 11.7%, and 21.7%) vs. (6.7%, 5%, and 5%) respectively ($\chi^{2:}$ 14.230, df: 3, P = 0.003) [Table 4].

The mean waist circumference was significantly higher in the central obesity group compared to controls (107.65±12.11 vs. 78.63±5.43; t = –16.938, df=118, p<0.001), indicating a markedly greater central fat accumulation among obese participants (Table 5).

The mean waist-to-height ratio was significantly higher in the central obesity group compared to controls (0.6435±0.07897 vs. 0.4815±0.08205; t = –11.019, df=118, p< 0.001), confirming a strong association between increased central adiposity and metabolic risk (Table 6).

DISCUSSION

In the present study, strong associations between central obesity and adverse metabolic, inflammatory, and hepatic outcomes, including insulin resistance, elevated liver enzymes, and advanced fibrosis and steatosis. Lifestyle differences were evident: physical inactivity was significantly more common among centrally obese patients (50% vs. 11.7%), aligning with epidemiological evidence linking sedentary behavior to obesity and MASLD.[17] Similarly, unhealthy dietary habits were more frequent in cases (41.7% vs. 6.7%), supporting the established link between poor diet quality and metabolic disorders.[18,19] These findings emphasize lifestyle modification as a key component in MASLD prevention and control.

Anthropometric results confirmed the superiority of central obesity indices (waist circumference and WtHR) over general obesity measures in predicting MASLD risk, reinforcing prior validations of WtHR as a simple and reliable screening tool.[20]

Clinically, insulin resistance was reflected by a significantly higher prevalence of acanthosis Nigricans in centrally obese individuals (61.7% vs. 11.7%), supporting its role as a non-invasive indicator of metabolic dysfunction and hepatic steatosis severity.[21,22]

Metabolic biomarkers demonstrated significantly higher fasting glucose, HOMA-IR, and CRP levels among cases, indicating marked insulin resistance and systemic inflammation - key drivers of MASLD pathogenesis.[21-23] Liver enzymes (ALT, AST) were also elevated, reflecting hepatocellular injury and inflammatory activity associated with MASLD.[23]

Lipid abnormalities, including increased triglycerides and total cholesterol with reduced HDL, were consistent with atherogenic dyslipidemia patterns reported in metabolic and hepatic disorders.[24]

Fibrosis assessment showed significantly higher FIB-4 scores and FibroScan stages among centrally obese participants, confirming central obesity's contribution to fibrosis progression. These findings support FIB-4 as a reliable non-invasive fibrosis index, though accuracy may decline in morbid obesity.[24,25] The combined use of elastography and fibrosis scoring enhances MASLD staging and risk stratification.[26]

CONCLUSION

Central obesity is a key determinant of MASLD severity, promoting insulin resistance, metabolic derangement, systemic inflammation, and fibrosis. Integrating WtHR screening, encouraging lifestyle modification, and using non-invasive fibrosis assessment are essential to improving MASLD management and outcomes.

Author's Contribution:

Concept & Design or acquisition of analysis or interpretation of data:	Ahmed M Shaker, Bilal Natiq Nuaman
Drafting or Revising Critically:	Ahmed M Shaker, Bilal Natiq Nuaman
Final Approval of version:	All the above authors
Agreement to accountable for all aspects of work:	All the above authors

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