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A Randomised, Controlled, Comparative Therapeutic Trial Of Panchakola Churna And Lifestyle - Dietary Modification In Patients With Non-Alcoholic Fatty Liver Disease (NAFLD)

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Abstract

Objective:
Non-alcoholic fatty liver disease (NAFLD) is one of the most common chronic liver diseases worldwide, with a rising prevalence and limited effective pharmacological treatment options. Ayurveda describes a condition comparable to NAFLD as Medosamchaya in Yakrit (Accumulation of fat in the liver), where derangement of Meda metabolism leads to fat accumulation in the liver. This study aimed to evaluate the Comparative efficacy of Panchakola Churna along with lifestyle and dietary modifications in the management of Medosamchaya in Yakrit (NAFLD) compared with lifestyle and dietary modifications alone in patients with Grade 1 and Grade 2 non-alcoholic fatty liver disease (NAFLD).
Methods:
A prospective, randomized, comparative clinical study was conducted on 54 patients with Grade 1 and Grade 2 NAFLD. Patients were randomly allocated into two groups (n = 27 each). Group A received lifestyle and dietary modification, while Group B received Panchakola Churna (3 g twice daily with warm water) along with lifestyle and dietary modification for 60 days. Biochemical parameters like LFT and Lipid profile, Ultrasonographic grading were assessed before and after treatment.
Results:
Both groups showed improvement in clinical and biochemical parameters; however, Group B demonstrated greater improvement in liver function,Lipid profiles, and ultrasonographic grading of fatty liver compared to Group A. The treatment was well tolerated, and no significant adverse effects were observed.
Conclusion:
Panchakola Churna, when administered along with lifestyle and dietary modification, was more effective than lifestyle and dietary modification alone in the management of Grade 1 and Grade 2 NAFLD.

KEYWORDS: Non-alcoholic fatty liver,Santarpanaja vikaras, Medosamchaya in Yakrit.

1.Introduction

Non-alcoholic fatty liver disease (NAFLD) is defined as the accumulation of fat in more than 5% of hepatocytes in the absence of significant alcohol consumption and other secondary causes of hepatic steatosis. It is one of the most common liver disorders worldwide and has become a major public health concern due to its increasing prevalence and potential progression to fibrosis, cirrhosis, and hepatocellular carcinoma. It is closely associated with obesity, insulin resistance, metabolic syndrome, sedentary lifestyle, and unhealthy dietary habits [1].

The pathophysiology of non-alcoholic fatty liver disease (NAFLD) is complex and multifactorial, characterized by excessive accumulation of triglycerides within hepatocytes due to an imbalance between lipid acquisition and disposal. Hepatic steatosis results primarily from increased free fatty acid influx and de novo lipogenesis, along with impaired very-low-density lipoprotein (VLDL) synthesis and secretion, leading to reduced lipid export from the liver. Progression from simple steatosis to non-alcoholic steatohepatitis (NASH) is driven by oxidative stress, mitochondrial dysfunction, inflammatory cytokines, insulin resistance, and alterations in gut microbiota that increase intestinal permeability and endotoxemia, thereby promoting hepatic inflammation and fibrosis [2]. Genetic susceptibility, particularly variants of PNPLA3 and TM6SF2, further influences hepatic fat accumulation and disease progression. In addition, activation of inflammatory pathways, including c-Jun N-terminal kinase (JNK), NOD-like receptors (NLRs), and dysregulation of nuclear receptors such as the farnesoid X receptor (FXR), contributes to chronic inflammation, fibrosis, and the progression of NAFLD to advanced liver disease [3].

According to Ayurveda, NAFLD can be understood as Medosamchaya in Yakrit. It develops due to continuous indulgence in Madhura, Snigdha and Guru Ahara, along with Divaswapna (day sleep) and Avyayama (lack of physical activity), leading to Kapha Dosha aggravation [4]. In this condition, Jatharagni is often hyperfunctional, resulting in rapid digestion of food, while Madhura-Snigdha Ahara produces Madhuratara and Atisnigdha Ahara Rasa, which preferentially nourishes Meda Dhatu and promotes its Vriddhi. Medo Dhatvagni remains normal or hyperactive, whereas reduced activity of Pachaka Ansha on Sthula Meda Dhatu further enhances Meda accumulation [5]. Excessive Meda causes Srotorodha, producing features such as Kshudra Shwasa, Kshudha, and Trishna. Due to obstruction of Medovaha Srotas, Vata Dosha becomes restricted to the Koshtha, stimulating appetite and promoting further Meda accumulation. The formed Medodhatu circulates through Rakta and is deposited in Yakrit. Simultaneously, vitiated Pitta causes Rakta Dushti in the Raktavaha Srotas (Yakrit). In due course, Khavaigunya develops in the liver, leading to Yakrit Shotha and ultimately Medosanchaya in Yakrit (fatty liver). As described under Yakritodara, Nidana Sevana simultaneously aggravates Rakta and Kapha, contributing to the manifestation of liver disease. Thus, the combined effects of dietary, metabolic, and genetic factors lead to the development and progression of Medosanchaya in Yakrit, which closely resembles the pathogenesis of Non-Alcoholic Fatty Liver Disease (NAFLD).

Although liver biopsy remains the gold standard for diagnosing non-alcoholic fatty liver disease (NAFLD), its routine use is limited because of its invasive nature, high cost, risk of complications, and poor patient acceptance. Advanced imaging modalities such as computed tomography (CT), magnetic resonance imaging (MRI), magnetic resonance spectroscopy (MRS), MRI-proton density fat fraction (MRI-PDFF), and magnetic resonance elastography (MRE) provide accurate assessment of hepatic steatosis and fibrosis but are expensive, time-consuming, and not readily available in routine clinical practice. Ultrasonography (USG) is a non-invasive, radiation-free, inexpensive, widely available, and reproducible imaging modality that provides reliable qualitative grading of hepatic steatosis, making it the preferred imaging tool for the present study [6]. In addition, liver function tests (LFT), including serum ALT and AST levels, are useful biochemical markers for assessing hepatocellular injury and monitoring therapeutic response, while the lipid profile (total cholesterol, triglycerides, LDL, and HDL) reflects abnormalities in lipid metabolism associated with NAFLD and helps evaluate metabolic improvement following treatment. Therefore, USG, LFT, and lipid profile were selected as the primary outcome measures owing to their clinical relevance, accessibility, cost-effectiveness, and suitability for serial assessment during follow-up [7].

Management of NAFLD primarily focuses on lifestyle modification; however, the long-term effectiveness of these measures alone is often limited. Ayurveda advocates Nidana Parivarjana, dietary regulation, lifestyle modification, and appropriate Aushadha for correcting metabolic disturbances. Panchakola Churna, possessing Deepana, Pachana, Kapha-Medohara, and Agni-stimulating properties, is traditionally indicated for disorders associated with impaired metabolism and excessive fat accumulation [8]. Therefore, the present study was undertaken to evaluate the efficacy of Panchakola Churna along with lifestyle and dietary modifications in the management of Medosamchaya in Yakrit (NAFLD) compared with lifestyle and dietary modifications alone.

2. Materials and Methods

The interventional component of this research was conducted as a double-arm randomized clinical trial. The study setting comprised patients who attended the Outpatient Department (O.P.D.) and Inpatient Department (I.P.D.) of Vikriti Vigyan, as well as the O.P.D. and I.P.D. of Kayachikitsa at Sir Sunderlal Hospital (SSH), Institute of Medical Sciences (I.M.S.), Banaras Hindu University (BHU). A total sample size of 54 patients was enrolled and divided equally into two groups containing 27 subjects each.
Group A (Control Group): Subjects (n = 27) were advised to undergo lifestyle modification along with dietary changes for a continuous period of 60 days (approximately 2 months).
Group B (Interventional Group): Subjects (n = 27) were administered Panchakola churna alongside the same lifestyle and dietary modifications continuously for a period of 60 days.
Ethical approval was acquired from the Institutional Ethics Committee (Clearance Number: IEC NO: 7437), and the trial was registered with the Clinical Trial Registry of India under registration number CTRI/2025/01/079000. Strictly maintaining confidentiality, all participating patients provided written informed consent prior to data recording, drug administration, or laboratory procedures. For the interventional phase, baseline comparability between Group A and Group B was evaluated using an unpaired t-test, while within-group comparisons of continuous variables over time were analysed using a paired t-test. Statistical significance was set at a threshold of $p < 0.05$, and all computations were carried out using the open-source software Jamovi (version 2.6.26).

2.1 Selection of study participants

INCLUSION CRITERIA
1) Subject with NAFLD Grade 1 and Grade2 (mild and moderate fatty infiltration as per USG
2) Age group of 30-70 years both male and females.
3) Subject willing to give informed consent and participate in the study.

EXCLUSION CRITERIA
1) Diagnosed cases of Renal, Respiratory, Cardiovascular and Neurological disorder.
2)Subject with Hepatocellular Carcinoma and Cirrhosis.
3) USG finding suggestive of fatty liver grade 3 and 4
4) Pregnant ladies and Lactating mothers.
5) Those who are consuming Alcohol.

2.2 Study procedures

The study procedure for the interventional phase involved a total sample size of 54 patients diagnosed with Medosamchaya in yakrit (NAFLD), who were registered from the O.P.D. of BHU on the basis of designated inclusion and exclusion criteria. These participants were systematically divided into two distinct parallel cohorts of 27 subjects each for a continuous treatment duration of 60 days (approximately 2 months). Group A (Control Group) was advised to strictly follow a regimen of lifestyle modification along with dietary changes. Group B (Interventional Group) was administered Panchakola churna—formulated at the Ayurvedic Pharmacy, FOA, IMS by washing, shade-drying, and grinding raw Pippali, Pippalimula, Chavya, Chitraka, and Nagara through an 80-mesh filter [9] —at a dose of 3 grams twice daily with lukewarm water as an Anupana, alongside identical lifestyle and dietary modifications. The mandated lifestyle and dietary rules for both groups included limiting daily excess oil intake to 10–15 ml, restricting daily ghee consumption to 5–10 ml, completely avoiding white sugar, and engaging in either a 30-minute brisk walk or a 1-hour normal walk daily with diet modification given as per dietary chart [10]. Clinical progress was tracked through 4 total follow-ups scheduled at precise intervals of 15 days throughout the therapeutic duration.
Monitoring of these lifestyle and dietary modifications was carried out through a structured and continuous self-reporting approach. All patients were instructed to maintain a daily diary documenting their dietary intake, including details of food items consumed, portion sizes, meal timings, and adherence to the prescribed diet chart. The diary also included records of lifestyle practices such as physical activity, sleep patterns, and avoidance of risk factors like sedentary habits and excess sugar intake. Compliance was assessed by comparing the recorded entries with the recommended dietary guidelines. In addition, a weekly recall method was also employed, wherein patients were interviewed to recollect and verify their dietary and lifestyle practices over the past seven days. This dual method

of daily documentation and periodic recall was used to ensure accurate monitoring, improve patient adherence, and provide reliable data for assessing the impact of lifestyle and dietary modifications on the progression and management of NAFLD.

2.3 Study assessment variable

To provide a comprehensive assessment of the therapeutic outcomes, the study evaluated specific biomarkers and structural indicators within each of the three objective parameters .For the Liver Function Test (LFT), key metabolic enzymes and hepatic markers were measured, including Serum Glutamic Oxaloacetic Transaminase (SGOT/AST), Serum Glutamic Pyruvic Transaminase (SGPT/ALT), Alkaline Phosphatase (ALP), Serum Bilirubin (Total, Direct, and Indirect), Total Protein, and Albumin levels to assess hepatocellular damage and liver synthetic function. The Lipid Profile parameter quantified crucial cardiovascular and metabolic circulating fractions, specifically evaluating Total Cholesterol, Triglycerides (TG), High-Density Lipoprotein (HDL), Low-Density Lipoprotein (LDL), and Very-Low-Density Lipoprotein (VLDL) cholesterol to track alterations in systemic lipid metabolism. Lastly, Ultrasonography (USG) of the whole abdomen served as the definitive imaging component, utilized to structurally grade the severity of hepatic steatosis (fatty liver change) by monitoring changes in liver size, parenchymal echogenicity, and acoustic attenuation.

2.4 Statistical consideration

2.4.1. Sample size

Total 54 patients are selected for the study according to the selection criterias and divided into 2 groups-

1.Group A Patients (CONTROL GROUP) -In this group 27 subjects were advised to do lifestyle modification with dietary changes for a period of 60 days (about 2 months).

2.Group B Patients -In this Group 27 subjects were advised to do Panchakola churnam along with lifestyle and dietary modifications was administered for a period of 60 days (about 2 months) continuously.

2.5. Statistical analysis

In this interventional study, the baseline comparability of the two randomized groups (Group A and Group B) was confirmed using an unpaired t-test. For within group comparison of continuous variables was analysed by paired t test. A p value less than 05 was considered as statistically significant. Data analysis was performed using opensource statistical software Jamovi version 2.6.26.

3. Results

3.1. Demographics of the study participants

3.1.1. Distribution of Study group patients of NAFLD according to Age (in years):

Table No 1: Showing Distribution of Study group patients of NAFLD according to Age

Age (in years)	Group A		Group B	
	Frequency	Percentage	Frequency	Percentage
30-40	3	11.1	7	25.9
41-50	14	51.9	8	29.6
51-60	10	37.0	12	44.4
Total	**27**	**100**	**27**	**100**

In Group A, the highest proportion of patients belonged to the 41–50 years age group, accounting for 51.9% (14 patients). This was followed by the 51–60 years age group with 37% (10 patients), while the 30–40 years age group had the smallest proportion, comprising 11.1% (3 patients) of the total.

In Group B, the majority of patients were in the 51–60 years age group, contributing 44.4% (12 patients), followed by the 41–50 years age group at 29.6% (8 patients). The 30–40 years age group accounted for 25.9% (7 patients), making it the least represented group in Group B.

3.1.2. Distribution of Study group patients of NAFLD according to Gender:

Table No 2: Showing Distribution of Study group patients of NAFLD according to Gender

Gender	Group A		Group B	
	Frequency	Percentage	Frequency	Percentage
Male	19	70.4	22	81.5
Female	8	29.6	5	18.5
Total	**27**	**100**	**27**	**100**

Interpretation: The above table presents the gender distribution of patients in both groups.
In Group A, the majority of patients were male, accounting for 70.4% (19 patients), while females represented 29.6% (8 patients) of the group.
In Group B, males also predominated, comprising 81.5% (22 patients), with females making up only 18.5% (5 patients) of the total.

3.1.3. Distribution of Study group patients of NAFLD according to Deha Prakriti:

Table No.3: Showing Distribution of Study group patients of NAFLD according to Deha Prakriti

Deha Prakriti	Group A		Group B	
	Frequency	Percentage	Frequency	Percentage
KP	9	33.3	13	48.1
PK	1	3.7	1	3.7
PV	1	3.7	1	3.7
VK	16	59.3	12	44.4
Total	**27**	**100**	**27**	**100**

Interpretation: The above table shows the distribution of Prakriti among patients in Groups A and B.
In Group A, the most common Prakriti was Vata–Kapha (VK), observed in 59.3% (16 patients), followed by Kapha–Pitta (KP) in 33.3% (9 patients). The least common Prakriti were Pitta–Kapha (PK) and Pitta–Vata (PV), each seen in 3.7% (1 patient).
In Group B, Kapha–Pitta (KP) was the most prevalent Prakriti at 48.1% (13 patients), followed by Vata–Kapha (VK) in 44.4% (12 patients). PK and PV were again the least common, each accounting for 3.7% (1 patient).

3.2. Effect on efficacy variables
3.2.1. LIVER FUNCTION TEST
3.2.1.1. SGPT: Result of Unpaired t test is as follows:

Table No.4: Inter group comparison of SGPT levels Before and after treatment

SGPT	Mean	SD	Test statistic	P value
Group A	7.33	11.726	-3.062	0.003 (Significant)
Group B	28	33.051		

3.2.1.2. SGOT: Result of Unpaired t test is as follows:

Table No.5: Inter group comparison of SGOT levels Before and after treatment

SGOT	Mean	SD	Test statistic	P value
Group A	4.05	8.065	-2.167	0.035 (Significant)
Group B	9.16	9.197		

3.2.1.3. TOTAL BILIRUBIN

Table No.6: Inter group comparison Total bilirubin levels Before and after treatment

Total Bilirubin	Mean		% of improvement	SD		t	P value
	BT	AT		BT	AT		
Group A	0.78	0.76	3.3	0.28	0.29	2.495	0.019
Group B	0.82	0.74	9.2	0.34	0.29	2.929	0.007

3.2.1.4. INDIRECT BILIRUBIN: Result of Unpaired t test is as follows:

Table No.7: Inter group comparison of Indirect Bilirubin levels Before and after treatment

INDIRECT BILIRUBIN	Mean	SD	Test statistic	P value
Group A	0.021	0.050	-1.118	0.269 (Not Significant)
Group B	0.036	0.049		

3.2.1.5. DIRECT BILIRUBIN: Result of Unpaired t test is as follows:

Table No.8: Inter group comparison of Direct Bilirubin levels Before and after treatment

DIRECT BILIRUBIN	Mean	SD	Test statistic	P value
Group A	0.010	0.021	-2.91	0.005 (Significant)
Group B	0.020	0.025		

3.2.1.6. TOTAL SERUM PROTEIN: Result of Unpaired t test is as follows:

Table No.9: Inter group comparison of Total Serum Protein level Before and after treatment

TOTAL SERUM PROTEIN	Mean	SD	Test statistic	P value
Group A	0.01	0.038	-0.337	0.738 (Not Significant)
Group B	0.01	0.042		

3.2.1.7. SERUM ALBUMIN: Result of Unpaired t test is as follows:

Table No.10: Inter group comparison of Serum Albumin levels Before and after treatment

SERUM ALBUMIN	Mean	SD	Test statistic	P value
Group A	0.009	0.048	0.350	0.727 (Not Significant)
Group B	0.01	0.070		

3.2.1.8. SERUM GLOBULIN: Result of Unpaired t test is as follows:

Table No.11: Inter group comparison of Serum Globulin levels Before and after treatment

SERUM GLOBULIN	Mean	SD	Test statistic	P value
Group A	0.02	0.0.35	1.352	0.182 (Not Significant)
Group B	0.01	0.032		

3.2.1.9. ALP: Result of Unpaired t test is as follows:

Table No.12: Inter group comparison of ALP levels Before and after treatment

ALP	Mean	SD	Test statistic	P value
Group A	26.46	36.162	-2.199	0.032 (Significant)
Group B	49.63	41.104		

3.2.2. LIPID PROFILE

3.2.2.1.TRIGLYCERIDE: Result of Unpaired t test is as follows:

Table No.13: Inter group comparison of Triglyceride levels Before and after treatment

TRIGLYCERIDE	Mean	SD	Test statistic	P value
Group A	18.95	20.881	-3.262	0.002 (Significant)
Group B	47.67	40.696		

3.2.2.2.TOTAL CHOLESTEROL: Result of Unpaired t test is as follows:

TableNo.14: Inter group comparison of Total Cholesterol levels Before and after treatment

TOTAL CHOLESTEROL	Mean	SD	Test statistic	P value
Group A	13.18	19.498	-1.689	0.097 (Not Significant)
Group B	24.04	27.114		

3.2.2.3.LDL: Result of Unpaired t test is as follows:

Table No.15: Inter group comparison of LDL levels Before and after treatment

LDL	Mean	SD	Test statistic	P value
Group A	11.08	12.825	-3.906	<0.001 (Significant)
Group B	25.87	14.916		

3.2.2.4. HDL: Result of Unpaired t test is as follows:

Table No.16: Inter group comparison of HDL levels Before and after treatment

HDL	Mean	SD	Test statistic	P value
Group A	1.283	4.453	6.572	<0.001 (Significant)
Group B	15.074	9.953		

3.2.2.5. VLDL: Result of Unpaired t test is as follows:

Table No,17: Inter group comparison of VLDL levels Before and after treatment

VLDL	Mean	SD	Test statistic	P value
Group A	0.59	1.505	-6.659	<0.001 (Significant)
Group B	12.85	9.453		

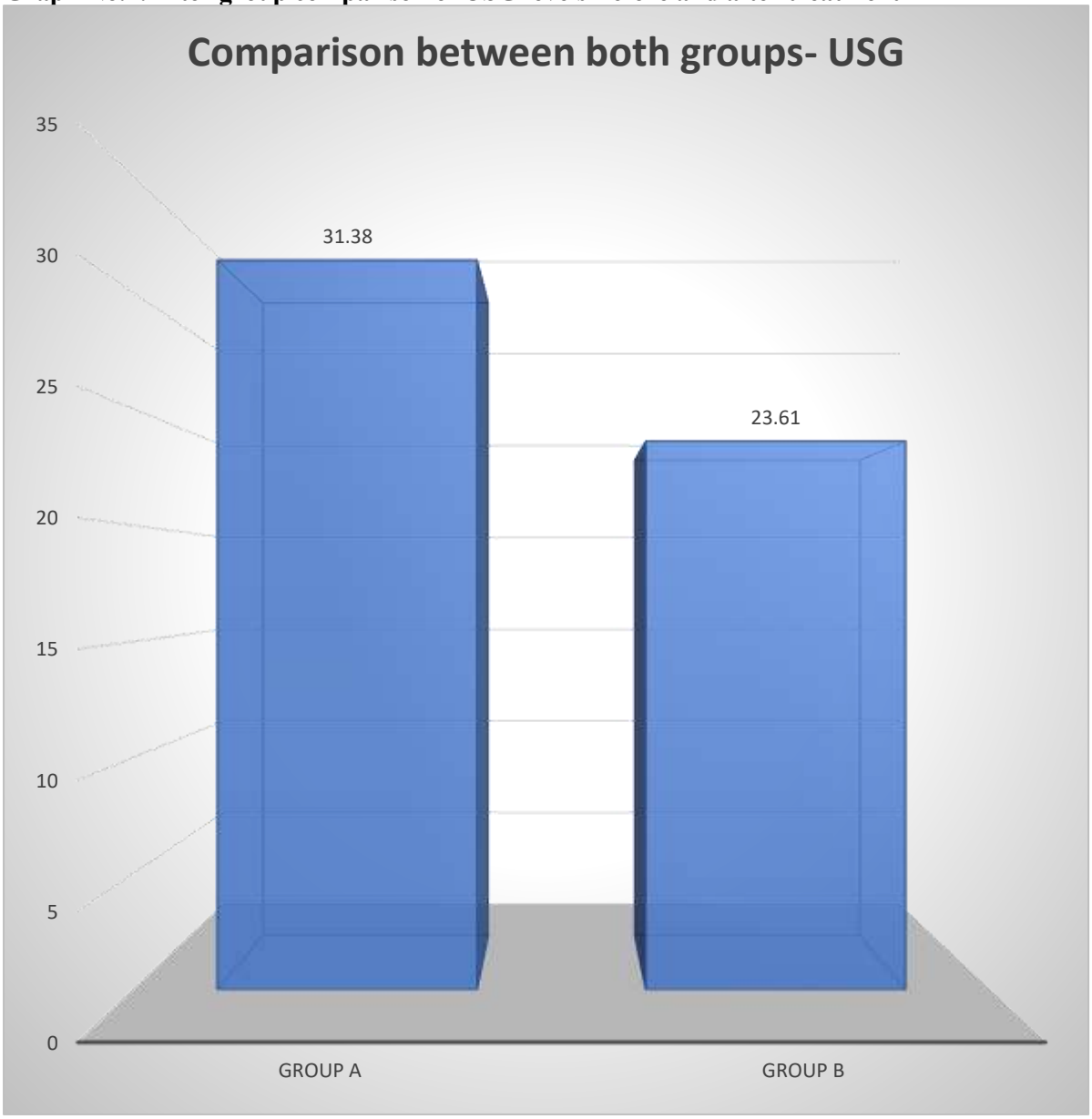
3.2.3. USG(ULTRASONOGRAPHY):

Result of before and after treatment by **Wilcoxon signed rank test** as follows:

Table No.18: Intra group comparison of USG levels Before and after treatment

USG	Mean Rank	Test statistic	P value
Group A	31.38	259.5	0.042 (Significant)
Group B	23.61		

Graph No.1: Inter group comparison of USG levels Before and after treatment



Interpretation: The Mann–Whitney U test was applied to compare the effect of treatment between the two groups with respect to USG. The results showed that Group A had a higher mean rank (31.38) compared to Group B (23.61). The test statistic was 259.5 with a p-value of 0.042, indicating that the difference between the two groups was statistically significant.
Since Group B has the lower mean rank, it suggests a greater reduction in USG compared to Group A. Hence, the treatment in Group B was found to be more effective than the treatment in Group A in reducing USG.

Table No.19: Summary of Comparison Between the Groups: Objective Parameters

Parameter	More Effective Group	Test Name	Test Statistic	P value	Significant?
SGPT	Group B	Unpaired t	-3.062	0.003	Yes
SGOT	Group B	Unpaired t	-2.167	0.035	Yes
TOTAL BILIRUBIN	Both Equal	Unpaired t	-1.796	0.078	No
INDIRECT BILIRUBIN	Both Equal	Unpaired t	-1.118	0.269	No
DIRECT BILIRUBIN	Group B	Unpaired t	-2.91	0.005	Yes
T. S. PROTEIN	Both Equal	Unpaired t	-0.337	0.738	No
S. ALBUMIN	Group B	Unpaired t	0.350	0.727	No
S. GLOBULIN	Both Equal	Unpaired t	1.352	0.182	No
A/G RIO	Both Equal	Unpaired t	1.391	0.170	No
ALP	Group B	Unpaired t	-2.199	0.032	Yes
TRIGLYCERIDE	Group B	Unpaired t	-3.262	0.002	Yes
T .CHOLESTEROL	Both Equal	Unpaired t	-1.689	0.097	No
LDL	Group B	Unpaired t	-3.906	<0.001	Yes
HDL	Group B	Unpaired t	6.572	<0.001	Yes
VLDL	Group B	Unpaired t	-6.659	<0.001	Yes

Table No.20: Summary of Comparison Between the Groups: Subjective Parameters

Parameter	More Effective Group	Test Name	Test Statistic (U)	P value	Significant?
USG	Group B	Mann–Whitney U	259.5	0.042	Yes

Interpretation: Group B showed a significantly greater treatment effect for most LFT Parameters (SGPT, SGOT, DIRECT BILIRUBIN, ALP), lipid parameters (LDL, HDL, VLDL, TRIGLYCERIDE).
Parameters like T .BILIRUBIN, IND BILIRUBIN, T. S. PROTEIN, S. GLOBULIN, S.ALBUMIN ,A/G ratio, and T .CHOLESTEROL showed no significant difference, indicating comparable effects between groups.
For the subjective parameter USG, Group B also had a significantly greater improvement.

3.3. Safety assessment

Patients' safety was assessed using medical history, clinical examination and laboratory reports. One patient from the Treatment Group refused to further continue in the study due to complaints of burning sensation of abdomen after consuming the study medication for 7 days. There were no significant alterations in the laboratory safety parameters in both study groups.

4.Discussion

The present clinical study was conducted on 54 patients, equally divided into two groups: Group A (lifestyle and dietary modification) and Group B (Panchakola Churna with Lifestyle and diet modification).
According to the demographic profile, The majority of patients were middle-aged adults, The predominance of NAFLD in the 30–40 and 40–50year age groups may be attributed to the peak occurrence of metabolic risk factors such as insulin resistance, central obesity, and sedentary lifestyle

during these decades, along with sufficient duration of exposure to unhealthy dietary habits, making this the period when the disease becomes clinically evident [11].
Male predominance (72%) was observed, consistent with studies showing higher NAFLD prevalence in men due to visceral adiposity, hormonal differences, and lifestyle factors. The prevalence and severity of NALFD are higher in men than in women during the reproductive age. However, after menopause, NAFLD occurs at a higher rate in women suggesting that estrogen protective. Estrogen exerts a protective effect against NAFLD by enhancing insulin sensitivity, promoting subcutaneous rather than visceral fat deposition, improving lipid metabolism by increasing HDL and reducing LDL levels, and directly inhibiting hepatic lipogenesis and inflammation, thereby reducing fat accumulation and progression of liver disease [12].
Both intervention groups showed statistically significant improvement in liver function parameters, lipid profile, and USG findings, confirming the effectiveness of lifestyle and dietary modifications in the management of NAFLD. However, Group B, which received Panchakola Churna along with lifestyle and diet modification, demonstrated markedly superior results. Significant reductions were observed in SGPT (40.2%), SGOT (18.9%), ALP (32.5%), triglycerides (30.8%), LDL (21.4%), and VLDL (22.8%), along with a notable increase in HDL (43.5%) and a remarkable 76.2% improvement in USG findings. These results indicate a highly significant therapeutic effect of Panchakola in improving both biochemical and structural parameters of NAFLD.

4.1. PROBABLE MODE OF ACTION OF THE TREATMENT

PANCHAKOLA CHURNA

Panchakola Churna, comprising Pippali, Pippalimoola, Chavya, Chitraka, and Shunthi, demonstrates significant therapeutic efficacy in Medosamchaya in Yakrit (NAFLD) when understood in the context of the Samprapti described in the present study. According to the thesis, the disease originates from continuous indulgence in Aharaja and Viharaja Nidanas such as Madhura, Snigdha, Guru Ahara, Avyayama, and Divasvapna, leading to Kapha Prakopa. This also results in the formation of Atisnigdha and Atimadhura Ahara Rasa, which preferentially nourishes Medo Dhatu due to Samanya-Vishesha Siddhanta. Simultaneously, Medo Dhatvagni becomes relatively increased, while Pachaka Ansha acting on Sthula Meda decreases, leading to excessive accumulation of Meda. This Meda circulates through Rakta and gets deposited in Yakrit, where, along with Rakta and Pitta Dushti, it produces Medosamchaya. Panchakola Churna, with its Katu Rasa, Ushna Virya, and Tikshna Guna, effectively counteracts these fundamental pathological change[13].
At the level of Agni, the Samprapti clearly indicates the presence of Tikshnagni or Vishamagni due to Avarana of Vata and Kapha predominance, rather than simple Agnimandya. Panchakola Churna acts by regulating this deranged Agni through its Deepana and Pachana properties, bringing it towards Samagni. By doing so, it prevents the formation of excessively Snigdha and Madhura Ahara Rasa, which is responsible for Meda vriddhi. Additionally, it helps in normalizing Medo Dhatvagni and correcting the imbalance between Dhatvagni and Pachaka Ansha, thereby reducing further Meda production and accumulation.
The Samprapti also emphasizes Srotorodha (Sanga) is a key factor in disease progression. Panchakola Churna, owing to its Tikshna and Ushna properties, performs Srotoshodhana by removing this obstruction and restoring normal flow within the channels. This facilitates proper circulation of Ahara Rasa and prevents its abnormal conversion into Meda. Furthermore, by clearing the obstruction in Medovaha Srotas, it alleviates the Avarana of Vata, thereby restoring the normal functioning of Samana and Vyana Vayu, which are essential for digestion, absorption, and distribution of nutrients.
In accordance with the described Samprapti, the excessive Meda formed is of both Abaddha (circulating lipids) and Baddha (stored fat) types, which accumulate particularly in Yakrit [13]. Panchakola Churna exerts a potent Lekhana and Medohara effect, which helps in reducing this pathological Meda. By virtue of its Kapha-Medohara action, it counteracts the Guru, Snigdha, and Picchila Guna of Meda and Kapha, thereby reducing hepatic fat deposition. Additionally, the formulation indirectly mitigates Pitta and Rakta Dushti at the Yakrit level by improving metabolic processes and preventing further inflammatory changes.
The individual components of Panchakola Churna possess bioactive phytochemicals that directly influence the pathogenesis of NAFLD. Pippali and Pippalimoola contain piperine[14], while Shunthi is

rich in gingerols and shogaols, which exhibit significant antioxidant and anti-inflammatory activities, thereby reducing oxidative stress and hepatocellular injury associated with fatty liver [15]. Chitraka contains plumbagin, known for its potent metabolic-stimulating and lipid-lowering effects, which aid in correcting deranged lipid metabolism and preventing excessive fat accumulation in the liver [16], while Chavya contributes to enhanced digestive and metabolic activity.

Previous pharmacological studies have demonstrated that these phytoconstituents improve hepatic enzyme levels, enhance lipid metabolism, and reduce inflammatory changes in the liver. Thus, the combined phytochemical action of Panchakola Churna supports its role in breaking the core pathology of NAFLD by targeting oxidative stress, inflammation, and abnormal lipid deposition in hepatocytes.

In conclusion, Panchakola Churna acts precisely in accordance with the Samprapti of Medosamchaya in Yakrit as described in the present study. It intervenes at multiple levels by correcting Agni Vaishamya, reducing Kapha-Meda Vriddhi, clearing Srotorodha, and normalizing Vata function. By breaking the pathological sequence of Ahara Rasa dushti, Meda accumulation, and Yakrit involvement, it effectively halts disease progression and promotes restoration of normal liver function, thereby establishing its importance as a rational therapeutic agent in the management of NAFLD.

4.2. LIFESTYLE WITH DIETARY MODIFICATION

Non-alcoholic fatty liver disease (NAFLD) is closely associated with modifiable lifestyle factors, particularly dietary patterns and physical inactivity, which contribute to the development of insulin resistance, central obesity, and hepatic fat accumulation. Diets rich in refined carbohydrates, added sugars (especially sugar-sweetened beverages such as tea and coffee), and excess caloric intake promote hepatic de novo lipogenesis, leading to triglyceride deposition within hepatocytes. Conversely, dietary modification focusing on calorie restriction, reduction of simple sugars, and adoption of a balanced diet rich in whole grains, fruits, vegetables, lean protein, and healthy fats has been shown to improve metabolic parameters and reduce liver fat. Increased intake of fibre and antioxidants further helps in reducing oxidative stress and inflammation, thereby preventing disease progression.

Lifestyle modification, particularly regular physical activity, plays a crucial role in the management of NAFLD. Both aerobic exercise and resistance training improve insulin sensitivity, enhance fatty acid oxidation, and reduce visceral adiposity, which are key mechanisms in decreasing hepatic fat content. Even in the absence of significant weight loss, exercise has been demonstrated to reduce liver fat and improve liver enzyme levels. A combination of moderate-intensity aerobic activity and strength training is therefore recommended to achieve optimal metabolic benefits[17].

Sustained weight reduction through dietary and lifestyle interventions remains the cornerstone of NAFLD management. A weight loss of approximately 5–10% has been associated with significant improvement in hepatic steatosis, inflammation, and even fibrosis in some cases. These beneficial effects are mediated through improved insulin sensitivity, decreased hepatic lipogenesis, reduced inflammatory cytokine production, and enhanced lipid metabolism. Therefore, comprehensive lifestyle modification integrating diet control, regular exercise, and behavioural changes is essential for both prevention and reversal of NAFLD and its progression to more advanced liver disease.

5.Conclusion

In conclusion, while lifestyle and dietary modifications alone play a crucial role in the management of NAFLD, their combination with Panchakola Churna provides a more pronounced and clinically significant outcome. The study highlights the synergistic effect of Deepana-Pachana and metabolic correction by Panchakola, along with regulation of Ahara and Vihara, in reversing the disease process. Hence, this integrated approach can be considered an effective, safe, and holistic management strategy for NAFLD in the present era.

Intergroup comparison further substantiated the superior efficacy of the combined therapeutic approach used in Group B. Statistically significant differences were observed in key parameters such as SGPT, SGOT, direct bilirubin, ALP, triglycerides, LDL, HDL, and VLDL, all favouring Group B. Additionally, USG findings, which reflect structural changes in the liver, showed a substantially greater improvement in Group B (76.2%) compared to Group A (29.5%), confirming better reversal of hepatic steatosis. While some parameters like total bilirubin, indirect bilirubin, total cholesterol, and serum globulin showed comparable effects between the groups, the overall outcome clearly indicates that the addition of Panchakola churna enhances the therapeutic efficacy beyond lifestyle modification alone.

These findings emphasize the importance of integrative management in NAFLD, combining dietary regulation, lifestyle correction, and targeted Ayurvedic intervention for optimal clinical outcomes.

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