

Article

Association of Menopause with Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD) and Quality of Life in Women

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Abstract

Background: Menopause represents a critical physiological transition associated with hormonal changes that influence both metabolic health and quality of life (QoL). Metabolic dysfunction-associated steatotic liver disease (MASLD), a common metabolic condition, is closely linked to menopause; however, its independent contribution to QoL impairment remains unclear. This study aimed to investigate the interplay between menopausal status, metabolic dysfunction, MASLD, and QoL in midlife women. **Methods:** A cross-sectional observational study was conducted including 80 women aged 45–55 years, comprising both premenopausal and menopausal participants. Clinical, anthropometric, biochemical, and imaging data were collected. MASLD was diagnosed using magnetic resonance imaging in the presence of metabolic dysfunction. Metabolic assessment included glucose, insulin, liver enzymes, C-reactive protein, and indices of insulin resistance (HOMA-IR) and sensitivity (QUICKI). QoL was evaluated using the Utian Quality of Life (UQOL) scale. Associations were examined using univariate and multivariable linear regression models. **Results:** MASLD prevalence was significantly higher in menopausal women compared with non-menopausal women (61.9% vs. 15.8%, $p < 0.001$). Metabolic parameters, particularly insulin resistance and body mass index, were strongly associated with MASLD. The mean total UQOL score indicated moderate QoL. In multivariable analysis, menopausal status was the only independent predictor of reduced total QoL ($b = -4.93$, $p = 0.01$) and occupational health domain ($b = -4.60$, $p = 0.001$). MASLD and metabolic parameters were not independently associated with overall QoL. Correlation analyses revealed modest associations between metabolic markers and specific QoL domains, particularly occupational and physical health. **Conclusions:** Menopause is the primary determinant of reduced QoL in midlife women, particularly affecting functional domains, while MASLD does not independently impact QoL despite its strong association with metabolic dysfunction. These findings suggest that menopausal status may play a more prominent role in quality-of-life outcomes than MASLD in women undergoing the menopausal transition. However, the cross-sectional design does not allow conclusions regarding causal or mechanistic relationships.



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1. Introduction

Menopause represents a critical physiological transition in a woman's life, marked by the permanent cessation of ovarian function and characterized by a decline in circulating estradiol and progesterone levels, alongside a compensatory increase in follicle-stimulating hormone (FSH) [1]. These endocrine changes exert widespread neurobiological and systemic effects, influencing thermoregulation, mood, cognition, sleep, and overall physiological homeostasis [1,2]. Importantly, menopause is not merely a symptomatic state, but a complex biological transition associated with increased vulnerability to urogenital, cardiovascular, skeletal, and psychological disorders [2]. While these changes develop gradually in natural menopause, they may occur more abruptly in surgical menopause, often resulting in more severe and persistent symptomatology [3,4].

Beyond its symptomatic burden, menopause is increasingly recognized as a key upstream determinant of metabolic dysfunction [5]. The decline in estrogen levels promotes visceral adiposity, insulin resistance, and alterations in body weight status, thereby creating a metabolic milieu that predisposes to metabolic dysfunction-associated steatotic liver disease (MASLD) [6]. MASLD has emerged as the most prevalent chronic liver disease worldwide, affecting approximately one-third of the adult population, with prevalence increasing from 22% in 1991 to 37% in 2019 in parallel with the global rise in obesity and metabolic disorders [7,8]. Conceptually, MASLD represents a downstream manifestation of systemic metabolic dysregulation and encompasses a spectrum ranging from simple steatosis to metabolic dysfunction-associated steatohepatitis (MASH), fibrosis, and cirrhosis [9].

Despite its systemic implications, MASLD presents a clinical paradox: while it is strongly associated with cardiometabolic risk and cardiovascular disease—the leading cause of mortality in affected individuals, it often remains asymptomatic in its early stages [10]. This dissociation between biological burden and clinical perception raises important questions regarding its relevance for patient-centered outcomes. From a pathophysiological perspective, menopause and MASLD are closely interconnected, with hormonal decline acting as a key driver of metabolic dysregulation and hepatic fat accumulation [6]. However, it remains unclear whether MASLD independently contributes to patient-perceived burden or primarily reflects upstream hormonal and metabolic processes.

Within this framework, quality of life (QoL) emerges as a critical outcome for understanding the broader impact of menopause and its metabolic consequences. QoL is defined by the World Health Organization as individuals' perception of their position in life in relation to their goals, expectations, standards, and concerns within their cultural and value systems [11]. As a multidimensional construct encompassing physical, psychological, social, and functional domains, QoL is particularly sensitive to physiological transitions and chronic conditions [11]. Menopause has been consistently associated with impaired QoL, largely driven by vasomotor symptoms, sleep disturbances, mood changes, and reduced physical capacity [12,13]. However, whether menopause-related metabolic alterations and MASLD contribute independently to QoL impairment remains insufficiently understood.

Furthermore, QoL is inherently domain-specific, and the extent to which menopause and metabolic dysfunction differentially affect specific dimensions, such as occupational, physical, emotional, and sexual health, has not been adequately explored [13]. Existing literature has largely examined menopausal symptoms or metabolic disease in isolation,

without addressing their combined and potentially interacting effects on functional well-being. This gap is particularly relevant given that patient-perceived outcomes may not align with biological disease severity, especially in conditions such as MASLD that are frequently clinically silent.

In addition to its hepatic and metabolic consequences, MASLD has been associated with impaired health-related QoL. Recent studies have demonstrated that individuals with MASLD report poorer general health perception, reduced QoL, and lower work productivity, even in populations with low fibrosis burden. Furthermore, MASLD and its associated metabolic comorbidities have been linked to adverse health-related QoL outcomes across multiple domains, highlighting the broader impact of the disease beyond liver-related morbidity [14,15]. Given the increasing prevalence of MASLD during the menopausal transition, understanding the relative contributions of menopausal status and MASLD to QoL represents an important clinical question.

Therefore, the present study aimed to investigate the interplay between menopausal status, metabolic dysfunction, and MASLD, with a primary focus on their impact on QoL in women aged 45–55 years. Specifically, the study sought to determine whether menopause and associated metabolic alterations constitute independent determinants of QoL impairment and to evaluate the extent to which MASLD contributes to patient-reported outcomes beyond hormonal influences.

2. Materials and Methods

A cross-sectional observational study was conducted to investigate the associations between menopausal status, metabolic dysfunction, MASLD, and QoL in midlife women. The study included 80 participants aged 45–55 years, comprising both premenopausal and menopausal women, recruited from outpatient endocrinology in a university hospital in Athens, Greece, using a random sampling approach. Inclusion criteria comprised women aged ≥ 45 years who were able to understand the study procedures and provide informed consent. Exclusion criteria included severe chronic diseases that could substantially affect metabolic status or quality-of-life outcomes, including active malignancy, advanced cardiovascular disease, chronic kidney disease, chronic inflammatory disorders, decompensated liver disease, and other major systemic illnesses. Additional exclusion criteria included endocrine disorders affecting glucose metabolism independently of metabolic dysfunction, hepatic diseases of non-metabolic origin, psychiatric disorders, and any clinical or cognitive condition that could interfere with participation or the reliability of the collected data. Hepatic steatosis was assessed using magnetic resonance imaging (MRI) of the upper abdomen. MRI examinations were reviewed for the presence or absence of hepatic steatosis. The assessment was qualitative and intended to establish MASLD status; quantitative liver fat fraction measurements, region-of-interest (ROI)-based analyses, and automated fat quantification techniques were not performed. Participants were classified according to the presence or absence of imaging evidence of hepatic steatosis.

For the purposes of the present analysis, participants with imaging-confirmed hepatic steatosis and at least one cardiometabolic risk factor were classified according to the contemporary MASLD nomenclature proposed by the multisociety Delphi consensus [8].

Menopausal status was determined based on detailed gynecological history obtained during the study interview and review of available medical records. Women were classified as postmenopausal if they reported permanent cessation of menstruation consistent with natural menopause or had undergone surgical menopause. The study population included women aged 45–55 years across different stages of the menopausal transition. Information regarding reproductive history, age at menopause, gynecological history, and use of menopausal hormone therapy was collected as part of the structured clinical assessment.

Hormonal measurements were not used for menopausal classification, and menopausal status was determined on clinical grounds. A small number of women with surgical menopause were included and classified as postmenopausal.

All participants underwent standardized clinical and biochemical assessment, including fasting blood sampling and oral glucose tolerance testing (OGTT). A standard 75 g OGTT was performed in all participants. Plasma glucose levels were measured following glucose administration. Glucose tolerance was interpreted according to the American Diabetes Association criteria, whereby normal glucose tolerance was defined as a 2 h plasma glucose level <140 mg/dL, impaired glucose tolerance as 140–199 mg/dL, and diabetes mellitus as ≥ 200 mg/dL [16]. Anthropometric measurements were obtained using calibrated instruments, and MRI examinations were performed according to established radiological protocols for the assessment of hepatic steatosis. All procedures were conducted under standardized conditions to ensure data accuracy and reproducibility.

Metabolic assessment included fasting measurements of plasma glucose, insulin, alanine aminotransferase (ALT), aspartate aminotransferase (AST), gamma-glutamyl transferase (GGT), and C-reactive protein (CRP), as well as oral glucose tolerance test. Insulin resistance and sensitivity were estimated using the homeostasis model assessment index (HOMA-IR) and the quantitative insulin sensitivity check index (QUICKI), while body mass index (BMI) was calculated as an anthropometric indicator of body weight status.

Menopausal status was determined based on clinical history and QoL was evaluated using the Utian Quality of Life (UQOL) scale, a validated instrument designed to evaluate well-being in midlife women. The UQOL questionnaire comprises four subscales: occupational health (score range: 7–35), physical health (7–35), emotional health (6–30), and sexual health (3–15) [17]. The total QoL score ranges from 23 to 115, with higher scores indicating better perceived QoL [17]. The internal consistency of the UQOL in the present study was high. Cronbach's alpha coefficient for the total scale was 0.890. For the individual subscales, Cronbach's alpha values were 0.840 for occupational health, 0.802 for physical health, 0.708 for emotional health, and 0.705 for sexual health, indicating good to excellent internal reliability. Statistical analysis included univariate and multivariable linear regression models to examine associations between clinical variables and QoL outcomes, with statistical significance set at $p < 0.05$. Data analysis was performed using IBM SPSS Statistics version 28.0 (IBM Corp., Armonk, NY, USA).

Fasting plasma glucose, liver enzymes, inflammatory markers, and hematological indices were interpreted according to the reference ranges used by the hospital laboratory. The reference ranges were as follows: fasting glucose 70–100 mg/dL, AST < 32 U/L, ALT < 33 U/L, GGT 5–36 U/L, alkaline phosphatase 35–104 U/L, total bilirubin 0.20–1.20 mg/dL, albumin 3.5–5.2 g/dL, LDH 135–214 U/L, CRP < 0.5 mg/dL, white blood cell count $4.0\text{--}9.0 \times 10^3/\mu\text{L}$, platelet count $120\text{--}380 \times 10^3/\mu\text{L}$, erythrocyte sedimentation rate 0–18 mm/h, AST/ALT ratio 0–1, and neutrophil-to-lymphocyte ratio (NLR) 0–3.

Insulin resistance was assessed using the Homeostasis Model Assessment of Insulin Resistance (HOMA-IR), calculated as fasting insulin ($\mu\text{U/mL}$) $\times$ fasting glucose (mmol/L)/22.5 [18]. Insulin sensitivity was evaluated using the Quantitative Insulin Sensitivity Check Index (QUICKI), calculated as $1/[\log(\text{fasting insulin}) + \log(\text{fasting glucose})]$ [19]. According to the laboratory reference ranges, HOMA-IR values between 0.5 and 1.4 were considered within the normal range, whereas values > 1.4 were classified as abnormal. QUICKI values 0.34 were considered normal, whereas values ≤ 0.34 were classified as abnormal. For descriptive purposes, HOMA-IR values 1.9 were considered indicative of early insulin resistance and values > 2.9 of significant insulin resistance. Normal and abnormal values for biochemical and hematological parameters were defined according to the reference ranges used by the hospital laboratory.

Continuous variables are presented as mean $\pm$ standard deviation (SD), median, and interquartile range (IQR), whereas categorical variables are presented as frequencies and percentages. Normality was assessed using the Kolmogorov–Smirnov test. Associations between categorical variables were evaluated using the chi-square test or chi-square test for trend, as appropriate. Correlations between continuous variables were assessed using Pearson’s correlation coefficient for normally distributed variables and Spearman’s rank correlation coefficient for non-normally distributed variables. Univariable and multivariable linear regression analyses were performed to identify factors associated with quality-of-life outcomes, including total UQOL score and its individual domains. Crude and adjusted regression coefficients (b), corresponding 95% confidence intervals (CIs), and *p*-values were calculated. Statistical significance was set at *p* < 0.05.

Covariates included in the multivariable models were selected a priori based on their clinical relevance and previously reported associations with quality of life, menopausal status, and metabolic dysfunction. Marital status and educational level were included as indicators of socioeconomic status, whereas HOMA-IR was included as a measure of insulin resistance. Menopausal status and MASLD status represented the primary variables of interest.

Multicollinearity was assessed using variance inflation factors (VIFs). Age was excluded from multivariable analyses because of collinearity with menopausal status. In addition, QUICKI was not included in multivariable models because of its strong inverse correlation with HOMA-IR (*r* = −0.95, *p* < 0.001). All VIF values in the final models were below 4, indicating acceptable levels of multicollinearity.

The study was conducted in accordance with the principles of the Declaration of Helsinki. Ethical approval was obtained from the hospital’s Ethics Committee (approval number: 347/28-07-2021). All participants provided written informed consent prior to participation and were informed that they were free to withdraw from the study at any time without any consequences. All data were fully anonymized before analysis to ensure participant confidentiality and compliance with data protection regulations.

3. Results

3.1. Participant Characteristics

A total of 80 women were included, with a mean age of 49.9 years (SD = 3.6), indicating a population centered around the menopausal transition. Menopausal women accounted for 52.5% of the sample, while 40% were diagnosed with MASLD. Metabolic disturbances were common, with 50% of participants presenting abnormal HOMA-IR values and 37.5% abnormal QUICKI values. Additionally, 68.8% had impaired glucose levels at 60 min and 60% elevated insulin levels at 30 min. Regarding body weight status, 53.8% had normal BMI, while 42.5% had overweight and 3.8% had obesity. Liver enzyme abnormalities were relatively limited (ALT: 5%; AST: 13.8%), whereas CRP elevation was observed in 21.3% of participants (Tables 1 and 2).

Table 1. Clinical characteristics of women as categorical variables.

Variable	<i>n</i>	%
Menopausal status		
No	38	47.5
Yes	42	52.5
MASLD		
No	48	60.0
Yes	32	40.0
HOMA index		

Table 1. *Cont.*

Variable	<i>n</i>	%
Normal values	40	50.0
Abnormal values	40	50.0
QUICKI index		
Normal values	50	62.5
Abnormal values	30	37.5
Glucose at 60 min		
Normal values	25	31.3
Abnormal values	55	68.8
Insulin at 30 min		
Normal values	32	40.0
Abnormal values	48	60.0
BMI		
Underweight	0	0.0
Normal weight	43	53.8
Overweight	34	42.5
Obese	3	3.8
ALT		
Normal values	76	95.0
Abnormal values	4	5.0
AST		
Normal values	69	86.3
Abnormal values	11	13.8
GGT		
Normal values	79	98.8
Abnormal values	1	1.2
CRP		
Normal values	63	78.8
Abnormal values	17	21.3

Abbreviations: MASLD, metabolic dysfunction-associated steatotic liver disease; HOMA, Homeostatic Model Assessment; QUICKI, Quantitative Insulin Sensitivity Check Index; BMI, body mass index; ALT, alanine aminotransferase; AST, aspartate aminotransferase; GGT, gamma-glutamyl transferase; CRP, C-reactive protein.

Table 2. Clinical characteristics of women as continuous variables.

Variable	Mean	SD	Median	IQR
HOMA	1.64	0.85	1.41	1.22
QUICKI	0.36	0.03	0.36	0.05
Glucose at 60 min	169.35	30.04	170	41.5
Insulin at 30 min	22.50	6.80	19.4	7.5
BMI	24.79	2.41	24.8	2.2
ALT	21.31	5.03	20	3
AST	23.69	5.40	22	6
GGT	12.89	7.34	9.8	10.9
CRP	0.73	1.95	0.41	0.37

Abbreviations: ALT, alanine aminotransferase; AST, aspartate aminotransferase; BMI, body mass index; CRP, C-reactive protein; GGT, gamma-glutamyl transferase; HOMA, Homeostatic Model Assessment; IQR, interquartile range; QUICKI, Quantitative Insulin Sensitivity Check Index; SD, standard deviation.

3.2. Association Between Menopause and MASLD

Menopausal status was strongly associated with MASLD prevalence. MASLD was present in 61.9% of menopausal women compared with 15.8% of non-menopausal women ($p < 0.001$), corresponding to a substantial absolute difference. Similarly, age ≥ 50 years was associated with higher MASLD prevalence (63.4% vs. 15.4%, $p < 0.001$), indicating overlapping effects of age and menopausal transition (Table 3).

Table 3. Association between independent variables and MASLD status.

Independent Variables	MASLD: No		MASLD: Yes		<i>p</i> -Value
	<i>n</i>	%	<i>n</i>	%	
Age					<0.001 ^a
<50 (median age)	33	84.6	6	15.4	
≥50	15	36.6	26	63.4	
Menopausal status					<0.001 ^a
No	32	84.2	6	15.8	
Yes	16	38.1	26	61.9	
HOMA index					<0.001 ^a
Normal values	36	90.0	4	10.0	
Abnormal values	12	30.0	28	70.0	
QUICKI					<0.001 ^a
Normal values	43	86.0	7	14.0	
Abnormal values	5	16.7	25	83.3	
Glucose at 60 min					0.62 ^a
Normal values	16	64.0	9	36.0	
Abnormal values	32	58.2	23	41.8	
Insulin at 30 min					0.01 ^a
Normal values	25	78.1	7	21.9	
Abnormal values	23	47.9	25	52.1	
BMI					<0.001 ^a
Normal weight	37	86.0	6	14.0	
Overweight/obese	11	29.7	26	70.3	
ALT					0.01 ^a
Normal values	48	63.2	28	36.8	
Abnormal values	0	0.0	4	100.0	
AST					<0.001 ^a
Normal values	48	69.6	21	30.4	
Abnormal values	0	0.0	11	100.0	
GGT					0.22 ^a
Normal values	48	60.8	31	39.2	
Abnormal values	0	0.0	1	100.0	
CRP					0.50 ^a
Normal values	39	61.9	24	38.1	
Abnormal values	9	52.9	8	47.1	

^a Chi-square test. **Abbreviations:** HOMA, Homeostatic Model Assessment; QUICKI, Quantitative Insulin Sensitivity Check Index; BMI, body mass index; ALT, alanine aminotransferase; AST, aspartate aminotransferase; GGT, gamma-glutamyl transferase; CRP, C-reactive protein.

Markers of insulin resistance and metabolic dysfunction demonstrated the strongest associations with MASLD status. Specifically, abnormal HOMA-IR values were associated with a seven-fold higher MASLD prevalence (70% vs. 10%, $p < 0.001$), while abnormal QUICKI values were associated with an approximately six-fold increase (83.3% vs. 14%, $p < 0.001$). Elevated insulin levels at 30 min were also significantly associated with MASLD ($p = 0.01$), although with a smaller effect size (Table 3).

BMI was also strongly associated with MASLD. Specifically, 70.3% of women with overweight or obesity had MASLD compared with only 14% of women with normal BMI ($p < 0.001$), indicating a marked difference in disease prevalence across BMI categories (Table 3).

Liver enzyme abnormalities demonstrated very strong associations with MASLD. All women with elevated ALT levels had MASLD (100%), compared with 36.8% of those with normal ALT values ($p = 0.01$). Similarly, 100% of women with elevated AST levels had MASLD, compared with 30.4% among those with normal AST values ($p < 0.001$). Although these findings indicate strong discriminatory capacity, the relatively small number of abnormal cases should be considered when interpreting these results. No statistically significant associations were identified for glucose at 60 min, GGT, or CRP ($p > 0.05$) (Table 3).

3.3. QoL

The mean total UQOL score was 72.4 (SD = 6.1), indicating a moderate level of perceived QoL. Among subdomains, occupational health had the highest mean score (27.2 ± 4.6), followed by physical (19.0 ± 2.8), emotional (16.9 ± 2.5), and sexual health (9.4 ± 1.4), suggesting relatively preserved functional capacity but lower scores in physical and emotional dimensions (Table 4).

Table 4. Descriptive results of the Utian Quality of Life (UQOL) scale.

Scale	Mean	SD	Median	IQR
Utian Quality of Life (total score)	72.4	6.1	72	8
Occupational health	27.2	4.6	27	5
Physical health	19.0	2.8	18.5	3
Emotional health	16.9	2.5	17	3.8
Sexual health	9.4	1.4	10	1

Abbreviations: UQOL, Utian Quality of Life; SD, standard deviation; IQR, interquartile range.

3.4. Association Between Clinical Parameters and QoL

Correlation analyses revealed that associations between clinical variables QoL were domain-specific and generally modest in magnitude. Insulin resistance (HOMA-IR) was weakly negatively correlated with occupational health ($r = -0.22$, $p < 0.05$), while insulin sensitivity (QUICKI) showed a moderate positive correlation with the same domain ($r = 0.31$, $p < 0.01$) (Table 5). These findings indicate that metabolic status is primarily linked to functional aspects of QoL rather than overall perception. Liver enzymes were selectively associated with quality-of-life domains. ALT levels were negatively correlated with occupational ($r = -0.25$, $p < 0.05$) and physical health ($r = -0.22$, $p < 0.05$), while AST was negatively associated with physical health ($r = -0.24$, $p < 0.05$) (Table 5). No statistically significant associations were observed between most metabolic parameters and the total UQOL score, emotional health, or sexual health domains ($p > 0.05$). In contrast, significant associations were identified primarily in the occupational and physical health domains, indicating that the impact of metabolic dysfunction is domain-specific rather than generalized across all dimensions of quality of life (Table 5).

Table 5. Correlations between clinical characteristics and quality of life.

Variable	UQOL (Total Score)	Occupational Health	Physical Health	Emotional Health	Sexual Health
HOMA	−0.16	−0.22 *	−0.10	0.15	−0.05
QUICKI	0.25 *	0.31 **	0.13	−0.15	0.07
Glucose at 60 min	−0.06	−0.12	0.08	0.05	−0.08
Insulin at 30 min	−0.03	−0.17	−0.09	0.19	0.04
Body mass index (BMI)	−0.17	−0.21	−0.08	0.08	−0.03
ALT	−0.21	−0.25 *	−0.22 *	0.20	0.02
AST	−0.20	−0.20	−0.24 *	0.13	0.02
GGT	−0.11	−0.17	−0.06	0.11	−0.01
CRP	−0.03	−0.07	−0.08	0.13	−0.01

Values represent correlation coefficients (r). **Abbreviations:** UQOL, Utian Quality of Life; BMI, body mass index; HOMA, Homeostatic Model Assessment; QUICKI, Quantitative Insulin Sensitivity Check Index; ALT, alanine aminotransferase; AST, aspartate aminotransferase; GGT, gamma-glutamyl transferase; CRP, C-reactive protein. * $p < 0.05$. ** $p < 0.01$.

3.5. Regression Analysis of Quality of Life

3.5.1. Total UQOL Score

Univariate linear regression analysis identified both age and menopausal status as statistically significant predictors of total UQOL score. Specifically, increasing age was associated with a reduction in UQOL score ($b = -0.48$, 95% CI: -0.85 to -0.11 , $p = 0.01$), indicating that each additional year of age corresponded to an average decrease of 0.48 units in overall quality-of-life score (Table S1).

Menopausal status demonstrated a stronger effect size, with menopausal women exhibiting significantly lower UQOL scores compared with non-menopausal women ($b = -3.98$, 95% CI: -6.56 to -1.39 , $p = 0.003$) (Table S1). This suggests that menopause is associated with a clinically meaningful reduction in perceived quality of life.

Other variables, including BMI ($b = -0.43$, $p = 0.14$), HOMA- IR ($b = -1.18$, $p = 0.15$), and MASLD status ($b = -1.35$, $p = 0.33$) were not statistically significant predictors in univariate analysis, although the direction of the coefficients was generally consistent with reduced quality of life (Table S1).

In the multivariable linear regression model, menopausal status remained the only statistically significant independent predictor of total UQOL score ($b = -4.93$, 95% CI: -8.82 to -1.04 , $p = 0.01$), while the effect of age was attenuated and no longer significant (Table 6). This attenuation suggests that the observed age effect in univariate analysis is likely mediated or confounded by menopausal status, reflecting the strong overlap between chronological aging and the menopausal transition. The magnitude of the regression coefficient indicates that menopause is associated with an average reduction of approximately 5 points in total UQOL score after adjustment, representing a meaningful decrement given the observed distribution of scores.

Table 6. Multivariable linear regression analysis with total UQOL score as the dependent variable.

Independent Variables	Adjusted Coefficient (b)	95% CI for b	p-Value	VIF
Marital status			0.75	1.07
Single/widowed/divorced	Reference category			
Married	−0.52	−3.76 to 2.71		
Educational level			0.29	1.09
Secondary education	Reference category			
Tertiary education	1.83	−2.37 to 6.04		
BMI	−0.18	−0.86 to 0.51	0.14	1.53
Menopausal status			0.01	2.14
No	Reference category			
Yes	−4.93	−8.82 to −1.04		
HOMA	0.05	−2.65 to 2.75	0.97	2.93
MASLD			0.71	2.40
No	Reference category			
Yes	0.78	−3.41 to 4.98		

Abbreviations: CI, confidence interval; VIF, variance inflation factor; BMI, body mass index; MASLD, metabolic dysfunction-associated steatotic liver disease; HOMA, Homeostatic Model Assessment; UQOL, Utian Quality of Life.

The multivariable model demonstrated acceptable statistical performance. The Durbin-Watson statistic was 2.2, indicating independence of residuals and absence of autocorrelation. Variance inflation factor (VIF) values for all predictors were below 4, confirming the absence of problematic multicollinearity. The model explained 4.7% of the variance in total UQOL score ($R^2 = 0.047$), indicating that although menopause is a statistically significant determinant, the majority of variability in QoL remains unexplained by the included variables.

Importantly, MASLD was not associated with total QoL in either univariate or multivariable models ($p > 0.05$), suggesting that the presence of MASLD does not independently contribute to overall quality-of-life impairment in this cohort (Table 6).

3.5.2. Occupational Health Domain

Univariate linear regression analysis identified multiple variables associated with occupational health. Increasing age was significantly associated with lower occupational health scores ($b = -0.50$, 95% CI: -0.77 to -0.23 , $p < 0.001$), indicating a decline in work-related functioning with advancing age. Menopausal status demonstrated a strong association with occupational health ($b = -4.18$, 95% CI: -6.01 to -2.35 , $p < 0.001$), with menopausal women reporting substantially lower scores (Table S2). Additionally, HOMA-IR showed a borderline statistically significant association ($b = -1.21$, 95% CI: -2.41 to -0.02 , $p = 0.05$), suggesting that increased insulin resistance may negatively affect occupational functioning (Table S2).

However, in the multivariable model, menopausal status remained the only statistically significant independent predictor ($b = -4.60$, 95% CI: -7.35 to -1.85 , $p = 0.001$) (Table 7). The effects of age, and HOMA-IR were no longer significant after adjustment, suggesting that these associations are either confounded by or mediated through menopausal status. The effect size of menopause in this model is substantial, indicating a reduction of approximately 4.6 points in occupational health score, which represents a meaningful impact on functional quality of life.

Table 7. Multivariable linear regression analysis with occupational health as the dependent variable.

Independent Variables	Adjusted Coefficient (b)	95% CI for b	p-Value	VIF
Marital status			0.84	1.07
Single/widowed/divorced	Reference category			
Married	−0.23	−2.52 to 2.06		
Educational level			0.30	1.09
Secondary education	Reference category			
Tertiary education	1.56	−1.42 to 4.54		
Body mass index (BMI)	−0.06	−0.54 to 0.43	0.82	1.53
Menopausal status			0.001	2.14
No	Reference category			
Yes	−4.60	−7.35 to −1.85		
HOMA-IR	0.54	−1.38 to 2.45	0.58	2.93
MASLD			0.95	2.40
No	Reference category			
Yes	−0.09	−3.06 to 2.88		

Abbreviations: CI, confidence interval; VIF, variance inflation factor; BMI, body mass index; MASLD, metabolic dysfunction-associated steatotic liver disease; HOMA, Homeostatic Model Assessment.

The model demonstrated good statistical validity, with a Durbin-Watson value of 2.2 indicating independence of residuals, and VIF values within acceptable limits (<4), excluding multicollinearity concerns. The explained variance was notably higher compared to the total UQOL model ($R^2 = 0.154$), indicating that the included predictors account for 15.4% of the variability in occupational health. This suggests that occupational functioning is more strongly influenced by clinical and demographic variables than overall quality of life.

MASLD was not a significant predictor of occupational health in either univariate or multivariable analysis ($p > 0.05$), further supporting the absence of an independent association between liver disease status and functional quality-of-life outcomes (Table 7).

3.5.3. Physical Health Domain

In univariate analysis, no statistically significant associations were identified between physical health and the examined variables, including age, BMI, menopausal status, HOMA-IR, MASLD, or menopausal symptoms ($p > 0.05$) (Table S3).

In the multivariable model, none of the included predictors demonstrated statistical significance, and the overall model was not significant (ANOVA $p = 0.91$), indicating that the selected variables did not meaningfully explain variability in physical health scores. The absence of significant findings suggests that physical health, as measured by UQOL, may be influenced by factors not captured in the present analysis or may reflect a relatively preserved domain within this population (Table 8).

Table 8. Multivariable linear regression analysis with physical health as the dependent variable.

Independent Variables	Adjusted Coefficient (b)	95% CI for b	p-Value	VIF
Marital status			0.58	1.07
Single/widowed/divorced	Reference category			
Married	−0.45	−2.03 to 1.14		
Educational level			0.93	1.09
Secondary education	Reference category			
Tertiary education	0.09	−1.98 to 2.16		
BMI	−0.02	−0.36 to 0.32	0.91	1.53
Menopausal status			0.76	2.14
No	Reference category			
Yes	0.29	−1.62 to 2.21		
HOMA-IR	−0.06	−1.38 to 1.27	0.93	2.93
MASLD			0.97	2.40
No	Reference category			
Yes	−0.04	−2.10 to 2.03		

Abbreviations: CI, confidence interval; VIF, variance inflation factor; BMI, body mass index; MASLD, metabolic dysfunction-associated steatotic liver disease; HOMA, Homeostatic Model Assessment.

3.5.4. Emotional Health Domain

Similarly, no statistically significant predictors of emotional health were identified in either univariate or multivariable analyses ($p > 0.05$) (Tables S4 and 9). Although some variables showed directional trends (e.g., negative association with BMI and positive association with MASLD), these did not reach statistical significance, and the multivariable model was not significant (ANOVA $p = 0.91$) (Tables S4 and 9). These findings suggest that emotional health may be influenced by psychosocial or behavioral factors not included in the current models, rather than the clinical and metabolic variables examined.

Table 9. Multivariable linear regression analysis with emotional health as the dependent variable.

Independent Variables	Adjusted Coefficient (b)	95% CI for b	p-Value	VIF
Marital status			0.48	1.07
Single/widowed/divorced	Reference category			
Married	−0.45	−1.71 to 0.81		
Educational level			0.42	1.09
Secondary education	Reference category			
Tertiary education	0.67	−0.97 to 2.31		
BMI	−0.11	−0.38 to 0.16	0.41	1.53
Menopausal status			0.49	2.14
No	Reference category			
Yes	−0.53	−2.05 to 0.98		
HOMA-IR	−0.47	−1.52 to 0.59	0.38	2.93
MASLD			0.16	2.40
No	Reference category			
Yes	1.17	−0.47 to 2.81		

Abbreviations: CI, confidence interval; VIF, BMI, body mass index; MASLD, metabolic dysfunction-associated steatotic liver disease; HOMA, Homeostatic Model Assessment.

3.5.5. Sexual Health Domain

No statistically significant associations were identified between sexual health and any of the examined predictors in either univariate or multivariable analyses ($p > 0.05$) (Tables S5 and 10). The multivariable model did not demonstrate statistical significance (ANOVA $p = 0.87$), indicating limited explanatory capacity of the included variables for this domain (Table 10). This suggests that sexual health outcomes may be influenced by factors beyond the scope of the present clinical and metabolic variables, including relational, psychological, or hormonal factors not directly assessed in this analysis.

Table 10. Multivariable linear regression analysis with sexual health as the dependent variable.

Independent Variables	Adjusted Coefficient (b)	95% CI for b	p-Value	VIF
Marital status			0.18	1.07
Single/widowed/divorced	Reference category			
Married	0.61	−0.19 to 1.39		
Educational level			0.36	1.09
Secondary education	Reference category			
Tertiary education	−0.48	−1.52 to 0.55		
BMI	−0.01	−0.16 to 0.18	0.92	1.53
Menopausal status			0.71	2.14
No	Reference category			
Yes	−0.09	−1.05 to 0.87		
HOMA-IR	0.04	−0.63 to 0.71	0.91	2.93
MASLD			0.61	2.40
No	Reference category			
Yes	−0.27	−1.30 to 0.77		

Abbreviations: CI, confidence interval; VIF, variance inflation factor; BMI, body mass index; MASLD, metabolic dysfunction-associated steatotic liver disease; HOMA, Homeostatic Model Assessment.

4. Discussion

The present study provides an integrated evaluation of the relationship between menopausal status, metabolic dysfunction, MASLD, and QoL in women undergoing the menopausal transition. The findings demonstrate that menopausal status is the principal determinant of reduced quality of life, particularly affecting total QoL and the occupational domain, whereas metabolic parameters and MASLD do not independently contribute to QoL impairment after adjustment. These results highlight a domain-specific pattern of QoL disruption, suggesting that endocrine changes associated with menopause exert a more immediate and perceptible impact on well-being than downstream metabolic manifestations [12,13,20].

The independent association between menopause and reduced QoL is consistent with the established understanding of menopause as a critical period of systemic physiological and psychosocial transition. Estrogen deficiency has been linked to vasomotor symptoms, sleep disturbances, mood instability, and reduced physical capacity, all of which contribute to impaired QoL [20,21]. In parallel, menopause induces profound metabolic alterations, including increased visceral adiposity and reduced insulin sensitivity [22]. Importantly, the findings of this study extend this framework by demonstrating that these changes do not affect all QoL domains equally but are primarily reflected in functional aspects of daily life, particularly occupational health. This domain-specific impairment likely reflects the cumulative burden of fatigue, metabolic inefficiency, and reduced cognitive and physical resilience [23].

Within this context, MASLD should be interpreted as part of a broader metabolic continuum rather than an isolated hepatic condition. MASLD is strongly associated with obesity, insulin resistance, and metabolic syndrome, and its global prevalence continues to increase substantially [7,8,24]. Importantly, the disease exhibits a clear sex-specific pattern,

with menopause representing a critical turning point in risk trajectory [24]. Evidence indicates that menopause is associated with more than a twofold increase in MASLD prevalence, largely mediated by hormonal changes [24]. Estrogens play a central role in regulating lipid metabolism by reducing hepatic lipogenesis, enhancing fatty acid oxidation, and promoting subcutaneous rather than visceral fat deposition. Their decline results in a metabolic shift toward increased visceral adiposity, systemic inflammation, and hepatic fat accumulation, thereby facilitating MASLD development [24,25].

Despite this strong biological linkage, MASLD was not independently associated with quality-of-life outcomes in the present study. This apparent discrepancy can be explained by the clinical characteristics of MASLD, which is frequently asymptomatic in its early stages and often diagnosed incidentally. As highlighted in recent literature, MASLD represents a spectrum of disease that may remain clinically silent despite significant metabolic dysregulation [2,8,26]. Jancova et al. [24] further emphasize that MASLD progression is driven by complex interactions between metabolic, hormonal, and inflammatory pathways, but its clinical manifestations may not become perceptible to patients until advanced stages. Therefore, while MASLD reflects substantial underlying metabolic disturbance, it may not directly translate into reduced patient-reported QoL, particularly in populations without advanced fibrosis or symptomatic disease [25].

The findings of this study suggest that menopausal status may be more closely associated with quality-of-life outcomes than MASLD in women undergoing the menopausal transition. However, because of the cross-sectional design, causal pathways and potential mediating relationships between menopause, metabolic dysfunction, MASLD, and QoL cannot be established. This interpretation is consistent with evidence demonstrating that hormonal changes during menopause influence not only fat distribution and insulin sensitivity but also inflammatory signaling and hepatic lipid handling [24]. Furthermore, postmenopausal women have been shown to be at increased risk not only for MASLD development but also for more severe disease phenotypes, including fibrosis, suggesting that hormonal status plays a critical role in both disease initiation and progression [24,25].

Although metabolic parameters such as insulin resistance indices and liver enzymes were not independently associated with overall QoL in multivariable models, correlation analyses revealed modest associations with specific domains, particularly occupational and physical health. These findings suggest that metabolic dysfunction may exert subtle and domain-specific effects rather than a generalized impact on perceived well-being. Insulin resistance, a central mechanism in MASLD pathophysiology, disrupts energy homeostasis and contributes to fatigue and reduced physical performance, which may preferentially affect functional domains of QoL [25–27]. This aligns with the concept that metabolic burden may influence daily functioning before it becomes clinically or subjectively evident at a global level [2].

A key contribution of this study is the identification of a domain-specific pattern of QoL impairment. While menopause demonstrated a significant and independent association with total and occupational QoL, no corresponding associations were observed for emotional, physical, or sexual health domains in adjusted analyses. This finding underscores the multidimensional nature of QoL and suggests that different domains are influenced by distinct biological and psychosocial determinants [28]. Emotional and sexual health, in particular, are shaped by complex interpersonal and sociocultural factors that extend beyond metabolic or endocrine status [3,11]. The absence of significant associations in these domains further supports the notion that menopause-related metabolic changes primarily affect functional capacity rather than all aspects of subjective well-being [12].

The relatively low explanatory power of the regression models highlights the inherent complexity of QoL as a construct. The low R^2 values observed in several models further

suggest that important biological, psychological, behavioral, and social determinants of QoL were not captured in the present analysis. QoL is influenced by a dynamic interplay of biological, psychological, and social factors and cannot be fully explained by clinical or metabolic variables alone [23,28]. While MASLD represents an important metabolic condition with long-term health implications, its contribution to QoL appears limited in the absence of advanced disease or overt symptomatology [8]. In contrast, menopause exerts a more immediate and multidimensional impact, reinforcing its central role in shaping patient-reported outcomes during midlife [13].

From a clinical perspective, these findings emphasize the importance of adopting a holistic and sex-specific approach to patient management. MASLD is associated with increased risk of cardiovascular disease, malignancy, and liver-related complications, making it a critical target for long-term prevention strategies [9]. However, the present results suggest that interventions aimed at improving QoL in midlife women should prioritize the management of menopausal symptoms and functional capacity, rather than focusing exclusively on metabolic or hepatic markers. This distinction is particularly relevant in clinical practice, where patient-reported outcomes are increasingly recognized as essential components of comprehensive care [13]. Accordingly, the present findings may be particularly relevant to women undergoing the menopausal transition, a period characterized by substantial hormonal and metabolic changes.

There are several limitations of this study that should be acknowledged. The cross-sectional design precludes causal inference, and the relatively small sample size may have limited statistical power, particularly for detecting modest associations between MASLD and individual QoL domains. Consequently, the possibility of type II error cannot be excluded. Menopausal status was determined using clinical history and gynecological assessment rather than hormonal measurements (e.g., FSH or estradiol levels). Although this approach reflects routine clinical practice and is widely used in epidemiological studies, some degree of misclassification cannot be excluded. Additionally, the absence of psychosocial variables may have contributed to the low explanatory power of the models and the lack of associations observed in certain QoL domains. Physical activity and exercise habits were not systematically assessed as part of the study protocol and therefore could not be included in the analyses. Given the established associations between physical activity, metabolic health, and quality of life, residual confounding related to lifestyle behaviors cannot be excluded. Given that MASLD is a chronic and progressive condition, longitudinal studies are needed to determine whether its impact on QoL becomes more pronounced with disease progression. Future research should also incorporate broader determinants of health, including psychological, behavioral, and social factors, to better capture the multidimensional nature of QoL in this population.

The present study was designed to evaluate the association between MASLD status and QoL across the menopausal transition. Therefore, the imaging protocol focused on the identification of hepatic steatosis rather than the assessment of fibrosis severity. Quantitative liver fat measurements, elastography, and fibrosis-specific assessments were not included in the study protocol. Future studies incorporating quantitative imaging techniques and validated fibrosis assessments may further clarify the relationship between liver disease severity and quality-of-life outcomes in this population.

The study included multiple correlation and regression analyses, which may increase the risk of type I error. As adjustments for multiple comparisons were not applied, findings from secondary and exploratory analyses should be interpreted cautiously and require confirmation in larger prospective studies.

An additional consideration is that the study population included women across the menopausal transition rather than an exclusively postmenopausal cohort. Consequently,

the observed associations may reflect the combined influence of menopausal transition, established menopause, and age-related metabolic changes. In women who are uniformly postmenopausal, the prevalence and severity of metabolic dysfunction and MASLD may differ according to time since menopause, potentially influencing their relationship with quality of life. Therefore, the present findings may not be fully generalizable to exclusively postmenopausal populations and should be interpreted within the context of women undergoing the menopausal transition.

In conclusion, menopause emerges as the primary determinant of reduced quality of life, particularly in the occupational domain, while MASLD and metabolic parameters do not independently influence QoL outcomes in this cohort. These findings indicate that menopausal status is more strongly associated with quality-of-life outcomes than MASLD in this cohort. Although menopause was associated with both metabolic dysfunction and reduced quality of life, the cross-sectional design precludes conclusions regarding causal or mechanistic pathways. This distinction underscores the need for integrated, women-centered approaches that address both endocrine and metabolic health across the lifespan.

Supplementary Materials: The following supporting information can be downloaded at: <https://www.mdpi.com/article/10.3390/livers6040057/s1>, Table S1: Univariate linear regression analysis with total UQOL score as the dependent variable; Table S2: Univariate linear regression analysis with occupational health as the dependent variable; Table S3: Univariate linear regression analysis with physical health as the dependent variable; Table S4: Univariate linear regression analysis with emotional health as the dependent variable; Table S5: Univariate linear regression analysis with sexual health as the dependent variable.

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Abbreviations

The following abbreviations are used in this manuscript:

ALT	Alanine aminotransferase
AST	Aspartate aminotransferase
BMI	Body mass index
CRP	C-reactive protein

FSH	Follicle-stimulating hormone
HOMA-IR	Homeostasis model assessment of insulin resistance
MASLD	Metabolic dysfunction-associated steatotic liver disease
MASH	Metabolic dysfunction-associated steatohepatitis
MRI	Magnetic resonance imaging
QoL	Quality of life
QUICKI	Quantitative insulin sensitivity check index
UQOL	Utian Quality of Life scale

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