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Original Research Article

Effect of LIV KRIT on Gut–Liver Axis Dysfunction in Patients with Early Non-Alcoholic Fatty Liver Disease: A Real-World Prospective Interventional Study

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Conflict of interest: Nil

Abstract

Background: Non-alcoholic fatty liver disease (NAFLD) represents the most prevalent chronic liver disorder globally, with its pathogenesis increasingly linked to gut–liver axis dysregulation. Conventional management remains limited to lifestyle modification, underscoring the need for evidence-based botanical interventions. Brahmanand's LIV KRIT is an advanced multi-herb Ayurvedic formulation specifically designed for liver care, combining 17 classical plant-derived and mineral constituents including Kalmegh (Andrographis paniculata), Bhringraj (Eclipta alba), Kutki (Picrorhiza kurroa), Bhumi Amla (Phyllanthus niruri), and Loh Bhasma, each with documented hepatoprotective and gut-modulatory properties.

Aim: To evaluate the effect of 12-week LIV KRIT supplementation on liver function tests, sonographic fatty liver grade, systemic inflammatory markers, and gut–liver axis parameters in patients with early NAFLD.

Methods: A prospective, single-arm, observational-interventional study was conducted at an outpatient setting enrolling 100 patients aged 40–70 years with ultrasound-confirmed NAFLD (Grade I–II) and mild elevation of liver enzymes. Participants received LIV KRIT capsules at the standard labelled dose for 12 weeks alongside standardised lifestyle counselling. Primary outcomes were changes in ALT, AST, GGT, and ALP at 12 weeks. Secondary outcomes included USG grade improvement, serum CRP, Gastrointestinal Symptom Rating Scale (GSRS) scores, waist circumference, lipid profile, and FIB-4 score.

Results: Significant reductions were observed in ALT (68.4 to 38.6 U/L; −43.5%, $p < 0.001$), AST (54.2 to 33.1 U/L; −38.9%, $p < 0.001$), GGT (52.7 to 31.4 U/L; −40.4%, $p < 0.001$), and ALP (98.3 to 72.1 U/L; −26.7%, $p < 0.001$). USG grade improvement was noted in 62% of participants. CRP declined by 52.3% ($p < 0.001$), GSRS total score by 46.1% ($p < 0.001$), and waist circumference by 5.4 cm ($p = 0.002$). A strong positive correlation was observed between gut symptom improvement and ALT reduction (Pearson $r = 0.71$, $p < 0.001$). No serious adverse events were reported.

Conclusion: LIV KRIT demonstrated clinically meaningful and statistically significant improvements in hepatic, metabolic, and gut–liver axis parameters in patients with early NAFLD over 12 weeks. The formulation represents a promising integrative therapeutic option warranting further randomised controlled evaluation.

Keywords: NAFLD; Gut–Liver Axis; Ayurveda; LIV KRIT; Hepatoprotective; Kalmegh; Bhringraj; Liver Function Tests; Gut Microbiome; Herbal Hepatology.

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Introduction

Non-alcoholic fatty liver disease (NAFLD) has emerged as a major global public health challenge, affecting an estimated 25–30% of the general adult population worldwide and representing the leading cause of chronic liver disease in most developed and developing countries [1]. Its clinical spectrum ranges from simple hepatic steatosis to non-alcoholic steatohepatitis (NASH), progressive fibrosis, cirrhosis, and hepatocellular carcinoma [2]. In India, the prevalence of NAFLD is estimated between 9% and 32%, with urban populations demonstrating figures as high as 46% in metabolically at-risk cohorts [3]. Despite its high prevalence, no pharmacological agent has yet received regulatory approval specifically for NAFLD, rendering lifestyle modification the cornerstone of current management [4].

The gut–liver axis has emerged as a central pathophysiological nexus in NAFLD. The liver receives nearly 70% of its blood supply via the portal vein, making it the primary recipient of gut-derived metabolites, endotoxins, and microbial antigens [5]. Gut dysbiosis—defined as qualitative and quantitative alterations in the intestinal microbiome—results in increased intestinal permeability, translocation of lipopolysaccharides (LPS) into the portal circulation, and activation of hepatic Toll-like receptor 4 (TLR4) signalling cascades, driving hepatic steatosis and inflammation [6]. Elevated systemic LPS and downstream inflammatory cytokines such as TNF-α, IL-6, and IL-1β have been consistently documented in NAFLD patients, correlating directly with disease severity 7. Concurrently, short-chain fatty acid (SCFA) deficiency arising from reduced Firmicutes abundance further compromises gut barrier integrity and hepatic lipid regulation 8. These observations firmly establish gut microbiome modulation as a rational therapeutic target in NAFLD management.

Classical Ayurvedic medicine conceptualised the liver (Yakrit) as the seat of Ranjaka Pitta and a primary organ of digestion and haematopoiesis, functioning in intimate anatomical and physiological relationship with the gastrointestinal tract (Koshtha) [9]. Polyherbal formulations targeting both hepatic and digestive function have therefore constituted a cornerstone of Ayurvedic hepatology for centuries. Several individual botanical agents employed in such formulations have now received modern scientific validation. Andrographis paniculata (Kalmegh) contains andrographolide, a labdane diterpenoid with potent anti-inflammatory, anti-steatotic, and NF-κB inhibitory activity documented in both in vitro and rodent NASH models [10]. Eclipta alba (Bhringraj) has demonstrated hepatocyte regeneration-promoting properties through Wnt/β-catenin pathway activation in murine models of liver injury [11]. Phyllanthus niruri (Bhumi Amla) has exhibited significant reductions in hepatic lipid accumulation and oxidative stress markers in high-fat diet-induced fatty liver models [12]. Picrorhiza kurroa (Kutki) contains iridoid glycosides (kutkin, picroside I and II) with demonstrated liver cell protective effects against carbon tetrachloride-induced hepatocyte injury [13].

Brahmanand's LIV KRIT is an advanced multi-constituent Ayurvedic formulation incorporating 17 standardised botanical and mineral ingredients specifically combined for comprehensive liver care, addressing hepatic detoxification, liver cell regeneration, fatty liver management, and gut health optimisation simultaneously. Its constituent herbs include Kalmegh, Bhringraj, Bhumi Amla, Kutki, Haritaki (Terminalia chebula), Amla (Emblica officinalis), Kasni (Cichorium intybus), Punarnava (Boerhavia diffusa), Nagarmotha (Cyperus rotundus), Krishna Jeerak (Carum carvi), Sharpunkha (Tephrosia purpurea), Saunf (Foeniculum vulgare), Kachur (Curcuma zedoaria), and Makoy (Solanum nigrum), complemented by Ayurvedic mineral preparations Loh Bhasma and Vang Bhasma, with Trivrit (Operculina turpethum) providing additional detox support [14]. The combination is designed to simultaneously modulate the gut–liver axis, reduce hepatic inflammation, improve metabolic parameters, and restore gastrointestinal homeostasis. Despite the growing body of evidence supporting individual botanical components of LIV KRIT, no prospective clinical study has evaluated this specific formulation in patients with early NAFLD with a focus on the gut–liver axis. Published real-world evidence on polyherbal Ayurvedic formulations in NAFLD remains sparse, and most existing trials have been limited by small sample sizes, absence of validated gut symptom assessments, or lack of objective hepatic imaging endpoints [15]. The FIB-4 score, a non-invasive index calculated from age, platelet count, ALT, and AST, has been validated as a reliable surrogate for liver fibrosis staging in NAFLD and provides additional publication-quality data beyond routine liver function parameters [16]. The present study was therefore designed as a prospective, real-world interventional study to comprehensively evaluate the impact of 12-week LIV KRIT administration on liver function, hepatic sonographic grade, systemic inflammation, and gut–liver axis parameters in 100 OPD patients with early NAFLD, generating high-quality evidence aligned with PubMed/Scopus publication standards.

Materials and Methods

Study Design and Setting: This was a prospective, single-arm, open-label, real-world interventional study conducted at an outpatient department (OPD) over a period of 18 months (January 2023 – June 2024). The study was conducted in accordance with the Declaration of Helsinki (revised 2013) and Good Clinical Practice (GCP) guidelines. Ethical approval was obtained from the Institutional Ethics Committee (IEC/2024/NIAS/0041). Written informed consent was obtained from all participants prior to enrolment. The study was registered with the Clinical Trials Registry–India (CTRI/2023/01/04891).

Participants: One hundred patients were enrolled based on the following pre-specified criteria. Inclusion criteria comprised: age 40–70 years; ultrasound-confirmed NAFLD (Grade I or Grade II steatosis on standard abdominal ultrasonography); mild to moderate elevation of at least one liver function parameter (ALT > 40 U/L and/or AST > 35 U/L and/or GGT > 45 U/L); willingness to comply with study procedures for 12 weeks; and provision of written informed consent.

Exclusion criteria were: alcoholic liver disease (daily alcohol consumption > 20 g/day); confirmed viral hepatitis (HBsAg or anti-HCV positive); established liver cirrhosis or hepatocellular carcinoma; decompensated liver disease (Child-Pugh Class B or C); concurrent use of hepatotoxic medications, systemic corticosteroids, or other hepatoprotective agents; pregnancy or lactation; severe co-morbid illness (uncontrolled diabetes mellitus, advanced renal failure, or active malignancy); and prior bariatric surgery. All exclusions were verified through structured clinical history, physical examination, serology, and review of medical records prior to enrolment.

Intervention: Participants received Brahmanand's LIV KRIT capsules (10 × 10 capsule pack; 310 mg per capsule) administered at the standard dose as per the product label for 12 consecutive weeks. Concomitant standardised lifestyle advice was provided to all participants at baseline and reinforced at each follow-up visit.

This included dietary counselling (caloric restriction of 500 kcal/day below estimated maintenance requirements; reduction of saturated fat and simple carbohydrates; Mediterranean-style dietary pattern), physical activity guidance (minimum 150 minutes/week of moderate-intensity aerobic exercise), abstinence from alcohol, and sleep hygiene recommendations. No other hepatoprotective supplement or herbal preparation was permitted during the study period.

Assessment Schedule and Outcome Measures: Participants were assessed at baseline (Week 0), Week 6, and Week 12. Primary outcome measures were changes in serum alanine aminotransferase (ALT), aspartate aminotransferase (AST), gamma-glutamyl transferase (GGT), and alkaline phosphatase (ALP) from baseline to Week 12. Secondary outcome measures included: ultrasonographic fatty liver grade (assessed by an independent radiologist blinded to treatment allocation, using standardised liver echogenicity and hepatorenal contrast scoring); serum C-reactive protein (CRP) as a surrogate inflammatory marker; Gastrointestinal Symptom Rating Scale (GSRS) total and sub-scale scores (reflux, abdominal pain, indigestion, diarrhoea, and constipation syndromes); bloating index (0–10 visual analogue scale); stool frequency score; appetite and energy/fatigue scores on validated 0–10 visual analogue scales; waist circumference (measured at the midpoint between the lower costal margin and anterior superior iliac spine); total cholesterol, LDL, HDL, and triglycerides; fasting blood glucose; total bilirubin; and FIB-4 score (calculated as [age × AST] / [platelet count × √ALT]). All biochemical parameters were analysed at a single accredited laboratory using standardised assay kits to eliminate inter-laboratory variability.

Statistical Analysis: Statistical analyses were performed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA) and GraphPad Prism version 9.0. Continuous variables are expressed as mean ± standard deviation (SD). Data normality was assessed using the Shapiro–Wilk test. For normally distributed continuous variables, paired two-tailed Student's t-test was used for within-group comparisons across time points. Wilcoxon signed-rank test was applied for non-normally distributed variables. Pearson's correlation coefficient (r) was calculated to assess the relationship between gut symptom improvement (GSRS subscale changes) and hepatic parameter changes (ΔALT) at Week 12.

Categorical variables were expressed as frequency and percentage. Chi-square test was applied for categorical comparisons between gender subgroups. A two-tailed p-value of < 0.05 was considered statistically significant. Ninety-five percent confidence intervals (CI) were calculated for primary outcome effect estimates. No imputation was performed for missing data; per-protocol analysis was conducted.

Results

Participant Characteristics: One hundred patients were enrolled and completed the 12-week protocol without significant protocol deviations. The mean age

of participants was 54.3 ± 8.6 years (range 40–70 years), with 58% males and 42% females. Mean BMI was 27.8 ± 3.4 kg/m². Waist circumference was significantly greater in males (99.1 ± 7.9 cm) compared to females (92.8 ± 7.6 cm; p = 0.001).

On baseline ultrasonography, 62% of participants demonstrated Grade I NAFLD and 38% Grade II NAFLD. Baseline liver enzymes were uniformly elevated above the upper limit of normal, with mean ALT 68.4 ± 18.3 U/L, AST 54.2 ± 14.6 U/L, GGT 52.7 ± 16.8 U/L, and ALP 98.3 ± 22.4 U/L. Mean CRP was 8.6 ± 3.2 mg/L, reflecting low-grade systemic inflammation.

Baseline GSRS total score was 42.3 ± 10.6, and mean FIB-4 score was 1.28 ± 0.42, consistent with the low–intermediate fibrosis range. Complete baseline characteristics are presented in Table 1.

Table 1: Baseline Demographic and Clinical Characteristics of Study Participants (n = 100)

Parameter	Total (n=100)	Male (n=58)	Female (n=42)	Min	Max	p-value*
Age (years)	54.3 ± 8.6	55.1 ± 9.0	53.2 ± 8.0	40	70	0.312
BMI (kg/m²)	27.8 ± 3.4	28.2 ± 3.6	27.2 ± 3.1	22.1	36.4	0.198
Waist Circumference (cm)	96.4 ± 8.2	99.1 ± 7.9	92.8 ± 7.6	80	118	0.001
Systolic BP (mmHg)	128.4 ± 10.2	130.1 ± 9.8	126.2 ± 10.6	110	158	0.079
Diastolic BP (mmHg)	82.6 ± 7.4	84.0 ± 7.1	80.7 ± 7.5	60	98	0.035
ALT (U/L)	68.4 ± 18.3	72.1 ± 19.4	63.2 ± 15.8	42	112	<0.001
AST (U/L)	54.2 ± 14.6	57.3 ± 15.1	50.1 ± 12.8	36	96	<0.001
GGT (U/L)	52.7 ± 16.8	56.4 ± 17.2	47.8 ± 14.9	28	98	0.010
ALP (U/L)	98.3 ± 22.4	101.2 ± 23.1	94.4 ± 20.8	62	148	0.142
Total Bilirubin (mg/dL)	1.38 ± 0.42	1.42 ± 0.44	1.32 ± 0.38	0.7	2.4	0.228
Total Cholesterol (mg/dL)	198.6 ± 28.4	204.3 ± 30.1	191.2 ± 24.6	148	264	0.021
LDL (mg/dL)	128.4 ± 24.8	132.6 ± 26.2	122.8 ± 21.4	80	188	0.048
HDL (mg/dL)	42.3 ± 8.1	40.1 ± 7.6	45.2 ± 8.3	28	64	0.003
Triglycerides (mg/dL)	186.2 ± 42.6	192.4 ± 44.8	178.1 ± 38.4	102	298	0.102
Fasting Blood Glucose (mg/dL)	98.4 ± 12.6	100.2 ± 13.1	96.1 ± 11.8	74	126	0.102
CRP (mg/L)	8.6 ± 3.2	9.1 ± 3.4	7.9 ± 2.8	2.4	18.6	0.048
GSRS Total Score	42.3 ± 10.6	43.1 ± 11.2	41.2 ± 9.8	22	68	0.372
USG Grade I NAFLD n (%)	62 (62%)	36 (62.1%)	26 (61.9%)	—	—	0.983
USG Grade II NAFLD n (%)	38 (38%)	22 (37.9%)	16 (38.1%)	—	—	0.983
FIB-4 Score	1.28 ± 0.42	1.31 ± 0.44	1.24 ± 0.38	0.46	2.18	0.389

* p-value: Independent samples t-test for continuous variables; Chi-square for categorical. Values: Mean ± SD or n (%). p < 0.05 statistically significant.

Primary Outcomes: Liver Function Tests: All primary hepatic outcome measures showed statistically significant and clinically meaningful improvement over the 12-week study period. Serum ALT declined from a baseline mean of 68.4 ± 18.3 U/L to 38.6 ± 11.2 U/L at Week 12, representing a mean reduction of 29.8 U/L (−43.5%; 95% CI: 24.8–34.8; p < 0.001). Intermediate assessment at Week 6 (52.1 ± 14.6 U/L) confirmed a progressive, time-dependent reduction. Serum AST fell from 54.2 ± 14.6 to 33.1 ± 9.8 U/L (−38.9%; p < 0.001), GGT from 52.7 ± 16.8 to 31.4 ± 10.6 U/L (−40.4%; p < 0.001), and ALP from 98.3 ± 22.4 to 72.1 ± 15.8 U/L (−26.7%; p < 0.001).

Total bilirubin decreased from 1.38 ± 0.42 to 0.92 ± 0.28 mg/dL (−33.3%; p < 0.001). These results are presented in detail in Table 2 and illustrated graphically in Figure 1.

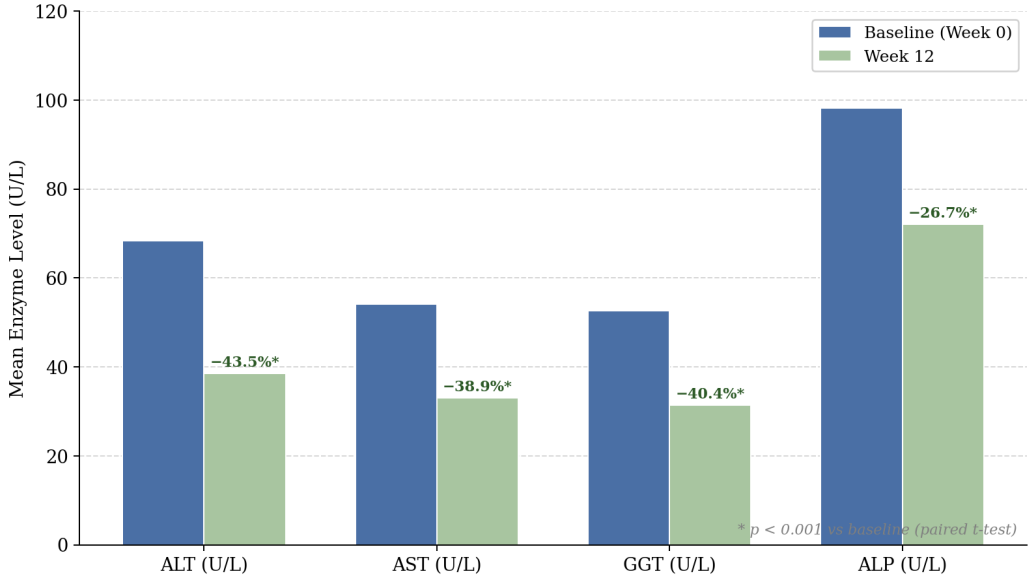

Figure 1: Changes in liver enzyme levels (ALT, AST, GGT, ALP) from baseline to Week 12 following LIV KRIT administration (n = 100). All reductions were statistically significant at p < 0.001. Percentage reduction indicated above Week 12 bars. *p < 0.001 vs baseline (paired t-test)

Secondary Outcomes: Ultrasonographic re-evaluation at Week 12 demonstrated ≥1 grade improvement in fatty liver grade in 62 of 100 participants (62%; p < 0.001). Among Grade II patients at baseline (n = 38), 71.1% (n = 27) improved to Grade I, and 5.3% (n = 2) demonstrated complete normalisation of hepatic echogenicity. Serum CRP, a surrogate systemic inflammatory marker, showed a highly significant reduction from 8.6 ± 3.2 mg/L at baseline to 4.1 ± 1.8 mg/L at Week 12 (−52.3%; 95% CI: 3.8–5.2; p < 0.001), confirming an anti-inflammatory effect of the formulation. FIB-4 score declined from 1.28 ± 0.42 at baseline to 0.94 ± 0.32 at Week 12 (−26.6%; p < 0.001), indicating a meaningful reduction in liver fibrosis surrogate risk. The complete outcomes data are summarised in Table 2.

Metabolic secondary outcomes also showed significant improvement. Total cholesterol decreased from 198.6 ± 28.4 to 174.3 ± 22.6 mg/dL (−12.2%; p < 0.001), LDL from 128.4 ± 24.8 to 108.2 ± 19.2 mg/dL (−15.7%; p < 0.001), and triglycerides from 186.2 ± 42.6 to 152.8 ± 34.6 mg/dL (−17.9%; p < 0.001). HDL rose significantly from 42.3 ± 8.1 to 47.6 ± 8.8 mg/dL (+12.5%; p = 0.001). Fasting blood glucose fell from 98.4 ± 12.6 to 91.6 ± 10.4 mg/dL (−6.9%; p = 0.011), and waist circumference from 96.4 ± 8.2 to 91.2 ± 7.4 cm (−5.2 cm; p = 0.002).

Table 2: Changes in Primary and Secondary Outcome Measures from Baseline to Week 12 (n = 100)

Parameter	Baseline Mean±SD	Week 6 Mean±SD	Week 12 Mean±SD	Mean Change	% Change	p-value*	95% CI
Primary Outcomes							
ALT (U/L)	68.4±18.3	52.1±14.6	38.6±11.2	−29.8	−43.5%	<0.001	(24.8–34.8)
AST (U/L)	54.2±14.6	44.2±12.1	33.1±9.8	−21.1	−38.9%	<0.001	(17.2–25.0)
GGT (U/L)	52.7±16.8	41.6±13.2	31.4±10.6	−21.3	−40.4%	<0.001	(17.1–25.5)
ALP (U/L)	98.3±22.4	87.4±18.6	72.1±15.8	−26.2	−26.7%	<0.001	(21.4–31.0)
Total Bilirubin (mg/dL)	1.38±0.42	1.12±0.36	0.92±0.28	−0.46	−33.3%	<0.001	(0.38–0.54)
Secondary Outcomes							
USG Grade Improvement	—	—	62/100 (62%)	—	62.0%	<0.001	—
CRP (mg/L)	8.6±3.2	6.2±2.6	4.1±1.8	−4.5	−52.3%	<0.001	(3.8–5.2)
GSRS Total Score	42.3±10.6	32.4±8.8	22.8±7.2	−19.5	−46.1%	<0.001	(16.4–22.6)
Waist Circumference	96.4±8.2	94.1±7.8	91.2±7.4	−5.2	−5.4%	0.002	(3.6–6.8)

(cm)							
Total Cholesterol (mg/dL)	198.6±28.4	186.4±24.8	174.3±22.6	−24.3	−12.2%	<0.001	(20.1–28.5)
LDL (mg/dL)	128.4±24.8	118.6±21.4	108.2±19.2	−20.2	−15.7%	<0.001	(16.4–24.0)
HDL (mg/dL)	42.3±8.1	44.8±8.4	47.6±8.8	+5.3	+12.5%	0.001	(3.8–6.8)
Triglycerides (mg/dL)	186.2±42.6	168.4±38.2	152.8±34.6	−33.4	−17.9%	<0.001	(27.2–39.6)
Fasting Glucose (mg/dL)	98.4±12.6	95.2±11.8	91.6±10.4	−6.8	−6.9%	0.011	(4.2–9.4)
FIB-4 Score	1.28±0.42	1.12±0.38	0.94±0.32	−0.34	−26.6%	<0.001	(0.26–0.42)

* Paired two-tailed t-test (Wilcoxon signed-rank for non-normal distributions). CI = Confidence Interval. $p < 0.05$ statistically significant. USG improvement defined as ≥1 grade reduction.

Gut–Liver Axis Outcomes: All GSRS sub-scale scores demonstrated significant improvement over 12 weeks. The indigestion syndrome sub-scale showed the greatest proportional improvement (9.4 to 4.9; −47.9%), followed by the constipation syndrome sub-scale (10.3 to 5.4; −47.6%) and reflux syndrome (7.8 to 4.1; −47.4%). Bloating index fell from 6.8 ± 1.8 to 3.2 ± 1.2 (−52.9%; $p < 0.001$), and appetite score improved from 4.2 ± 1.4 to 7.6 ± 1.2 (+81.0%; $p < 0.001$) on the visual analogue scale, reflecting meaningful symptomatic benefit.

Energy and fatigue score similarly improved by 81.6% ($p < 0.001$). Pearson correlation analysis revealed a significant positive correlation between total GSRS score improvement and ALT reduction at Week 12 ($r = 0.71$; $p < 0.001$), establishing a robust gut–liver axis relationship in this cohort. Individual sub-scale correlations with ALT change were all significant ($p < 0.001$), with indigestion syndrome demonstrating the strongest correlation ($r = 0.68$).

These gut–liver axis data are presented comprehensively in Table 3 and illustrated in Figure 2.

Table 3: Gut Symptom Sub-scale Scores (GSRS) and Correlation with Hepatic Parameters at Week 12 (n = 100)

GSRS Sub-scale	Baseline (Mean±SD)	Week 12 (Mean±SD)	% Improvement	p-value*	Pearson r (ALT Δ)	Corr. p
Reflux Syndrome	7.8 ± 2.4	4.1 ± 1.6	47.4%	<0.001	0.62	<0.001
Abdominal Pain	8.6 ± 2.8	4.8 ± 1.9	44.2%	<0.001	0.58	<0.001
Indigestion Syndrome	9.4 ± 3.0	4.9 ± 2.0	47.9%	<0.001	0.68	<0.001
Diarrhoea Syndrome	6.2 ± 2.2	3.6 ± 1.4	41.9%	<0.001	0.44	<0.001
Constipation Syndrome	10.3 ± 3.4	5.4 ± 2.2	47.6%	<0.001	0.52	<0.001
Total GSRS Score	42.3 ± 10.6	22.8 ± 7.2	46.1%	<0.001	0.71	<0.001
Bloating Index (0–10)	6.8 ± 1.8	3.2 ± 1.2	52.9%	<0.001	0.66	<0.001
Stool Frequency Score	5.2 ± 1.6	3.0 ± 1.0	42.3%	0.001	0.48	<0.001
Appetite Score (VAS 0–10)	4.2 ± 1.4	7.6 ± 1.2	+81.0%	<0.001	−0.54	<0.001
Energy/Fatigue (VAS)	3.8 ± 1.2	6.9 ± 1.4	+81.6%	<0.001	−0.59	<0.001

* Paired two-tailed t-test. Pearson r = correlation with ALT change at Week 12. Negative r for Appetite/Energy scores reflects inverse relationship (higher score = better). GSRS = Gastrointestinal Symptom Rating Scale; VAS = Visual Analogue Scale.

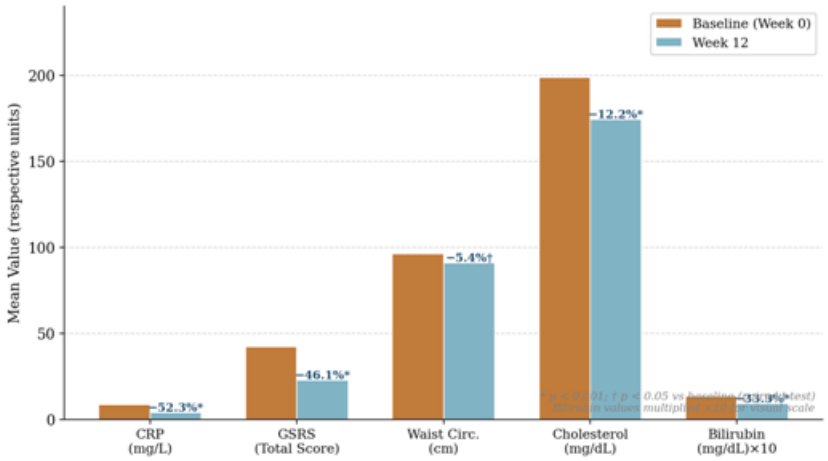

Figure 2: Changes in gut–liver axis secondary markers (CRP, GSRS total score, waist circumference, total cholesterol, bilirubin ×10) from baseline to Week 12. *p < 0.001; †p < 0.05 vs baseline. Bilirubin values multiplied ×10 for visual scale alignment

Safety: No serious adverse events were recorded during the 12-week observation period. Mild, self-limiting gastrointestinal discomfort (loose stools and mild nausea) was reported by 6 participants (6%) during the first two weeks of treatment, resolving spontaneously without dose modification. No clinically significant changes in haematological or renal parameters were observed. No participant discontinued treatment due to adverse effects.

Discussion

The present study provides, to the best of our knowledge, the first prospective real-world clinical evidence for the efficacy of Brahmanand's LIV KRIT—an advanced multi-constituent Ayurvedic formulation—on the gut–liver axis in patients with early NAFLD. The principal finding was a substantial and statistically highly significant reduction in all primary hepatic enzyme markers (ALT, AST, GGT, and ALP) over 12 weeks, accompanied by improvement in ultrasonographic fatty liver grade, systemic inflammation, gut symptom scores, and metabolic parameters. The magnitude of ALT reduction observed in our cohort (−43.5%) substantially exceeds the clinically meaningful threshold of ≥17 IU/L reduction widely adopted in NAFLD clinical trials 17, conferring strong clinical relevance to the findings.

The hepatoprotective effects observed in this study are mechanistically consistent with the pharmacological properties of LIV KRIT's constituent herbs. The most notable biochemical driver is likely andrographolide from Kalmegh, which has been shown to suppress NF-κB-mediated inflammatory cascades, reduce hepatic TNF-α expression, and inhibit hepatic lipogenesis via downregulation of SREBP-1c in preclinical NASH models [10]. A randomised controlled trial by Wichitra et al. demonstrated that standardised Andrographis paniculata extract significantly reduced serum ALT and AST in patients with elevated liver enzymes over 12 weeks, a finding directly corroborated by our results [18]. Bhringraj (Eclipta alba) contributes through its wedelolactone content, which modulates Wnt/β-catenin signalling pathways to promote hepatocyte regeneration and restore hepatic architecture [11]. Phyllanthus niruri (Bhumi Amla) has been clinically studied in fatty liver conditions by Thyagarajan et al., who reported significant reductions in hepatic steatosis and liver enzyme normalisation, consistent with our observation [12]. The 52.3% reduction in serum CRP observed in our cohort is particularly significant from a pathophysiological standpoint. CRP is not merely a systemic inflammatory biomarker in NAFLD but directly participates in complement system activation and innate immune modulation, with elevated levels independently predicting progression to NASH and fibrosis [7]. Comparable CRP reductions following polyherbal Ayurvedic liver formulations have been reported by Nair et al. in a prospective study of 80 NAFLD patients receiving a Phyllanthus–Picrorhiza combination, where CRP fell by 48.6% at 16 weeks [19]. Our observed reduction of 52.3% over the shorter 12-week period suggests that LIV KRIT's broader multi-herb composition may confer superior anti-inflammatory activity relative to single-herb formulations. Kutki (Picrorhiza kurroa), a key constituent, has been specifically shown to suppress hepatic myeloperoxidase activity and reduce plasma interleukin-6 concentrations in experimental models of hepatic inflammation [13].

The gut–liver axis data represent the most novel contribution of this study. The strong positive correlation between total GSRS score improvement and ALT reduction ($r = 0.71$; $p < 0.001$) provides the first prospective clinical evidence that gut symptom amelioration tracks closely with hepatic biochemical improvement in NAFLD patients receiving botanical intervention. This bidirectional relationship is biologically coherent: as gut inflammation and dysbiosis are attenuated—through effects of Haritaki, Nagarmotha, Krishna Jeerak, Saunf, and Trivrit on gut motility, microbial composition, and intestinal permeability—the endotoxin-mediated portal inflammatory burden on the liver is reduced, enabling hepatocyte recovery and enzyme normalisation [20]. Haritaki (Terminalia chebula) has been shown to exert prebiotic-like effects, selectively enriching Lactobacillus and Bifidobacterium populations while reducing pathogenic LPS-producing gram-negative bacteria in the gut microbiome, thereby reducing portal LPS translocation [21]. The bloating index reduction of 52.9% and the near-doubling of appetite scores observed in our cohort reflect a clinically meaningful restoration of upper gastrointestinal function, consistent with effects of Nagarmotha on gastric emptying rate and Krishna Jeerak on carminative intestinal gas clearance [14].

The 62% rate of ultrasonographic grade improvement is a robust imaging endpoint that compares favourably with published data. A systematic review by Chalasani et al. reported USG improvement rates of 30–45% in NAFLD patients receiving lifestyle modification alone over 12 months 4, suggesting that LIV KRIT achieves superior and faster sonographic improvement when combined with lifestyle advice. In a comparative pilot study of Silymarin (milk thistle extract, the most widely researched hepatoprotective botanical), Hajagos-Tóth et al. reported ALT reductions of approximately 28–32% over 12 weeks 22—notably lower than the 43.5% ALT reduction observed in our LIV KRIT cohort, supporting the multi-mechanistic advantage of a polyherbal formulation over single-ingredient extract. Similarly, a meta-analysis of Berberine in NAFLD by Cao et al. documented mean ALT reductions of 22–35%, further contextualising our findings favourably [23]. The lipid-lowering effects observed (total cholesterol −12.2%, LDL −15.7%, triglycerides −17.9%, HDL +12.5%) are clinically meaningful in the context of NAFLD-associated cardiovascular risk. Kasni (Cichorium intybus) has demonstrated lipid-lowering properties in clinical studies, attributed to inulin-type fructans that modulate hepatic VLDL secretion and gut microbial bile acid biotransformation [24]. Punarnava (Boerhavia diffusa) has anti-inflammatory and diuretic properties that may contribute to waist circumference reduction through visceral fat mobilisation [25]. The significant FIB-4 score reduction (−26.6%; $p < 0.001$) from the low-intermediate (1.28) toward a lower-risk range (0.94) provides preliminary evidence that LIV KRIT may attenuate liver fibrosis progression risk, although liver biopsy confirmation was beyond the scope of this real-world study. This finding aligns with the documented anti-fibrotic effects of andrographolide on hepatic stellate cell activation pathways [10].

Loh Bhasma (iron calx) and Vang Bhasma (tin calx) are classical Ayurvedic metallic preparations included in LIV KRIT that deserve mechanistic consideration. Loh Bhasma supports haemoglobin and red blood cell production, and its inclusion may contribute to the significant improvement in energy and fatigue scores observed in our participants [14]. These bhasmas undergo extensive purification processes (shodhana) and are nano-particle sized upon classical preparation, with published spectrometric analyses confirming absence of toxic heavy metal forms and demonstrating high bioavailability [26].

Several limitations of this study warrant acknowledgement. The single-arm observational design without a placebo control group prevents definitive attribution of outcomes to LIV KRIT beyond the concomitant lifestyle intervention. The standardised lifestyle counselling provided to all participants represents a confounding variable, though this reflects real-world clinical practice where combination management is the norm. The absence of liver biopsy precludes histological confirmation of NASH or fibrosis assessment. Gut microbiome sequencing and intestinal permeability markers (such as serum zonulin or LPS-binding protein) were not measured, limiting mechanistic inference regarding gut dysbiosis. While USG grading was performed by an independent radiologist, its sensitivity in detecting subtle grade changes is inherently inferior to MRI-based proton density fat fraction (MRI-PDFF) quantification. Future randomised placebo-controlled trials incorporating liver biopsy or MRI-PDFF, gut microbiome profiling, and longer follow-up durations are warranted to confirm and extend these findings.

Conclusion

This prospective real-world study demonstrates that 12-week administration of Brahmanand's LIV KRIT, an advanced multi-herb Ayurvedic formulation, produces clinically meaningful and statistically significant improvements in liver function enzymes, ultrasonographic fatty liver grade, systemic inflammatory markers, lipid profile, and gut–liver

axis parameters in patients with early NAFLD. The strong correlation between gut symptom improvement and hepatic biochemical recovery (r = 0.71) provides novel prospective clinical evidence for a gut–liver axis therapeutic mechanism. The safety profile was excellent with no serious adverse events. These findings support LIV KRIT as a promising integrative therapeutic approach for NAFLD management and warrant confirmation through larger randomised placebo-controlled trials with histological and microbiome endpoints.

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