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Research Article

Effect of *Kutaki* (*Picrorhiza Kurroa* Royle Ex Benth) on Dyslipidemia

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Abstract: Dyslipidemia is a lifestyle disorder related to a metabolic disorder of lipoprotein metabolism. It is mainly characterized by raised (TCH) total cholesterol, low-density lipoprotein (LDL) cholesterol, triglyceride (TG) levels, and a fall in the high-density lipoprotein (HDL) cholesterol levels. It is the key contributing cause of atherosclerosis, coronary artery ailment, and cerebrovascular disease. It can be equated with *medodushti* and included under *Santarpanjanya vyadhi* as "*Medoroga*. This study assesses the effect of Kutaki *(Picrorhiza kurroa* Royle ex Benth) on Dyslipidemia. A total of 30 patients with Dyslipidemia were treated with Kutaki tablets 500 mg twice a day before meals with lukewarm water for 60 days. They were assessed for Lipid levels, Fasting glucose level, AST, ALT, S.Creatinine, and Serum Urea on days 0, 30, and 60. Significant improvement was observed in all parameters after the completion of treatment. *Tikta Rasa (bitter taste), Ushna Virya (hot potency), Agnideepan, Pachana,* and *Lekhana* properties of Kutaki help break Samprapti. Active ingredients of Kutaki possess Hypolipidemic and choleretic properties, which help correct Lipid levels. Hepatoprotective and nephroprotective properties help in maintaining liver and kidney functions normally. It helps in improving deranged Agni and constipation. Kutaki can be useful in treating Dyslipidemia with any side effects.

Keywords- Dyslipidemia, Kutaki, Medoroga, Medodushti, Picrorhiza.

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1. INTRODUCTION

Dyslipidemia is a metabolic ailment of fat metabolism characterized by a fall in the (HDL) High-Density Lipoprotein cholesterol levels and increased triglycerides, LDL cholesterol, and total cholesterol.[1] The main cause of it is unhealthy dietary habits and lack of exercise. In dyslipidemia, the blood contains more lipids due to either an increased synthesis rate or a slower rate of lipoprotein breakdown. Dyslipidemia is an important contributing factor for atherosclerosis, coronary artery disease, and cerebrovascular disorder; Coronary Heart Disease incidence rises by 1% to 2% for every 1% increase in cholesterol levels.[2] According to numerous research, dyslipidemia in India ranges from 10 percent to 73 percent, based on factors including age, socioeconomic status, place of residence, and dietary and physical activity habits.[3] According to WHO, 23.6 million individuals globally will be affected by cardiovascular diseases by 2030.[4] Direct descriptions of Dyslipidemia are not mentioned in *Ayurvedic Samhita;* its etiopathogenesis and symptoms can be similar *medodushti*. It can be included under *Santarpanjanya vyadhi* as "*Medoroga*." Agni is needed for all metabolic processes in the body. Proper functioning of Agni results in maintaining a balance of *dosha, dhatu,* and *mala.* Derangement of *Agni* causes an imbalance of *dosha, dhatu,* and *mala. Mandagni* results in the formation of "*Ama*" (undigested/partially digested food), which is the root cause for the formation of all diseases. When *mandagni* is present at the *meda dhatu,* the successive formation of *dhatus* will not occur, and *apachita meda dhatu* will be in excess. This excess formed *meda dhatu* gets accumulated in the body, causing *Medoroga*.[5] *In Ayurveda, Aam* can be considered the primary root cause of all metabolism-related disorders. This *Ama* obstructs the *strotasa* leading to disease formation. Raised lipids are considered as Ama, produced due to impaired body metabolism. In *Ayurveda,* the best treatment for this condition involves the correction of *Agni, aampachana,* and *medanashana,* which causes the breaking of *samprapti*. Hence, the selected drugs should be *deepana, pachana*, *medohar, lekhana*, and *srotoshodhaka* properties to manage this condition.[6] Various single drugs and formulations with the above properties can be used to manage *Medoroga*. Kutaki (*Picrorhiza kurroa* Royle Ex Benth) is mentioned in Lekhaniya maha kashaya by Acharya Charak.[7] which can be used in this condition as it possesses all the above properties as listed in Table no 1.

1.1. *Need of the study*

Statins, bile acid sequestrants, cholesterol absorption inhibitors, and nicotinic acid are medications used to treat high cholesterol. Statins are commonly used for lowering serum lipid levels. It reduces the risk of Atherosclerotic cardiovascular disease (ASCVD) by 15% to 37%, but a residual 60% to 80% of ASCVD risk remains. Although effective pharmacological treatment for Dyslipidemia has been developed, long-term use of these drugs carries both costs and risks. Adverse effects of statins include myalgias, arthralgias, gastrointestinal symptoms, and increased liver function tests. Hence it cannot be given for liver and renal disorders .[8]Thus, there is a need for effective and safe herbal antihyperlipidemic drugs while simultaneously reducing the severe side effects. Many research studies have been carried out on Dyslipidemia in modern medicine and *Ayurveda*. Modern drugs used in treating Dyslipidemia are effective but cannot be used in liver and renal disorders due to their hepatotoxicity and nephrotoxicity. Animal studies have been conducted to prove the antihyperlipidemic and hepatoprotective actions of Kutaki.[9-11]This study is undertaken to evaluate the effect of *Kutaki* on Dyslipidemia. We aim to study the effect of Kutaki *(Picrorhiza kurroa* Royle ex Benth) on Dyslipidemia. The objective is to study the effect of *Kutaki* on serum lipid levels and to study the effect of *Kutaki* on liver and kidney function tests and blood glucose levels (fasting).

2. MATERIALS AND METHODS

2.1. *Material*

Trial drug-*Kutaki (Picrorhiza kurroa* Royle ex Benth) Kutaki is mentioned in Brihattrayi, Laghutrayi, and various Nighantus.

Fig 1: Dried Rhizomes of Kutaki

Fig 2: Tablets Kutaki (250mg)

2.2. *Source of obtaining*

Dried rhizomes of *Kutaki* were procured from the authentic shop, and the Dravyaguna Department of MGACH & RC made an identification.

2.3. *Identification characters*

The rhizomes are 4 to 8 mm thick, 2.5 to 8 cm long, subcylindrical, slightly curved or straight, and greyish-brown in color externally; the surface is rough due to the presence of stretch marks in its length, spherical scars of the root system, and bud scales, and sometimes roots connected. A feathery top of leaves encircles the upper end of the growing bud, and at times the corks exfoliate to expose the cortex. [12] [Fig 1]

2.4. *Preparation of Tablets*

Tablets were prepared as per the standard operating procedure mentioned in Sharangadhar Samhita in Dattatreya Rasashala, MGACH & RC [Fig 2]

Table 1: Properties of Kutaki[13-15]

Gana	*Bhedaniya, Lekhaniya*
Botanical Name	*Picrorhiza kurroa Royle ex Benth*
Family Name	*Scrophulariaceae*
English Name	*Black Hellebore*
Sanskrit	*Tikta*
Hindi	*Kutki*
Marathi	*Kutki, Kali kutki*
Rasa(taste)	*Katu(Pungent), Tikta(Bitter)*
Guna(property)	*Laghu(Light)*
Virya(potency)	*Ushna(Hot)*
Vipaka(metabolism)	*Katu(Pungent)*
Property	*Hridya, Pittahara, Deepan, Bhedan, Jvarahara, Lekhana, Amapachana.*
Action & uses	Hepatoprotective, immunomodulatory agent particularly for liver disorders & Jaundice, and fever.
Part use	Dried rhizomes
Chemical constituents	Bitter glycoside: Picroside, Kutkoside, Glucoside (Picrorhizin). Organic acids, resin, sugar and tannins, apocycyninandrosin, Androsin, Kutkiol, Kutkisterol, Apocyanin, D-mannitol, Iridoid glycosides, Phenol glucosides, and Picein as shown in (figure no 3).

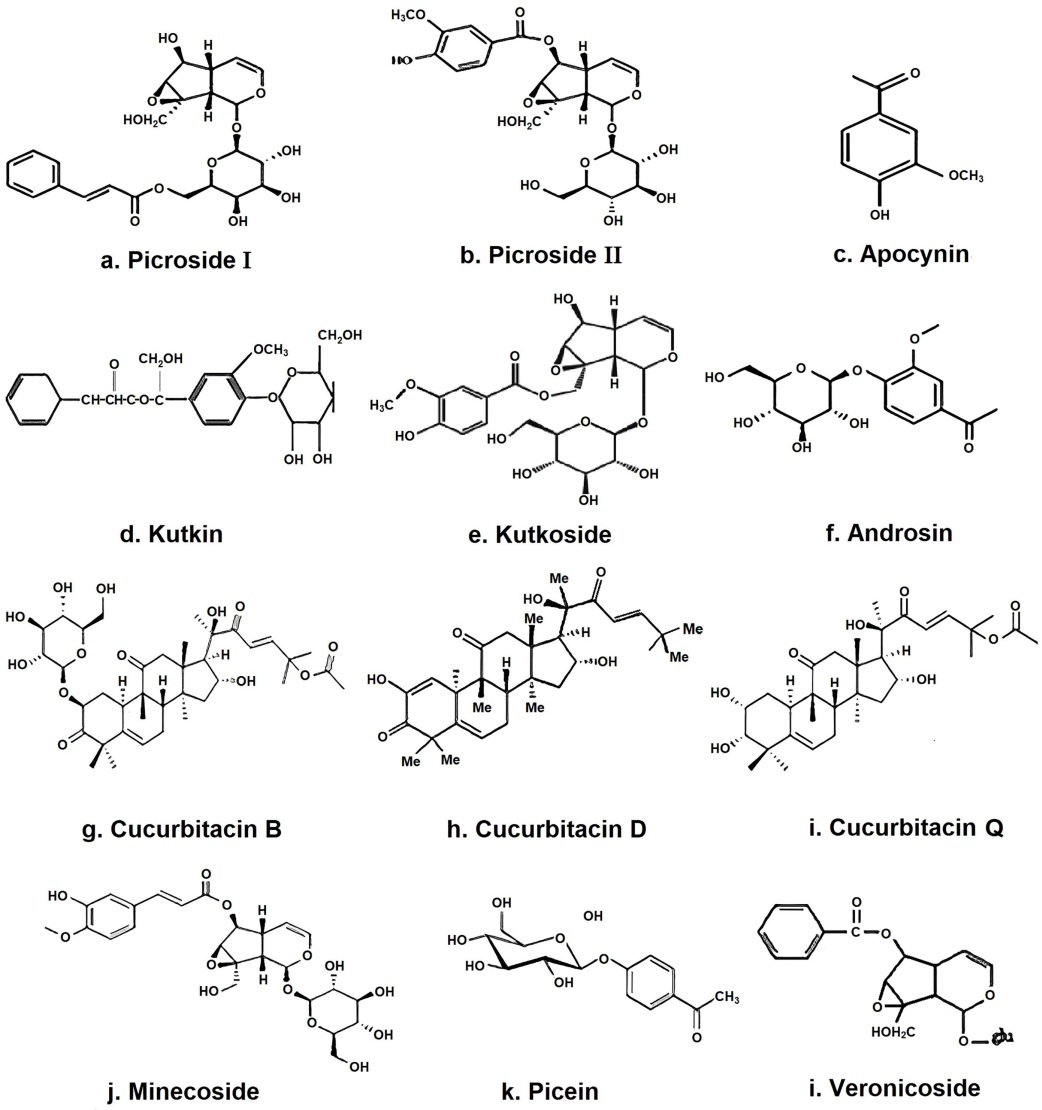

Fig 3: Showing Phytoconstituents extracted from Picrorhiza [16,17]

Above mentioned, phytoconstituents are Picroside I/II/III, Kutkoside, and Glucoside (Picrorhizin). In addition, organic acids, resin, sugar and tannins, apocycyninandrosin, Androsin, Kutkiol, Kutkisterol, and Apocyanin possess hypolipidemic, hepatoprotective, choleretic action, which is useful for improving lipid levels.

2.5. Dose

1- 3 gm of the drug in powder form.[18]

2.6. Aushasha sevankala

According to *Sharangdhar samhita, lekhana* drugs should be administered before meals; hence *Kutaki* is given before meals.[19]

2.7. Safety Study of Kutaki

A. Bal Krishna et al. conducted a toxicological study using a single dose of *Picrorhiza kurroa* rhizome extract in Animals-Wistar rats. They concluded that Picrorhiza showed no significant toxic effects. This data may be enough to start an interventional trial on standardized formulations of the extracts of Picrorrhiza based on the preclinical safety data.[20]

2.8. Methods

2.8.1. Place of work

Patients were selected from OPD as well as IPD of our Institute.

2.8.2. Institutional Ethical clearance

Ethical clearance and permission were obtained from the Institutional ethical committee (DMIMS (DU)/IEC/ 2018-19/7559). All investigations were conducted in the Pathology Laboratory of MGAC using the Randox assay kit method. Written informed consent from all patients was taken.

Type of Study - Clinical single arm.

Sample size- 30.

2.9. Criteria for Inclusion

1. Patients of either sex are between 30 and 60 years old.
2. Patients diagnosed having Dyslipidemia as per "(NCEP: ATP III, 2001)National Cholesterol Education Program and Adult Treatment Panel III" [21] as follows-

- TCH- Total cholesterol equal to or more than 200 mg/dL and

- LDL-Low Density lipoproteins –from 130 - 189 mg/dL and
- TG- Triglyceride levels ranging from 150 - 499 mg/dL and
- HDL – High-Density lipoproteins less than 40mg/dL.

3. Dyslipidemia associated with controlled NIDDM and hypertension

2.10. Exclusion criteria

1. Known cases of Unstable Angina (TIA) transient ischemic attack, stroke, MI- myocardial infarction, cardiovascular and major surgeries within 6 months before the screening visit.
2. Known cases of Tuberculosis, Carcinoma, Endocrine disorders, Renal or Liver disorder.
4. Patients with uncontrolled hypertension, Diabetes mellitus.
5. Drug-induced Dyslipidemia.

2.11. Withdrawal criteria

1. If any unwanted or untoward side effects are observed, then they will be treated free of cost
2. If the patient is no more willing to continue the treatment.

2.12. A stepwise method of study

IEC Clearance was taken

↓

Patients were selected according to the inclusion criteria.

↓

Written consent was taken.

↓

Investigations Lipid Profile, AST, ALT, S. Creatinine, S. Urea, BSL-fasting

↓

Kutaki-500mg is given twice a day before a meal with water.

↓

Duration-60 days

↓

Follow up-day 30 & 60

Table 2: Posology

Dose	500mg
Route of Administration	Orally in tablet form
Time	8 am & 8 pm before a meal
Anupan	Water
Frequency	Two times a day
Duration	60 days
Follow up During treatment(Review) After treatment (Follow up)	 Days 30 & 60 120 days

2.13. Assessment

2.14. Objective Criteria-On days 0, 30 and 60

- Body Mass Index-BMI
- TCH- Total cholesterol level
- HDL-High Density Lipoproteins level
- LDL-Low Density Lipoproteins level
- TG-Serum Triglycerides level
- Aspartate Aminotransferase (AST
- Alanine Aminotransferase ALT
- Serum urea
- Serum Creatinine
- Fasting-Blood sugar level

2.15. Investigations proposed

Lipid Profile

- TCH- Total cholesterol level
- HDL-High Density Lipoproteins level
- LDL-Low Density Lipoproteins level
- TG-Serum Triglycerides level
- Aspartate Aminotransferase (AST
- Alanine Aminotransferase ALT
- Serum urea
- Serum Creatinine
- Fasting-Blood sugar level

2.16. Statistical Analysis

The chi-square test and "student's paired t-test" were applied in the statistical data analysis, which used descriptive and inferential statistics. The analysis was conducted using SPSS 27.0 and Graph Pad Prism 7.0 software, with a significance level of ($p < 0.05$).

3. RESULTS

Data from 30 patients were collected by filling out predesigned case proforma, and assessment was done by doing investigations before, during, and after the completion of treatment.

Table. 3 Showing Demographic data		
Demographic Data	**Total patients(n=30)**	**Percentage**
Age Group(in years)		
30-40	9	30
41-50	6	20
51-60	15	50
Gender		
Male	16	53.33
Female	14	46.67
Total	30	100
Occupation		
Farmer	8	26.67
Housewife	10	33.33
Private Job	5	16.67
Service	7	23.33
Family History		
Yes	15	50
No	15	50
Prakruti		
Kapha Vataj	10	33.33
Pitta Kaphaj	13	43.33
Vataj Pittaj	7	23.33
Dietary habit		
Vegetarian	19	63.33
Mixed	11	36.67
Fried food		
Yes	20	66.67
No	10	33.33
Diwaswapa		
Yes	18	60
No	12	40
Physical Exercise		
Yes	11	36.67
No	19	63.33

Age Wise distribution of patients showed that out of 30 patients, a maximum of 15 (50%) patients were between the age group of 51-60 years, 9 patients (30%) were between 30-40 years, and 6 patients (20%) were between the age group of 41-50 years. The mean age was 47.93±10.93(30-60 years). Gender-wise distribution of patients showed that out of 30 patients, a maximum of 16 (53.33%) were Male, and 14 (46.67%) were female. Occupation-wise distribution of patients showed that out of 30 patients, a maximum of 10 (33.33%) patients were Housewives, 8 patients (26.67%) were farmers, 7 patients (23.33%) indulged in service, and 5 patients (16.67%) were doing private jobs. Family History wise distribution of patients showed that out of 30,15 patients (50%) had positive Family History, which was absent in the remaining 50% of patients. Prakruti-wise distribution of patients showed that out of 30 patients, a maximum of 13 (43.33%) patients had Pitta Kaphaj Prakruti, 10 patients (33.33%) had Kapha Vataj Prakruti, and 7 patients (23.33%) had Vataj Pittaj Prakruti. Dietary habit-wise distribution of patients showed that out of 30 patients, a maximum of 19 (63.33%) were vegetarian, whereas 11 patients (36.67%) had mixed dietary habits. The habit of fried food consumption was present in 20 patients (66.67%), and it was absent in 10 patients (33.33%). History of Diwaswapa was present in 18 patients (60%) and it was absent in 12 patients (40 %). History of Physical Exercise was present in 11 patients (36.67%) and absent in 19 patients (63.33%).

Table 4: Showing Comparison of the effect of therapy on Constipation and Agni on days 0, 30 and 60			
(n=30)	**Day 0**	**Day 30**	**Day 60**
Constipation			
Present	15(50%)	11(36.67%)	0(0%)
Absent	15(50%)	19(63.33%)	30(100%)
χ 2 & p-value	-	1.08,p=0.29,NS	20,p=0.0001,S
Agni			
Manda	11(36.67%)	7(23.33%)	2(6.67%)
Sama	9(30%)	17(56.67%)	28(93.33%)
Tikshna	7(23.33%)	3(10%)	0(0%)
Vishama	3(10%)	3(10%)	0(0%)

In the present study, it was observed that on day 0, Constipation was present in 15 (50%)patients, which was slightly reduced and present in 11 patients (36.67%) with nonsignificant $\chi 2$ and p values (1.08, p=0.29) on day 30. On day 60, no patient had constipation and thus showed highly significant improvement with $\chi 2$ and p-value (20, p=0.0001, S). In the present study, it was observed that on day 0, 11(36.67%), 9(30%), 7(23.33%), and 3(10%) patients had Mandagni, samagni, Tikshnagni, and Vishamagni, respectively which was present in 7 (23.33%), 17(56.67%), 3(10%) and 3(10%) respectively on day 30. On day 60, only 2(6.67%) patients had mandagni, and the remaining 28 patients had samagni, thus showing statistically significant improvement with $\chi 2$ and p-value (25.99,p=0.0001, S).

Table 5: Comparison of the effect of therapy on body weight, BMI, and BGL on days 0, 30 and 60

Body Weight in Kg						
Day	**Mean**	**N**	**SD**	**SE Mean**	**Mean Difference**	**t & p-value**
0	66.00	30	9.88	1.80	-	-
30	64.70	30	9.28	1.69	1.30±1.08	6.54 P=0.0001,S
60	63.20	30	8.77	1.60	2.80±2	7.64, P=0.0001,S
BMI						
0	26.52	30	3.41	0.62	-	-
30	25.98	30	3.19	0.58	0.54±0.49	5.92 P=0.0001,S
Day	**Mean of BGL**	**N**	**SD**	**SE Mean**	**Mean Difference**	**t & p-value**
0	102.63	30	8.43	1.53	-	-
30	99.56	30	5.79	1.05	3.06±5.96	2.81 P=0.009,S
60	98.76	30	6	1.09	3.86±8.43	2.51 P=0.018,S

The present study observed that on day 0, the mean body weight was 66.00, which was reduced to 64.70 and 63.20 on day 30 and day 60 with statistically significant (t=6.54 P=0.0001) and (t=7.64 P=0.0001) value respectively. The present study observed that on day 0, the mean BMI was 26.52, reduced to 25.980 and 25.401 on day 30 and day 60 with statistically significant (t=5.92 P=0.0001) and (t=7.98 P=0.0001) values, respectively. In this study, on day 0, the mean blood sugar level was 102.63, reduced to 99.56 and 98.76 on day 30 and day 60 with statistically significant (t=2.81 P=0.009) and (t=2.51 P=0.018) values, respectively.

Table 6: Comparison of the effect of therapy on Lipid Profile on days 0, 30 and 60

Day	**Mean**	**N**	**SD**	**SE Mean**	**Mean Difference**	**t & p-value**
TCH						
0	240.23	30	20.96	3.82	-	-
30	215.36	30	22.01	4.01	24.86±12.06	11.28 P=0.0001,S
60	197.36	30	17.70	3.23	42.86±18.76	12.51 P=0.0001,S
HDL						
0	38.76	30	5.95	1.08	-	-
30	40.66	30	5.65	1.03	1.90±1.06	6.48 P=0.0001,S
60	42.13	30	5.67	1.03	3.36±2.61	7.04 P=0.0001,S
LDL						
0	163.50	30	17.25	3.14	-	-
30	140.26	30	18.04	3.29	23.23±11.06	11.49 P=0.0001,S
60	124.33	30	14.09	2.57	39.16±17.52	12.24 P=0.0001,S
TG						
0	194.46	30	34.60	6.31	-	-
30	174.03	30	34.09	6.22	20.43±12.37	9.04 P=0.0001,S
60	156.43	30	33.52	6.12	38.03±22.26	9.35 P=0.0001,S
VLDL						
0	38.66	30	6.79	1.24	-	-
30	34.76	30	6.70	1.22	3.90±2.18	9.76 P=0.0001,S
60	30.90	30	6.67	1.21	7.76±4.22	10.07 P=0.0001,S

In this study, on day 0, the mean of Total Cholesterol was 240.23, reduced to 215.36 and 197.36 on day 30 and day 60 with statistically significant (t=11.28 P=0.0001) and (t=12.51 P=0.0001) values, respectively. On day 0, the mean of High-Density Lipoprotein was 38.76 which was increased to 40.66 and 42.13 on day 30 and day 60 with statistically significant (t=6.48 P=0.0001) and (t=7.04 P=0.0001) value respectively. In this study, on day 0, the mean of Low-Density Lipoprotein was 163.50, reduced to 140.26 and 124.33 on day 30 and day 60 with statistically significant (t=11.49 P=0.0001) and (t=12.24 P=0.0001) value respectively. In this study, on day 0, the mean of Triglyceride was 194.46, which was reduced to 174.03 and 156.43 on day 30 and day 60 with statistically significant (t=9.04, P=0.0001) and (t=9.35 P=0.0001) value respectively. In this study, on day 0, the mean of Very Low-Density Lipoprotein was 38.66which was reduced to 34.76 and 30.90 on day 30 and day 60 with statistically significant (t=9.76 P=0.0001) and (t=10.07 P=0.0001) value respectively.

Table 7: Comparison of the effect of therapy on AST, ALT, S. Creatinine, and S.Urea on days 0, 30 and 60

Day	Mean	N	SD	SE Mean	Mean Difference	t & p-value
AST						
0	28	30	7.73	1.41	-	-
30	26	30	5.30	0.96	2±3.76	2.90 P=0.007,S
60	24.43	30	5.50	1	3.56±6.21	3.14 P=0.004,S
ALT						
0	31.06	30	12.13	2.21	-	-
30	26.33	30	7.96	1.45	4.73±7.13	3.63 P=0.001,S
60	23.56	30	6.99	1.27	7.50±10.53	3.90 P=0.001,S
S.Creatinine						
0	0.95	30	0.25	0.04	-	-
30	0.92	30	0.18	0.03	0.03±0.11	1.48 P=0.14,NS
60	0.84	30	0.12	0.02	0.11±0.25	2.40 P=0.023,S
S.Urea						
0	25.46	30	6.43	1.17	-	-
30	24.96	30	6.08	1.11	0.50±2.78	0.98 P=0.33,NS
60	24.73	30	6.43	1.17	0.73±5.19	0.77 P=0.47,NS

In this study, on day 0, the mean of AST was 28 which was reduced to 26 and 24.43 on day 30 and day 60 with statistically significant (t=2.90 P=0.007) and (t=3.14 P=0.004) values, respectively. In this study, on day 0, the mean of AST was 31.06 which was reduced to 26.33 and 23.56 on day 30 and day 60 with statistically significant (t=3.63, P=0.001) and (t=3.90 P=0.001) values respectively. In this study, on day 0, the mean of Sr. Creatinine was 0.95, which was reduced to 0.92 and 0.84 on day 30 and day 60 with statistically non-significant (t=1.48, P=0.14) and statistically significant (t=2.40 P=0.023) value respectively. In this study, on day 0, the mean of Sr. Urea was 25.46 which was reduced to 24.96 and 24.73 on day 30 and day 60 with statistically non-significant (t=0.98 P=0.33) and statistically significant (t=0.77 P=0.47) values respectively.

4. DISCUSSION

The present clinical study was done to evaluate the effects of Kutaki on Dyslipidemia. In this study total of 30 patients who have Dyslipidemia were enrolled and treated with Kutaki tablet 500 mg two times a day before taking food with water for 60 days (Table no 2). Patients were assessed for Lipid levels, Fasting glucose level, AST, ALT, S.Creatinine, and Serum Urea on days 0, 30, and 60.

4.1. Demographic data

In the present study, Demographic data showed (Table 3) that the majority of patients, 15(50%), were in the age group of 51-60 years; advanced age is a major risk factor for dyslipidemia. The number of males in the present study was slightly more than in females. Dyslipidemia is more common in males than females. Regarding occupation, it was observed that homemakers followed by farmers are in greater numbers. Family history was present and absent in fifty percent of patients each. It was observed that Pittakaphaja prakriti, Vegetarians, non-addicted to any bad habits, were more in number. In this study, most subjects had a habit of consuming fried food and daytime sleep. All are contributing factors in developing dyslipidemia. In this study, 50% of subjects had constipation on a day which was improved and not present after completion of treatment. Before treatment, 3% of patients had samagni, and the remaining had manda, tikshna, and vishmagni, which showed improvement, and after completion of treatment, 93.33% of patients had samagni (Table 4).

4.2. Effect on objective parameters

Similarly, statistically significant (0.0001) reduction was observed in "Fasting glucose level, body weight, BMI (Table 5), Total cholesterol, Low-density lipoprotein, Triglycerides, Very low-density lipoproteins" and a statistically significant (0.0001) increase in "High-density lipoproteins" (Table 6). In addition, AST and ALT levels showed a statistically significant (0.0001) reduction. Still, the values are within normal limits before treatment (Table no.7). Serum Urea and Serum Creatinine showed no significant changes but that are within the normal range (Table no.7). Only non-elevation of AST, ALT, Serum Urea, and Serum Creatinine suggest that Kutaki had no adverse effect on liver and kidney due to its nephroprotective and hepatoprotective properties.

4.3. The action of Picrorhiza on Breaking of Pathogenesis

In Charaka Samhita, *Nidan Parivarjana* (avoidance of causative agents), *Shodhana* (Five purification measures), and *Shamana Chikitsa* (systemic internal medication) are described for the management of Medoroga. In *Shaman Chikitsa, deepana (*appetizer*), pachana* (digestive), and *lekhana* (scraping) drugs are described for *agnidipana* (appetite stimulation) and *amapachana* (digestion of undigested/partially food). Various herbs described in Ayurveda have lekhana and *kaphamedahar dravyas*, which can be used in *Medorog.*[22,23]*Kutaki* (*Picrorhiza kurroa* Royle ex. Benth) is one of the *lekhana dravya* described in Charaka samhita in *Lekhaniya maha kashaya* (Class of drugs causing scraping action). *Lekhana* and *ruksha* drugs like Kutaki, *Yava* (barley), and Musta are beneficial in reducing *Meda* and *Kapha.*[24] *Kutaki* has *Tikta Rasa (bitter taste), Ushna Virya (hot potency), Agnideepan, Pachana,* and *Lekhana* properties in Samhita. It is mainly used in all types of liver disorders like Jaundice and blood disorders.[25]*Amaghni* is the name given for *Kutaki* in *Dhanvantari nighantu* for its property of removing *Ama*. Thus it helps to cure diseases. It stimulates and gives strength to the liver.

4.4. Probable mode of action of Kutaki

Kutaki possesses *Kaphamedahar* property which helps to alleviate the increased *Kapha* and *Meda*. *Mandagni* gets corrected by *the Agnideepan* property of *Kutaki; the amapachana property* helps remove *Ama* and improves liver function, leading to breaking *Samprapti (*pathogenesis*). Lekhana* property helps in scraping action, thereby aiding in removing accumulated *Meda. Kutaki* has a straight action on the liver function. When the liver function improves, cholesterol synthesis may be checked, and cholesterol excretion may be improved by stimulating bile production with its secretion. The choleretic action of *P. kurroa* is known. Figure 3 shows it contains "iridoid glycosides (including pikuroside, picroside I, II, III, kutkoside, and 6-feruloyl catalpol), organic acids such as cinnamic and vanillic acids, glycosides like androsin, apocynin, and cucurbitacin". Its pharmacodynamic activity on lipids, primarily relevant to lipid diseases, can be predicted based on these qualities. [26]

4.5. Experimental studies conducted

Picroside I have already been demonstrated to be active in various liver damage models. [27] In a 12-week trial in rats supplied with a high-fatty food, 50 to 200 mg per kg of Picrorhiza kurroa every day appeared to correct most lipid metabolic markers (triglycerides, cholesterol, LDL-C) while not affecting HDL-C; these effects were assumed to be attributable to assisting the liver. PX-407-induced lipid changes have also been observed elsewhere. It has hepatoprotective, anti-cholestatic, antioxidant, antidiabetic, and immune-modulating properties. Kutaki's hepatoprotective properties can be ascribed to its ability to scavenge free radicals. It has also been demonstrated to accelerate rat liver regeneration, most likely through increased nucleic acid and protein synthesis.[28] Picrorhiza restored reduced glutathione levels in malaria-infected rats, improving detoxification and antioxidation and preserving a normal oxidation-reduction balance.[29] The active components "picroside I and kutkoside" protect the liver against damage induced by galactosamine in rats which is proved in the experimental study.[30] The experimental study conducted on mice in which Nimesulide was given Picrorhiza kurroa showed a protective effect at 2 doses of 250 mg per kg and 500 mg per kg for 14 days. To evaluate the impact of Nimesulide and Pk on the kidneys, renal function tests were performed, and urine PGE2 was assessed. Levels of Serum creatinine and Serum urea significantly decreased in rats given low and high doses of Picrorhiza. It demonstrated the nephrotoxic potential of Nimesulide, and Pk is an effective herbal anti-inflammatory and nephroprotective substitute.[31] Dyslipidemia is a lifestyle disorder and is related to unhealthy dietary habits, obesity, consumption of alcohol, habits like smoking, tobacco, and tea, family history, presence of hypertension, diabetes mellitus, and obesity. All risk variables do not play a role in disease development, but they differ from person to person. All etiological components don't need to be present in dyslipidemia's pathogenesis; nonetheless, certain of them can induce it.[32] Husain GM et al. studied the Potential mechanism of anti-diabetic activity of Picrorhiza kurroa. They revealed that standardized PkE increased the insulin-mediated translocation of GLUT-4 from the cytosol to the plasma membrane, resulting in better glucose uptake by skeletal muscles and improved glycaemic control in diabetic rats. Effect on GLUT-4 GLUT-4 protein content in total membrane fractions of STZinduced diabetic rats was significantly reduced compared to normal control. PkE significantly increased the GLUT-4 protein level compared to diabetic control. Glibenclamide treatment did not significantly increase GLUT-4 protein compared to diabetic control.[33] In this study, faulty dietary habits, deranged Agni, lack of exercise, and obesity can cause dyslipidemia.

5. CONCLUSION

This study concluded that old age, pittakaphaja prakriti, consumption of fried food, daytime sleep, sedentary lifestyle, and psychological stress are contributing factors involved in Dyslipidemia. Kutaki helps in correcting constipation, Agni and helps in improving metabolism. Kutaki had no adverse effect on the liver and kidney due to its nephroprotective and hepatoprotective properties. Due to Kaphamedohar and lekhan properties, it helps reduce Fasting glucose level, body weight, BMI, (TCH) Total cholesterol, (LDL) Low-density lipoprotein, (TG) Triglycerides, (VLDL), Very low-density lipoproteins and in increasing (HDL) High-density lipoproteins. Hence it can be used in Dyslipidemia.

6. LIMITATIONS

It is a single-arm study and includes less sample size.

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8. AUTHORS CONTRIBUTION STATEMENT

Dr. Sadhana Misar Wajpeyi conceptualized the idea for conducting the study and collected data, and prepared the manuscript. Dr. Ketki Wajpeyi helped in the assessment of patients and preparation of the manuscript.

9. CONFLICT OF INTEREST

Conflict of interest declared none.

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