

# Efficacy and Safety of Phytosomal Curcumin in Non-Alcoholic Fatty Liver Disease: A Randomized Controlled Trial

## Authors

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## ABSTRACT

**Objective** Non-alcoholic fatty liver disease (NAFLD) is a common liver disease characterized by excess lipid deposition in the hepatic tissue and subsequent oxidative and inflammatory damage. Curcumin is a dietary polyphenol with lipid-modifying, antioxidant and anti-inflammatory properties. This study aimed to evaluate the efficacy and safety of supplementation with phytosomal curcumin in subjects with NAFLD.

**Methods** Patients diagnosed with NAFLD (grades 1–3 according to liver ultrasonography) were randomly assigned to the curcumin (phytosomal form; 1 000 mg/day in 2 divided doses) (n = 50) or placebo group (n = 52) for a period of 8 weeks. All patients received dietary and lifestyle advises before the start of trial. Anthropometric measurements, hepatic enzymes, and liver ultrasonography were assessed at baseline and after 8 weeks of follow-up.

**Results** 87 subjects (n = 44 and 43 in the curcumin and control group, respectively) completed the trial. Supplementation with curcumin was associated with a reduction in body mass index ( $-0.99 \pm 1.25$  vs.  $-0.15 \pm 1.31$  in the curcumin and placebo groups, respectively;  $p = 0.003$ ) and waist circumference ( $-1.74 \pm 2.58$  vs.  $-0.23 \pm 3.49$  in the curcumin and placebo groups, respectively;  $p = 0.024$ ). Ultrasonographic findings were improved in 75.0% of subjects in the curcumin group, while the rate of improvement in the control group was 4.7% ( $p < 0.001$ ). Serum levels of aspartate aminotransferase and alanine aminotransferase were reduced by the end of trial in the curcumin group ( $p < 0.001$ ) but elevated in the control group ( $p < 0.001$ ). Curcumin was safe and well tolerated during the course of trial.

**Conclusion** Short-term supplementation with curcumin improves liver fat and transaminase levels in patients with NAFLD.

## Introduction

Non-alcoholic fatty liver disease (NAFLD) is a common hepatic disease characterized by fatty infiltration in hepatocytes [1]. The prevalence NAFLD ranges from 15–30% in the overall Western populations [2–5], while its prevalence increases to 50–90% in obese individuals, more than 70% in type 2 diabetic patients and nearly 100% in those subjects with both conditions [6]. Excessive fat accumulation and insulin resistance appear to play important roles in the pathogenesis of NAFLD [7]. NAFLD encompasses a spectrum of liver abnormalities starting from simple hepatic steatosis, steatohepatitis, liver fibrosis and cirrhosis which can finally progress to hepatocellular carcinoma [8]. Although pathogenesis of NAFLD is complex and remains uncertain, it has been hypothesized that lipid

deposition in the liver is the primary event involved and subsequently a variety of secondary processes, such as oxidative stress, lipid peroxidation, inflammatory response, hepatic fibrosis and apoptosis, are triggered [9]. Owing to the increased global rates of obesity, a condition that is strongly associated with NAFLD, development of novel effective therapies with minimal side-effects to control or slow the progression of this disease is required.

Curcumin is a yellow phenolic compound that was initially isolated from *Curcuma longa* L. (turmeric) rhizomes in 1815 [10]. Curcumin has been shown to possess numerous pharmacological effects including antioxidant, anticarcinogenic, anti-inflammatory, antidepressant, lipid-modifying, anti-arthritis, properties have been described for this polyphenol [11–23]. Numerous pharmaco-

logical properties of curcumin are due to the interaction of this natural product with various molecular targets such as enzymes, receptors, transcription factors, growth factors, cytokines, adipokines, and other important biomolecules [24–29]. Furthermore, this polyphenol improves insulin sensitivity [15, 30, 31] and mitochondrial function [32], suppresses adipogenesis [33], reduces inflammation [34, 35], fibrosis [36], and oxidative stress [37–39], that are causal risk factors of NAFLD. More importantly, experimental studies in rodents with diet-induced NAFLD have suggested that curcumin treatment can reduce hepatic lipid accumulation and progression of NAFLD to non-alcoholic steatohepatitis (NASH) [40, 41]. In clinical practice, although there is evidence on the efficacy of curcumin supplementation in lowering both plasma triglyceride and cholesterol levels [42–46], there has been no study targeting subjects with NAFLD. Hence, the aim of the present study was to evaluate the efficacy and safety of supplementation with curcumin in subjects with NAFLD.

## Materials and Methods

### Subjects

Studied subjects were selected from adults referring to the Gastroenterology and Hepatology Clinic of the Baqiyatallah Hospital (Tehran, Iran) for whom a diagnosis of NAFLD (grades 1–3) was made according to liver ultrasonography. Exclusion criteria were pregnancy or breastfeeding, NAFLD secondary to alcohol consumption, smoking, consumption of hypoglycemic, hypolipidemic and anti-inflammatory medications as well as any drug known to affect hepatic function, and presence of hepatitis, coronary, renal, pulmonary and thyroid diseases. All patients received dietary and lifestyle advises before the start of trial.

Eligible subjects were randomly (via alternative assignment to capsules bottles coded as A or B) allocated to the curcumin (1 000 mg/day in 2 divided doses) (n = 50) or control (n = 52) group. Curcumin was administered in the form of 500 mg capsules, and patients were advised to take the capsules after meal. Administered curcumin had a phytosomal formulation (Meriva®; Indena S.p.A, Milan, Italy) that contained a complex of curcumin and soy phosphatidylcholine in a 1:2 weight ratio, and 2 parts of microcrystalline to improve flowability, with an overall content of curcumin in the final product of around 20% [47].

The study protocol was given approval by the institutional Ethics Committee and written informed consent was obtained from participants. The clinical trial protocol has been registered under the Iranian Registry of Clinical Trials (IRCT) ID: IRCT2015122525641N2.

### Anthropometric measurements

Height and weight of subjects were measured with the accuracy of 0.1 cm and 0.1 kg, respectively. Measurements were performed in the standing position, with minimal clothing and no shoes at baseline and the 8<sup>th</sup> week. Body mass index was calculated as weight (kg) divided by height (m) squared.

### Blood sampling and biochemical measurements

Fasted blood samples were collected from the drawn from a cubital vein at baseline and after 8 weeks of supplementation. Blood samples were centrifuged for 10 min at a speed of 2 000–1 500 rpm

to separate the serum. Serum samples were kept at  $-80 + ^\circ\text{C}$  until analyses. Serum levels of alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), total and direct bilirubin were measured at baseline and at the end of study using routine enzymatic assays with commercial kits (Pars Azmoon, Tehran, Iran).

### Liver doppler sonography

Sonography of the liver is performed when the patient has fasted for 8–12 h before the examination. The examination is performed using 3.5- or 5-MHz convex probe.

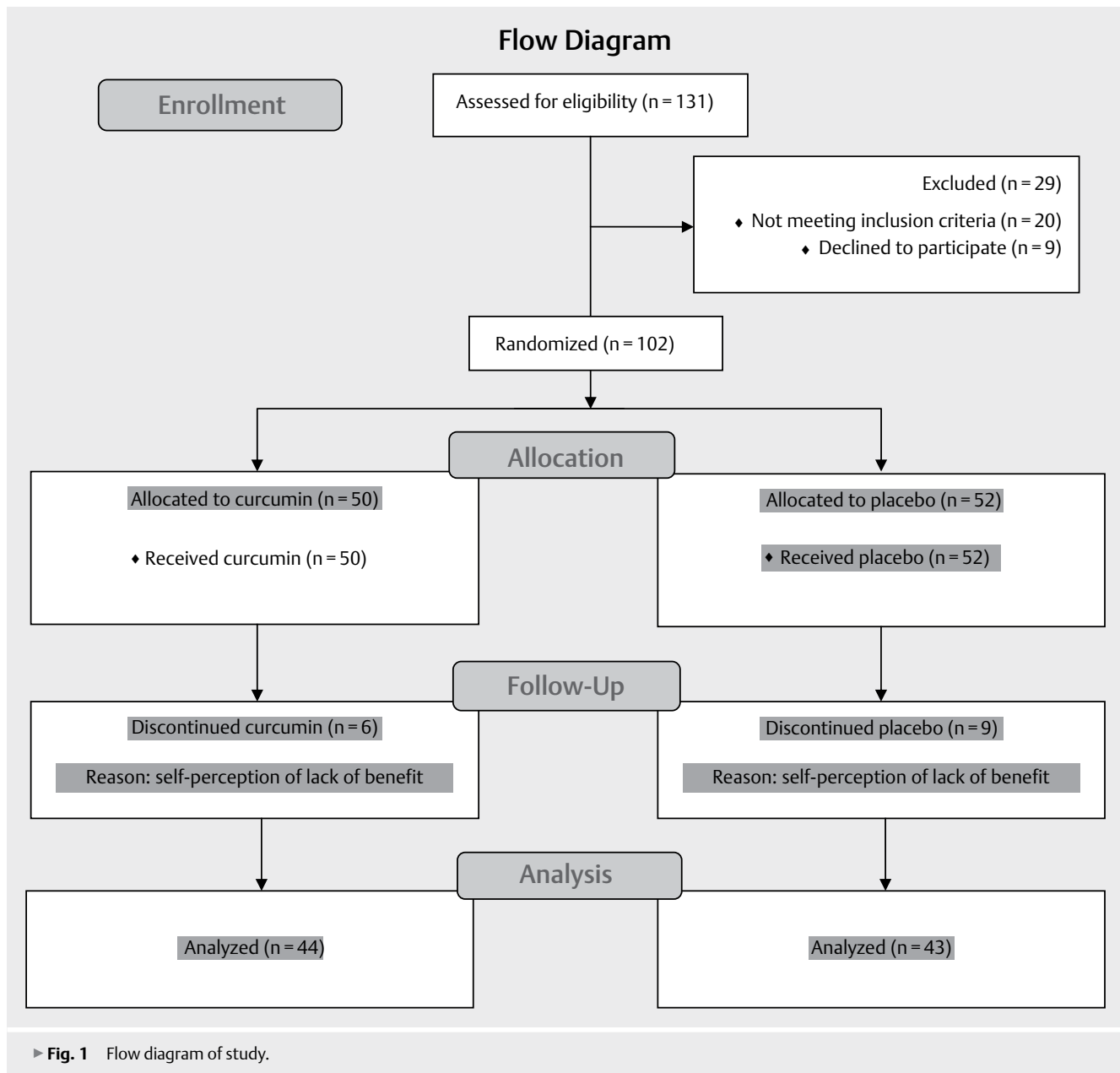
Liver fat content and the severity of hepatic steatosis was evaluated at baseline and after 8 weeks of supplementation using ultrasonography with a Mindray DC-8 diagnostic ultrasound system (convex 3.5–5.0 MHz) at the beginning and the end of the study. Ultrasound assessments were performed by the same expert radiologist blinded to the type of allocation, and with the same instrument. Assessments were performed in the fasted state (8–12 h), with the subjects in the supine position.

Right and left lobes of the upper and lower surfaces of liver were studied. Echogenicity of the liver, the presence or absence of bulky tumors cystic or solid and calcification was assessed. Intrahepatic bile ducts, portal vein and hepatic artery were evaluated. Hepatic steatosis was graded as 0 (lack of fat accumulation), 1 (mild increase in echogenicity with normal visualization of the diaphragm and intrahepatic vessel borders), 2 (moderate increase in echogenicity with slightly impaired visualization of the diaphragm and intrahepatic vessel borders), and 3 (severe increase in echogenicity with markedly impaired visualization of the diaphragm, intrahepatic vessel borders and the posterior portion of the right hepatic lobe).

The portal vein was evaluated in a sagittal oblique view demonstrating the vessels' longest axis. The antegrade hepatopetal flow throughout the entire cardiac cycle was considered as the portal vein flow. The length of the liver on the sagittal view in the mid-clavicular line was considered as liver size and was measured in the left posterior-oblique position.

### Statistical analysis

Statistical analyses were performed using the SPSS software version 11.5 (SPSS Inc., Chicago, Illinois, USA). The study population size was estimated at the significance level of 95%, with a power of 80% and an effect size of 0.7 for AST and ALT. Data were expressed as mean  $\pm$  SD or number (%). Within-group comparisons were performed using paired samples t-test (for normally distributed data) or Wilcoxon signed-ranks test (for non-normally distributed data). Between-group comparisons were performed using independent samples t-test (for normally distributed data) or Mann-Whitney U test (for non-normally distributed data). Categorical variables were compared using Fisher's Exact test. Binary logistic regression analysis was used to adjust for the effect of potential confounders on the association between curcumin supplementation and changes the severity of NAFLD according to ultrasonographic findings. For this analysis, change in the ultrasonographic findings (categorized as either improvement or lack of improvement) was entered into the model as dependent variable. A p-value of  $< 0.05$  was considered as statistically significant in all analyses. All analyses were performed per protocol.



## Results

### Baseline characteristics

86 subjects completed the study. There were 6 drop-outs in the curcumin group and 9 in the placebo group. All drop-outs were because of stopping the medication owing to the self-perception of lack of benefit (► **Fig. 1**). Drop-out rate did not differ between the study groups ( $p = 0.579$ ). The study groups were comparable in terms of age, gender, BMI, SBP, DBP, ALT, AST, ALP, total and direct bilirubin, smoking history and frequency of type 2 diabetes, coronary heart disease, obesity, hypertension, hyperlipidemia and hypertension (► **Table 1**). However, there was a significant baseline difference in waist circumference between the study groups (► **Table 1**). The severity of fatty liver based on parenchymal echogenicity and hepatic vein flow were comparable between the

groups, yet portal vein diameter and liver volume were different (► **Table 1**).

### Anthropometrics and blood pressure

Supplementation with curcumin was associated with significant reductions in BMI ( $p < 0.001$ ) and waist circumference ( $p < 0.001$ ) by the end of trial; however, no significant anthropometric change was observed with placebo ( $p > 0.05$ ) (► **Table 2**). Between-group comparison of changes revealed a significant difference between curcumin and placebo groups with respect to BMI ( $p = 0.003$ ) and waist circumference ( $p = 0.024$ ) (► **Table 3**). With respect to blood pressure, there was no significant change in SBP and DBP in any of the study group by the end of trial (► **Table 2**), nor was there any significant difference between the groups in terms of blood pressure changes during the course of study ( $p > 0.05$ ) (► **Table 3**).

► **Table 1** Comparison of baseline and demographic characteristics between the study groups.

	Curcumin	Placebo	p-Value
<b>N</b>	44	43	
<b>Female (%)</b>	20 (45.5)	16 (37.2)	0.516
<b>Age (y)</b>	44.98 ± 12.59	47.21 ± 10.29	0.369
<b>BMI (kg/m<sup>2</sup>)</b>	28.97 ± 3.42	29.07 ± 3.48	0.899
<b>Waist circumference (cm)</b>	97.07 ± 11.04	101.90 ± 11.43	0.048
<b>SBP (cmHg)</b>	12.00 ± 0.94	12.32 ± 1.07	0.126
<b>DBP (cmHg)</b>	7.82 ± 0.62	8.02 ± 0.46	0.116
<b>Total bilirubin (mg/dL)</b>	0.73 ± 0.17	0.77 ± 0.23	0.354
<b>Direct bilirubin (mg/dL)</b>	0.42 ± 0.15	0.36 ± 0.14	0.075
<b>ALT (U/L)</b>	35.46 ± 22.97	36.81 ± 24.32	0.790
<b>AST (U/L)</b>	27.63 ± 11.35	27.44 ± 10.01	0.934
<b>ALP (U/L)</b>	172.52 ± 40.65	170.77 ± 40.80	0.841
<b>Hepatic vein flow velocity (cm/sec)</b>	17.15 ± 2.16	17.29 ± 3.44	0.829
<b>Portal vein diameter (mm)</b>	11.41 ± 1.18	10.54 ± 1.99	0.015
<b>Liver size (mm)</b>	154.75 ± 11.62	160.12 ± 11.89	0.036
<b>Obesity (%)</b>	12 (27.3)	14 (32.6)	0.644
<b>Hypertension (%)</b>	9 (20.5)	12 (27.9)	0.461
<b>Dyslipidemia (%)</b>	21 (47.7)	29 (67.4)	0.083
<b>Diabetes (%)</b>	11 (25.0)	10 (23.3)	1.000
<b>CHD (%)</b>	6 (13.6)	6 (14.0)	1.000

Values are expressed as mean ± SD or number (%). SBP: systolic blood pressure; DBP: diastolic blood pressure; ALT: alanine aminotransferase; AST: aspartate aminotransferase; ALP: alkaline phosphatase; CHD: coronary heart disease

## Liver doppler solography

Comparison of liver ultrasonographic findings revealed a significant improvement in the curcumin vs. placebo group ( $p < 0.001$ ) (► **Table 4**). Ultrasonographic findings were improved in 75.0% of subjects in the curcumin group, while the rate of improvement in the placebo group was 4.7% ( $p < 0.001$ ). The frequency of increased liver fat content in the curcumin group was 4.5% but 25.6% of subjects in the placebo group had their liver fat content increased ( $p = 0.007$ ) (► **Table 4**).

Findings of Doppler solography indicated an increase in hepatic vein flow ( $p < 0.001$ ) accompanied by reduction of portal vein diameter ( $p < 0.001$ ) and liver volume ( $p = 0.001$ ) following treatment with curcumin, yet reverse effects were observed in the placebo group (► **Table 2**). Between-group comparisons revealed significant improvements in hepatic vein flow ( $p < 0.001$ ), portal vein diameter ( $p < 0.001$ ) and liver volume ( $p < 0.001$ ) in the curcumin vs. placebo group (► **Table 3**).

## Liver enzymes and bilirubin

Consistent with the findings of liver ultrasonography, serum levels of AST and ALT were reduced by the end of trial in the curcumin group ( $p < 0.001$ ) but elevated in the placebo group ( $p < 0.001$ ) (► **Table 2**). The effect of curcumin in reducing serum AST ( $p < 0.001$ ) and ALT ( $p < 0.001$ ) levels was also significant in the between-group comparison (► **Table 3**). Comparison of baseline vs. end-trial serum ALP concentrations did not reveal a significant alteration in any of the study groups (► **Table 2**), nor was there a significant difference between the groups in terms of ALP changes during the study period ( $p > 0.05$ ) (► **Table 3**). With respect to total and direct bilirubin concentrations, no significant difference was observed between curcumin and placebo groups ( $p > 0.05$ ) (► **Table 2, 3**).

## Regression analysis

Binary logistic regression analysis was performed to adjust the ultrasonographic findings – as the primary efficacy measure – for baseline waist circumference, liver volume, portal vein diameter,

► **Table 2** Within-group comparison of biochemical parameters between curcumin and placebo groups.

	Curcumin			Placebo		
	Pre-trial	Post-trial	p-Value	Pre-trial	Post-trial	p-Value
<b>BMI (kg/m<sup>2</sup>)</b>	28.97 ± 3.42	27.98 ± 3.05	<0.001	29.07 ± 3.48	28.92 ± 3.69	0.461
<b>Waist circumference (cm)</b>	97.07 ± 11.04	95.33 ± 10.77	<0.001	101.90 ± 11.43	101.66 ± 11.25	0.664
<b>SBP (cmHg)</b>	12.00 ± 0.94	12.07 ± 0.69	0.417	12.32 ± 1.07	12.23 ± 0.75	0.493
<b>DBP (cmHg)</b>	7.82 ± 0.62	7.89 ± 0.62	0.439	8.02 ± 0.46	7.91 ± 0.65	0.251
<b>Total bilirubin (mg/dL)</b>	0.73 ± 0.17	0.66 ± 0.17	<0.001	0.77 ± 0.23	0.72 ± 0.18	0.001
<b>Direct bilirubin (mg/dL)</b>	0.42 ± 0.15	0.40 ± 0.14	0.162	0.36 ± 0.14	0.37 ± 0.14	0.793
<b>ALT (U/L)</b>	35.46 ± 22.97	24.85 ± 12.84	<0.001	36.81 ± 24.32	41.33 ± 23.97	<0.001
<b>AST (U/L)</b>	27.63 ± 11.35	20.68 ± 6.65	<0.001	27.44 ± 10.01	31.23 ± 12.80	<0.001
<b>ALP (U/L)</b>	172.52 ± 40.65	172.91 ± 39.85	0.912	170.77 ± 40.80	172.59 ± 43.37	0.399
<b>Hepatic vein flow velocity (cm/sec)</b>	17.15 ± 2.16	21.35 ± 3.35	<0.001	17.29 ± 3.44	15.04 ± 3.88	<0.001
<b>Portal vein diameter (mm)</b>	11.41 ± 1.18	10.70 ± 1.05	<0.001	10.54 ± 1.99	11.49 ± 1.61	<0.001
<b>Liver size (mm)</b>	154.75 ± 11.62	152.82 ± 10.17	0.003	160.12 ± 11.89	161.88 ± 27.03	<0.001

Values are expressed as mean ± SD. SBP: systolic blood pressure; DBP: diastolic blood pressure; ALT: alanine aminotransferase; AST: aspartate aminotransferase; ALP: alkaline phosphatase

presence of dyslipidemia and serum direct bilirubin concentrations as potential confounders of treatment response. Selection of confounders was based on the baseline difference between curcumin and placebo groups using a *p*-value cut-off of 0.1. The effect of curcumin in reducing liver fat remained significant (*p* < 0.001) after adjustment for the above-mentioned confounders (► **Table 5**). The crude unadjusted effect of curcumin supplementation on the improvement of liver fat content was 61.50 (95% CI: 12.73, 297.04) (*p* < 0.001).

## Safety

Curcumin was safe and well tolerated in this trial. There was no report of severe adverse events.

## Discussion

Findings of the present study suggest that supplementation with phytosomal curcumin is well tolerated and significantly reduces fatty liver parameters in individuals with NAFLD. This is consistent with the results of previous studies showing that curcumin regu-

lates hepatic lipogenesis through AMP-activated protein kinase activation which leads to inhibition of hepatic lipid accumulation [48]. Thus, curcumin can blunt the “first hit” of NAFLD pathogenesis which involves development of hepatic steatosis [9]. Taking both ultrasonographic and biochemical (serum transaminase changes) findings together, it appears that the efficacy of curcumin supplementation in patients with NAFLD is not only statistically significant but also clinically relevant owing to the high improvement rate (75.0%) that was observed. However, a more definite judgment on the clinical relevance of the effects necessitates longitudinal designs assessing the efficacy of curcumin in preventing or slowing the progression of NAFLD into severe outcomes such as NASH and cirrhosis.

Several studies have indicated potential benefits of curcumin in the prevention and treatment of obesity, diabetes, and metabolic syndrome [49]. Curcumin decreases the production of reactive oxygen species by inhibition of hepatic stellate cell activation and reduction of tissue inhibitor of metalloprotease-1 release [50]. Another possible mechanism of action of curcumin may involve peroxisome proliferator-activated receptor- $\gamma$  (PPAR- $\gamma$ ) activation which can improve insulin sensitivity [51]. Moreover, the potent antioxidant effect of curcumin in lowering lipid peroxidation and oxidative stress in different tissues including the liver has been previously described [52]. These antioxidant effects of curcumin may be explained by its metal chelating ability modulatory effect on the expression of antioxidant enzymes such as superoxide dismutase, catalase and glutathione peroxidase [53, 54].

Different histological changes may occur during NAFLD including macrovesicular steatosis, lobular inflammatory infiltration, hepatocellular ballooning, formation of Mallory bodies, and perisinusoidal fibrosis [55–58]. It has been demonstrated that curcumin supplementation reduces aminotransferases levels and improves both steatosis and inflammation in liver tissues [59]. Moreover, curcumin inhibits NF- $\kappa$ B activation, decreases downstream induction of ICAM-1, COX-2 and MCP-1, and reduces intrahepatic gene expression of monocyte chemoattractant protein-1, CD11b, procollagen type I, and tissue inhibitor of metalloprotease-1 leading to the mitigation of the development and progression of hepatic inflammation and fibrosis [50, 60]. Also, curcumin significantly decreases serum levels of TNF- $\alpha$  and IL-6 [61], 2 pro-inflammatory cytokines involved in the “second hit” of the pathogenesis of NAFLD [9]. Lifestyle modification through adherence to a healthy diet and optimal physical activity is the first step in the prevention and treatment of NAFLD [62, 63]. In the present study, all patients received dietary and lifestyle advises. Along with the observed im-

► **Table 3** Between-group comparison of biochemical parameters between curcumin and placebo groups.

	Curcumin	Placebo	<i>p</i> -Value
BMI (kg/m <sup>2</sup> )	-0.99 ± 1.25	-0.15 ± 1.31	0.003
Waist circumference (cm)	-1.74 ± 2.58	-0.23 ± 3.49	0.024
SBP (cmHg)	0.07 ± 0.59	-0.08 ± 0.96	0.185
DBP (cmHg)	0.07 ± 0.59	-0.12 ± 0.66	0.116
Total bilirubin (mg/dL)	-0.07 ± 0.08	-0.06 ± 0.11	0.271
Direct bilirubin (mg/dL)	-0.02 ± 0.10	0.005 ± 0.12	0.271
ALT (U/L)	-10.61 ± 15.49	4.51 ± 7.40	<0.001
AST (U/L)	-6.95 ± 7.47	3.79 ± 6.43	<0.001
ALP (U/L)	0.39 ± 22.96	1.82 ± 14.00	0.727
Hepatic vein flow velocity (cm/sec)	4.20 ± 2.77	-2.25 ± 2.78	<0.001
Portal vein diameter (mm)	-0.70 ± 0.73	0.95 ± 1.43	<0.001
Liver size (mm)	-1.93 ± 4.00	1.76 ± 22.89	<0.001

Values are expressed as mean ± SD. SBP: systolic blood pressure; DBP: diastolic blood pressure; ALT: alanine aminotransferase; AST: aspartate aminotransferase; ALP: alkaline phosphatase

► **Table 4** Comparison of NAFLD severity within and between the study groups.

		Curcuminoids			Placebo			<i>p</i> -Value *
		Before	After	<i>p</i>	Before	After	<i>P</i>	
NAFLD severity	Grade 0	0 (0)	15 (34.1)	<0.001	0 (0)	1 (2.3)	0.013	<0.001
	Grade 1	17 (38.6)	21 (47.7)		17 (39.5)	10 (23.3)		
	Grade 2	23 (52.3)	6 (13.6)		19 (44.2)	21 (48.8)		
	Grade 3	4 (9.1)	2 (4.5)		7 (16.3)	11 (25.6)		

\* Comparison of change values between the study groups

► **Table 5** Binary logistic regression analysis for the effect of curcumin supplementation on liver fat content after adjustment for potential confounders.

Variables entered in the model	Odds ratio	95% CI	p-Value
Baseline waist circumference	1.02	0.96, 1.09	0.432
Baseline liver size	0.98	0.92, 1.03	0.420
Baseline portal vein diameter	0.78	0.52, 1.18	0.237
Curcumin treatment	96.50	14.30, 651.22	<0.001

Change in the ultrasonographic findings (categorized as either improvement or lack of improvement) was entered into the model as dependent variable, and the variables listed in the table were entered as independent factors

Improvements in the ultrasonographic and biochemical indices, curcumin supplementation resulted in a significant reduction in anthropometric indices including BMI and waist circumference. It is worth noting that curcumin has been shown to enhance physical performance and reduce physiological fatigue and [64] this might have contributed to the therapeutic effects of curcumin in reducing BMI and other indices of NAFLD.

On the other hand, our results support the efficacy and safety of curcumin supplementation as it has been previously reported by several studies in both healthy subjects and patients with different diseases [65–68].

The main limitation of the present study was that histological changes were not assessed because liver biopsy is an invasive diagnostic test limited by ethical issues; therefore, hepatic findings were evaluated by hepatic ultrasound which has been shown to have high sensitivity, specificity and accuracy for the assessment of NAFLD [69, 70]. Besides, some other indices such as gamma-glutamyl-transferase and spleen size that could serve as indices of NAFLD progression were not assessed in this study and deserve to be measured in future trials of curcumin. Another limitation is the short duration of follow-up which impedes judgment on the long-term efficacy of curcumin supplementation particularly in terms of reducing vascular and hepatic outcomes. Nonetheless, that curcumin exerts protective effects as early as 8 weeks of supplementation is a proof for the relevance of supplementation with this agent. Finally, given the small population size, some between-group differences in baseline characteristics were present in spite of randomization. Hence, confirmatory evidence from intention-to-treat analyses in larger trials would be ideal to confirm the positive findings observed in the present study.

In conclusion, results of this study revealed that short-term curcumin supplementation improves sonographic findings of NAFLD and hepatic transaminase levels. However, future studies are warranted to elucidate if the anti-steatotic properties of curcumin are dose-dependent, and also the value of curcumin in reducing cardiovascular and hepatic endpoints such as cirrhosis and hepatocellular carcinoma.

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## Conflict of Interest

The authors have no competing interest to declare.

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