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Original Research Article

Bioavailable curcumin (CurcuBoost®) as an adjunct therapy for liver dysfunction: a randomized controlled study

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ABSTRACT

Background: Chronic liver dysfunction is associated with persistent liver enzyme elevations, hepatocellular injury and impaired quality of life. Curcumin has demonstrated hepatoprotective properties. This study aimed to evaluate the efficacy, safety and tolerability of CurcuBoost®, a bioavailable curcumin formulation, as an adjunct to standard of care (SOC) in adults with deranged liver function.

Methods: In this randomized, open-label, parallel-group trial, 50 adults with biochemical evidence of liver dysfunction were randomized (1:1) to CurcuBoost® (500 mg orally) plus SOC or SOC alone for 12 weeks. The primary endpoint was change in serum liver enzymes from baseline to Week 12. Secondary endpoints included changes in liver disease quality of life (LDQOL) scores and safety outcomes. Analyses used a modified intention-to-treat approach with ANCOVA.

Results: All 50 participants completed the study. Compared with SOC alone (n=25), CurcuBoost® plus SOC (n=25) produced greater reductions in alanine aminotransferase (−109.72 vs −93.39 IU/l; p=0.0012), aspartate aminotransferase (−107.07 vs −86.81 IU/l; p=0.0001), alkaline phosphatase (−354.42 vs −311.75 IU/l; p<0.0001) and gamma-glutamyl transferase (−44.46 vs −38.78 IU/l; p=0.0364) at Week 12. Total bilirubin also decreased significantly more with CurcuBoost® (p=0.0008), while renal function remained stable. LDQOL scores improved in both groups, with greater improvement in the CurcuBoost® plus SOC group (p<0.0001). No serious adverse events were reported.

Conclusions: Adjunctive treatment with CurcuBoost® significantly improved liver enzyme profiles and liver-related quality of life compared with standard of care alone, with an excellent safety profile.

Keywords: Curcumin, Curcuminoids, CurcuBoost, Hepatoprotection, Liver dysfunction, Liver enzymes, Non-alcoholic fatty liver disease

INTRODUCTION

Liver dysfunction is a major global health problem characterized by impaired hepatic metabolism, inflammation, oxidative stress and progressive tissue injury. It commonly arises from conditions such as non-alcoholic fatty liver disease (NAFLD), viral hepatitis, alcoholic liver disease and cirrhosis, all of which contribute substantially to global morbidity and mortality.¹⁻⁴ Clinically, liver dysfunction is identified by elevated serum liver enzymes, including alanine

aminotransferase (ALT) and aspartate aminotransferase (AST), markers of hepatocellular injury and alkaline phosphatase (ALP) and gamma-glutamyltransferase (GGT), indicators of cholestasis.

Persistent elevations in these biomarkers reflect ongoing hepatic injury and increased risk of disease progression.^{1,2,5} Lifestyle modification remains the cornerstone of management, particularly for metabolic liver disease; however, dietary and weight-loss interventions often fail to sustain normalization of liver

enzymes or prevent disease progression.^{6,7} Pharmacological therapies mainly target underlying etiologies or metabolic risk factors and have limited ability to restore hepatic function.⁸ Therefore, safe and effective adjunctive therapies that complement the SOC are needed to improve outcomes in patients with liver dysfunction. Oxidative stress and chronic inflammation are central to liver injury. Excessive generation of reactive oxygen species (ROS) and pro-inflammatory cytokines promotes hepatocellular damage, lipid peroxidation, stellate cell activation and fibrogenesis, leading to progressive hepatic impairment and the development of steatohepatitis, fibrosis and cirrhosis.⁹

Curcumin, the principal polyphenolic compound derived from *Curcuma longa* (turmeric), has gained considerable interest due to its antioxidant, anti-inflammatory and anti-fibrotic properties.^{9,10} It modulates several molecular pathways involved in liver injury, including inhibition of nuclear factor- κ B (NF- κ B), suppression of pro-inflammatory cytokines, reduction of oxidative stress and inhibition of hepatic stellate cell activation.⁹⁻¹¹ Preclinical studies demonstrate that curcumin can reduce increased liver enzyme levels, improve histopathological features such as steatosis and necroinflammation and enhance antioxidant defenses.^{9,10,12}

Clinical studies further suggest potential benefits of curcumin supplementation in liver disorders. Randomized controlled trials in patients with NAFLD and related metabolic conditions report significant reductions in ALT and AST levels compared with placebo.¹³⁻¹⁵ Some studies also indicate improvements in hepatic steatosis, inflammatory markers and antioxidant status.¹⁶⁻²⁰ However, interpretation of these findings is limited by heterogeneity in study design, dosage and curcumin formulations. Importantly, the poor oral bioavailability of conventional curcumin preparations has hindered consistent clinical translation, emphasizing the need for optimized formulations with improved absorption.

CurcuBoost® is a novel curcumin formulation developed to improve bioavailability and maximize biological activity. Given curcumin's well-established mechanistic rationale, supportive preclinical data and emerging clinical evidence and considering the persistent unmet need for effective adjunctive therapies in liver dysfunction, the present randomized clinical trial was undertaken to evaluate the efficacy, safety and tolerability of CurcuBoost® when administered in addition to SOC.

The primary objective was to assess changes in serum liver enzyme levels (ALT, AST, ALP and GGT) over a 12-week intervention period, with secondary objectives including evaluation of liver-related quality of life and safety outcomes. This study aims to provide robust clinical evidence to clarify the role of a bioavailable curcumin formulation as an adjunct to the management of patients with deranged liver function.

METHODS

Study design

This study was a randomized, open-label, parallel-group clinical trial conducted at a single center between 28 February 2025 and 5 August 2025.

Ethical considerations

The study was approved by the Central Independent Ethics Committee (IEC Reference No. CIEC060225 dated 11th Feb 2025). The trial was prospectively registered with the Clinical Trials Registry prior to participant enrolment. All study procedures were conducted in accordance with the International Council for Harmonization Good Clinical Practice (ICH-GCP) guidelines, the Indian Council of Medical Research (ICMR) National Ethical Guidelines for Biomedical and Health Research Involving Human Participants (2017) and applicable regulatory requirements. Written informed consent was obtained from all participants before enrolment.

Study population

Eligible participants were adults aged 18 to 75 years with biochemical evidence of liver dysfunction, defined as total bilirubin greater than the upper limit of normal (ULN) and $\leq 3 \times$ ULN, ALT, AST or ALP $> 3 \times$ ULN and $\leq 5 \times$ ULN or Gamma-Glutamyl Transferase $> \text{ULN}$ and $\leq 2 \times$ ULN. Participants were required to have stable chronic liver conditions, including but not limited to non-alcoholic fatty liver disease, non-alcoholic steatohepatitis, chronic hepatitis or early fibrosis/cirrhosis, without evidence of acute hepatic decompensation. Key exclusion criteria included severe liver disease and uncontrolled comorbidities. The eligibility criteria were prospectively specified in the study protocol and were based on standard clinical assessment and management principles for chronic liver disease.²¹

Randomization and treatment

Participants meeting eligibility criteria were randomized in a 1:1 ratio via a computer-generated sequence to receive either CurcuBoost® plus SOC or SOC alone for 12 weeks (84 $\pm$ 3 days). CurcuBoost® was administered orally as 500 mg capsules. Standard of care was provided per protocol and in the investigator's judgment. All participants were encouraged to maintain compliance with the assigned treatment regimen and compliance was monitored throughout the study.

Assessments and endpoints

Clinical laboratory evaluations included serum liver enzyme measurements, including ALP, AST, ALT and GGT. Other biochemical parameters assessed were bilirubin and renal function tests. All these parameters were assessed at baseline and at weeks 6 and 12. Vital

signs, physical examinations and adverse event monitoring were conducted at each study visit. Liver disease-related quality of life was assessed using the LDQOL 1.0 questionnaire developed by Gralnek et al.²² The questionnaire was administered at baseline and follow-up visits according to the published methodology.

The primary efficacy endpoint was the change in serum liver enzyme levels from baseline to Week 12. Secondary endpoints included changes in LDQOL score and the incidence of adverse events and serious adverse events throughout the study period.

Sample size calculation

The sample size was planned to detect a clinically meaningful mean difference of 15-20 units in the ALT/AST between the two groups. The expected standard deviation was approximately 20 in each group. Using a two-sided significance level of 5% ($\alpha=0.05$) and 80% power ($1-\beta=0.80$), 25 subjects per group were estimated to be sufficient to detect the expected treatment effect.

Statistical analysis

All statistical analyses were predefined and conducted according to a modified intention-to-treat (mITT) principle. The mITT population included all randomized participants who received at least one dose of the study medication and had at least one post-baseline efficacy assessment. As no significant protocol deviations were identified during the study, the mITT and per-protocol analysis populations were identical. The safety population comprised all participants who received at least one dose of the study medication.

Continuous variables were summarized using descriptive statistics, including the number of observations, mean, standard deviation, median, minimum and maximum. Categorical variables were summarized as frequencies and percentages. Between-group comparisons of continuous outcomes were performed using Student's independent t-test. For outcomes expressed as change from baseline, analysis of covariance (ANCOVA) models was applied with baseline values included as covariates. Within-group comparisons were conducted using paired t-tests.

Categorical variables were compared between groups using the chi-square test or Fisher's exact test, as appropriate based on expected cell counts. Adverse events were summarized descriptively within the safety population. Events that increased in severity or newly occurred after initiation of study treatment were classified as treatment-emergent adverse events. For inferential analyses, the corresponding test statistics (t or F), degrees of freedom, p values and Cohen's d effect sizes were reported, as appropriate. All statistical tests were two-sided and p values < 0.05 were considered statistically significant. All analyses were performed using R statistical

software (version 4.4.1; R Foundation for Statistical Computing, Vienna, Austria).

RESULTS

Participant disposition and baseline characteristics

Fifty participants were randomized to receive CurcuBoost® in combination with SOC (n=25) or SOC alone (n=25). All randomized participants received treatment and completed the study, yielding 100% retention in both groups and full evaluability for mITT and safety analyses. Baseline demographic and disease characteristics were comparable between treatment arms, confirming successful randomization (Table 1). No statistically significant between-group differences were observed for age, sex distribution or BMI at enrolment and the clinical status of liver dysfunction was similar across groups, ensuring balanced comparison of outcomes.

Liver biochemistry (primary efficacy outcomes)

CurcuBoost® administered alongside SOC demonstrated superior hepatoprotective effects compared with SOC alone over the 12-week treatment period, with statistically and clinically meaningful improvements observed across key liver enzymes (Table 2).

Participants receiving CurcuBoost®+SOC showed significantly greater reductions from baseline to Week 12 in serum ALP, with a mean between-group difference of -42.67 IU/l (95% CI: -68.8 to -16.5; Cohen's d=0.93; p<0.0001). Similarly, AST decreased significantly more in the CurcuBoost® group (mean difference -20.26 IU/l; 95% CI: -32.9 to -7.6; d=0.90; p=0.0001) and ALT (mean difference -16.33 IU/l; 95% CI: -28.9 to -3.8; d=0.74; p=0.0012). These findings indicate large-to-moderate treatment effects, reflecting clinically meaningful improvements in hepatocellular function.

GGT also demonstrated a greater reduction in the CurcuBoost®+SOC arm. The between-group difference in change from baseline was -5.68 IU/l (95% CI: -14.8 to 3.5; d=0.35; p=0.0364), indicating a small-to-moderate effect size.

Total serum bilirubin levels were significantly lower at Week 12 in the CurcuBoost® + SOC group compared with SOC alone (mean difference -0.33 mg/dl; 95% CI: -0.51 to -0.15; d=1.05; p=0.0007), indicating a large treatment effect and improved hepatic clearance. Although change-from-baseline values showed high variability in the SOC group, the Week 12 comparison provides a more stable estimate of treatment effect.

In contrast, no statistically significant between-group difference was observed for serum creatinine (p=0.2830), indicating preserved renal function in both treatment groups. Overall, ANCOVA-adjusted analyses demonstrated that CurcuBoost®+SOC was associated with

significantly greater improvements in key liver biochemistry parameters compared with SOC alone. The consistency of findings across multiple endpoints, along with large effect sizes for ALP, AST, ALT and bilirubin, supports the robustness and clinical relevance of the observed hepatoprotective effects.

Quality of life (secondary efficacy outcomes)

Clinically meaningful improvements in patient-reported outcomes were observed in both treatment groups, with significantly greater benefits in the CurcuBoost®+SOC arm (Table 3). At week 12, participants receiving CurcuBoost®+SOC demonstrated a significantly greater reduction in LDQOL scores compared with SOC alone, with a mean between-group difference of -3.88 points (95% CI: -6.33 to -1.43; Cohen's $d=0.89$; $p < 0.0001$), indicating a large treatment effect and substantial improvement in liver disease-related quality of life. In contrast, no statistically significant difference was observed at Week 6 (mean difference -2.16 points; 95% CI: -6.73 to 2.41; $d=0.27$; $p=0.2823$), suggesting that the differences in treatment effects became more pronounced

over time. Consistent with these findings, the absolute LDQOL score at study completion was significantly lower in the CurcuBoost®+SOC group than in the SOC alone group ($p < 0.0001$), reflecting greater alleviation of liver disease-related symptoms and functional impairment. Overall, the observed improvements in LDQOL align with the biochemical outcomes, supporting a concordance between objective liver function recovery and patient-reported symptomatic and functional outcomes.

Safety

CurcuBoost®+SOC was well tolerated and safe. Only four mild treatment-emergent adverse events were reported, one in the CurcuBoost® + SOC arm (4.0%) and three in the SOC arm (12.0%), all resolving without medical intervention and none attributed to the investigational product (Table 4). There were no serious adverse events, deaths or discontinuations due to adverse events reported and no clinically meaningful changes in vital signs, hematology or renal function parameters were observed. These results support a favorable safety profile, consistent with existing data on curcumin-based interventions.

Table 1: Baseline demographic and clinical characteristics (mITT population, n=50).

Characteristic	CurcuBoost® + SOC (n=25)	SOC (n=25)	P value
Age (years), mean±SD	37.48±10.92	42.92±13.63	0.1263
Female sex, N (%)	11 (44.0%)	14 (56.0%)	0.3961
BMI (kg/m ²), mean±SD	25.20±1.70	25.75±1.62	0.2497
Height (cm), mean±SD	167.40±3.45	166.75±2.56	0.4550
Weight (kg), mean±SD	70.67±5.87	71.59±4.66	0.5423

Data presented as mean±SD and %(n) as specified. BMI: Body-mass-index, Pvalues <0.05 were considered statistically significant. SOC: Standard of care.

Table 2: Biochemical Outcomes (mITT Population n=50).

Outcomes	CurcuBoost®+SOC (n=25)	SOC alone (n=25)	Mean difference (95% CI)	Cohen's d	F-statistic	P value
ALP (IU/l)						
Baseline	430.55±35.62	445.38±32.45	—	—	2.37	0.1305
Week 12	76.13±21.51	133.63±50.39	—	—	27.53	<0.0001
ΔWeek 12	-354.42±43.01	-311.75±48.77	-42.67 (-68.8 to -16.5)	0.93	23.68	<0.0001
AST (IU/l)						
Baseline	136.76±18.67	138.13±16.10	—	—	0.08	0.7825
Week 12	29.69±9.22	51.32±22.99	—	—	19.06	0.0001
Δ Week 12	-107.07±19.88	-86.81±24.87	-20.26 (-32.9 to -7.6)	0.90	18.78	0.0001
ALT (IU/l)						
Baseline	145.24±16.94	146.46±16.45	—	—	0.07	0.7972
Week 12	35.52±15.82	53.07±19.75	—	—	12.02	0.0012
ΔWeek 12	-109.72±20.42	-93.39±23.54	-16.33 (-28.9 to -3.8)	0.74	11.85	0.0012
GGT (IU/l)						
Baseline	82.73±9.83	86.95±8.33	—	—	2.68	0.109
Week 12	38.27±15.89	48.17±12.88	—	—	5.85	0.0195
ΔWeek 12	-44.46±16.19	-38.78±16.06	-5.68 (-14.8 to 3.5)	0.35	4.64	0.0364
Total bilirubin (mg/dl)						
Baseline	1.68±0.29	2.00±2.01	—	—	0.63	0.4358

Continued.

Outcomes	CurcuBoost®+SOC (n=25)	SOC alone (n=25)	Mean difference (95% CI)	Cohen's d	F-statistic	P value
Week 12	0.74±0.18	1.07±0.40	—	—	13.88	0.0007
ΔWeek 12	-0.94±0.30	-0.93±1.99	-0.01 (-0.80 to 0.78)	0.01	12.88	0.0008

Data are presented as mean±standard deviation (SD). P values for baseline and post-baseline comparisons were calculated using Student's t-test. P values for change-from-baseline analyses were derived from ANCOVA models adjusting for baseline values. F-statistics for Baseline and Week 12 between-group comparisons are from Student's independent t-test ($F=t^2$; $df=1,48$); F-statistics for Δ Week 12 rows are the ANCOVA F-test for the group effect, adjusting for baseline values ($df=1,47$).

Table 3: LDQOL total score outcomes (mITT Population, n=50).

Assessment	CurcuBoost®+SOC (n=25)	SOC alone (n=25)	Mean difference (95% CI)	Cohen's d	F statistic	P value
Baseline	64.44±3.62	64.44±4.21	—	—	0.00	>0.9999
Week 6	47.20±8.35	49.36±5.19	—	—	1.21	0.2785
Week 12	10.28±2.13	14.16±3.00	—	—	27.84	<0.0001
ΔWeek 6	-17.24±9.79	-15.08±5.93	-2.16 (-6.73 to 2.41)	0.27	1.18	0.2823
ΔWeek 12	-54.16±4.78	-50.28±3.93	-3.88 (-6.33 to -1.43)	0.89	27.87	<0.0001

Data presented as Mean±SD. For baseline, week 6 and week 12, p-values were calculated using Student's t-test. P values for baseline and post-baseline comparisons were calculated using Student's t-test. P values for change-from-baseline analyses were derived from ANCOVA models adjusting for baseline values. F-statistics for Baseline, Week 6 and Week 12 between-group comparisons are from Student's independent t-test ($F=t^2$; $df=1,48$); F-statistics for Δ Week 6 and ΔWeek 12 rows are the ANCOVA F-test for the group effect, adjusting for baseline values ($df=1,47$).

Table 4: Summary of treatment-emergent adverse events (mITT population, n=50).

Outcome	CurcuBoost® + SOC (N=25)	SOC (N=25)
Subjects with ≥1 TEAE, n (%)	1 (4%)	3 (12%)
TEAEs, total	1 (4%)	3 (12%)
Most frequent event	Cough, 1(4%)	Fever, 3(12%)
Severity	Mild	Mild
Serious AEs	0	0
Treatment-related AEs	0	0
Discontinuations due to AEs	0	0

DISCUSSION

In this randomized clinical trial, adjunctive treatment with CurcuBoost® in combination with SOC resulted in significantly greater improvements in liver enzyme profiles, including ALT, AST, ALP and GGT, compared with SOC alone over a 12-week period. These biochemical benefits were accompanied by significant improvements in LDQOL scores, suggesting that enzyme normalization translated into meaningful patient-reported functional and symptomatic gains. Importantly, CurcuBoost® was well tolerated, with a low incidence of mild adverse events comparable to SOC, supporting its favorable safety profile.

The observed reductions in ALT and AST are consistent with a substantial body of clinical evidence supporting the hepatoprotective effects of curcumin. In a GRADE-assessed meta-analysis of 15 RCTs (905 participants), curcumin significantly reduced ALT (weighted mean difference -4.10) and AST (-3.27) versus control, although effects on ALP were not statistically significant across trials.¹³ Similarly, another meta-analysis reported

reductions in ALT and AST levels with curcumin/turmeric supplementation in NAFLD patients.²³ Our results align closely with this literature and further support curcumin's role in attenuating hepatocellular injury. Notably, the present study extends prior evidence by demonstrating significant improvements not only in transaminases but also in ALP and GGT.

Previous pooled analyses have shown less consistent effects of curcumin on cholestatic markers such as ALP and GGT, although subgroup analyses suggest that longer treatment duration and optimized formulations may enhance these outcomes.²³ The significant reductions in ALP and GGT observed in our cohort suggest that enhanced-bioavailability formulations such as CurcuBoost® may exert broader effects on liver biochemistry than conventional curcumin preparations, a finding that is supported by emerging clinical data on bioavailable curcumin formulations.²⁴ The hepatoprotective effects observed in this trial are biologically plausible in light of curcumin's well-characterized anti-inflammatory and antioxidant

properties. Curcumin has been shown to inhibit key pro-inflammatory signaling pathways, including nuclear factor- κ B (NF- κ B), reduce the production of inflammatory cytokines and attenuate oxidative stress by enhancing endogenous antioxidant defenses.^{9,25} These mechanisms directly target central drivers of liver injury across etiologies, including hepatocellular damage and fibrogenesis. Preclinical meta-analyses further support this mechanistic basis, demonstrating that curcumin significantly reduces ALT and AST while improving histological markers of inflammation and fibrosis in animal models of liver injury.²⁶

Additionally, curcumin's inhibitory effects on hepatic stellate cell activation provide a plausible explanation for its potential to slow fibrotic progression, even though histological outcomes were beyond the scope of the present study.²⁶ Collectively, these mechanistic insights provide a coherent biological framework for the enzyme reductions and clinical improvements observed with CurcuBoost®.

Beyond biochemical improvements, the significant enhancement in LDQOL scores observed with CurcuBoost® underscores the clinical relevance of adjunctive curcumin therapy. Chronic liver disease is frequently associated with impaired quality of life due to fatigue, physical limitations and psychological distress, which are not always captured by liver enzyme changes alone. Evidence from randomized trials in populations with liver disease has shown that curcumin supplementation can improve multiple quality-of-life domains, including physical functioning, emotional well-being and disease-specific symptom burden.²⁷ The observed improvements in LDQOL in our study are consistent with these findings and may be mediated by reductions in systemic inflammation and oxidative stress, mechanisms previously linked to symptom burden in metabolic liver disease.^{9,20}

CurcuBoost® demonstrated an excellent safety and tolerability profile, with only a single mild treatment-emergent adverse event reported in the intervention group. This finding aligns with prior clinical studies reporting that curcumin is generally safe across a wide range of doses and treatment durations, including in patients with liver disease.¹³ The favorable safety profile is particularly relevant given historical concerns regarding curcumin's limited bioavailability; novel formulations designed to enhance absorption, such as CurcuBoost®, appear to maintain safety while potentially improving clinical efficacy.

The findings of this study have important clinical implications. Given that many patients with chronic liver dysfunction fail to achieve adequate biochemical or symptomatic improvement with lifestyle modification and pharmacotherapy alone, adjunctive curcumin supplementation may offer a practical strategy to enhance treatment response. The observed improvements across multiple liver enzymes, including ALT, AST, ALP and GGT, suggest a broad hepatoprotective effect that may

translate into reduced risk of disease progression when applied in routine clinical practice. Furthermore, aligning biochemical benefits with quality-of-life improvements highlights the potential for curcumin-based interventions to deliver patient-centered benefits beyond surrogate laboratory markers.

Key strengths of this study include its randomized design, comprehensive assessment of multiple liver enzymes, incorporation of patient-reported quality-of-life outcomes and use of a standardized curcumin formulation with enhanced bioavailability.

However, several limitations of the study should be acknowledged. The 12-week duration, while sufficient to detect significant biochemical and quality-of-life changes, may not capture longer-term outcomes such as fibrosis regression or clinical disease progression. Additionally, the heterogeneous etiologies of liver dysfunction in the study population may limit generalizability to specific disease subgroups. Although prior meta-analyses have reported variable effects of curcumin on ALP and GGT.¹³ Our positive findings for these markers warrant confirmation in larger, multicenter trials with longer follow-up and stratification by disease etiology.

CONCLUSION

In conclusion, this randomized clinical trial demonstrates that CurcuBoost® administered as an adjunct to SOC significantly improves liver enzyme profiles and liver-related quality of life while maintaining an excellent safety profile. These findings are consistent with and extend existing evidence supporting curcumin's hepatoprotective effects. Future studies should focus on longer-term interventions, histological and mechanistic endpoints and stratified analyses across liver disease subtypes to further define the role of this bioavailable curcumin formulation in managing chronic liver dysfunction.

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Ethical approval: The study was approved by the Institutional Ethics Committee

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