


Research Article
EFFECT OF ERANDAPATHRAKSHARA (*RICINUS COMMUNIS* LINN.) ALONG WITH HINGU (*FERULA NARTHEX* LINN.) IN DYSLIPIDEMIA
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ABSTRACT

Cardiovascular diseases (CVD) are of prime importance among lifestyle disease and dyslipidemia is an independent and modifiable risk factor for CVD, together with smoking and sedentary habits, fatty diets and stressful life situations. Dyslipidemia can be included under the term *Medovikaras* in Ayurveda. A combination of *Erandapathrakshara* (*Ricinus communis* Linn) and *Hingu* (*Ferula narthex* Linn) is mentioned in *Bhavaprakasa* for curing *Medovridhi*. At present era, many modern medications are available for curing the elevated lipid levels. But they cause many side effects which are extremely hazardous to human health. So a clinical study is selected with an interest to revalidate the ancient scientific knowledge. Genuine samples of both *Erandapathra* (*Ricinus communis* Linn) and *Hingu* (*Ferula narthex* Linn) were collected. *Thekshara* of *Erandapathra* and powder of purified *Hingu* were given to the patient with *Mandam* (supernatant liquid portion of rice soup) as *Anupaana* for a period of 1 month. Patients of both sexes in the age group of 30-60 years with serum LDL above 130mg/dl, non HDL-C above 139mg/dl from OPD. The statistical analysis of changes in Lipid profile values before treatment, after treatment, and after follow up are the assessment criteria. Trial drugs were found to be effective in reducing T. Cholesterol, LDL, triglycerides and Non-HDL-c but not effective in reducing VLDL and increasing HDL and no side effects were observed during study period. From the present study we can conclude that the trial drugs were effective and can be used safely in clinical practice in future.

KEYWORDS: Dyslipidemia, *Ricinus communis* Linn, *Ferula narthex*. Linn, LDL, Non-HDL-C, T. cholesterol, VLDL, Triglycerides, HDL.

INTRODUCTION

Ayurveda offers a strong framework for multimodal preventive medicine. It is also patient friendly, non-pharma compliment for managing chronic diseases refractory to allopathic therapies. Traditional medicine uses different plant parts and extracts derived from medicinal plants. High cost of new drugs, increased side effects of modern medicines, microbial resistance and emerging diseases are some of the reasons for more interest from public in using complementary and alternative medicine nowadays. But most of the researchers consider Ayurveda as pseudoscientific as it lack scientific evidence in treatment. So it is our duty to conduct more researches in the field of Ayurveda and ignite this as evidence based science.

A combination of *Erandapathrakshara* and *Hingu* is mentioned in *Bhavaprakasa* for curing *Medovridhi*^[1]. *Medovridhi* may be correlated to the condition dyslipidemia. Dyslipidemia is the elevation of plasma cholesterol, triglycerides, or both or a low level of High density lipoprotein^[2]. This may contribute to the formation of atherosclerosis. The incidence of dyslipidemia is increasing in many developing countries due to westernization of diets and other lifestyle changes. Correction of dyslipidemia can reduce the risk of heart disease by 30% over a 5 year period^[3]. NCEP (National Cholesterol Education Programme) guidelines of United

States recommend cholesterol levels less than 200mg/dl^[4]. The values exceeding 240mg/dl are considered as high risk factor. The prevalence of dyslipidemia is very high in India which calls for urgent lifestyle intervention strategies to prevent and manage this important cardiovascular risk factor.

Updated 2016 guidelines deal with the management of dyslipidemia as an essential and integral part of cardiovascular disease prevention^[5]. The conventional cholesterol lowering drugs like Statins, Bile acid sequestrants, Ezetimibe and Fibrates causes many side effects including elevation of liver enzymes, nausea, constipation, hyperurecemia etc^[6]. So there is a need to invent a new, cost effective and safe medicine to cure dyslipidemia.

Medicinal plants have been identified and used from prehistoric times. Plants make many chemical compounds for biological functions, including defence against insects, fungi and herbivorous mammals. Over 12000 active compounds are known to science. So more works should be conducted in these herbs to revalidate the ancient scientific knowledge. As evidence based medicine is the need of the hour, this study is an attempt to test clinically the efficacy of *Erandapathrakshara* along with *Hingu* in dyslipidemia.

AIMS AND OBJECTIVES

To evaluate clinically the effect of *Erandapathrakshara* along with *Hingu* in dyslipidemia by orally administering the drug for 30 days and to assess the lipid profile values before treatment (BT), after treatment (AT) and after 30 days follow up.

MATERIALS AND METHODS**Collection of the drug**

The study drug *Eranda* (*Ricinus communis* Linn) (20kg) was collected from its natural habitat, Karunagappally, Kollam district, Kerala. 500g of Medicinal *Hingu* (*Ferula narthex* Linn) was purchased for the study.

Preparation of drug

The fresh plants were collected, leaves were separated along with petiole, washed well and shade dried. The *Kshara* was prepared from it according to the method mentioned in AFI.

Drug is cut into small pieces and dried well. Pieces are put in earthen pot and burnt to ash. The ash is allowed to cool. Water is added in the ratio 6:1 and mixed well. Then strained through a piece of cloth. This process of straining done 2-3 times till a clear liquid is obtained. This liquid is then put in an iron/earthen vessel and heated over a moderate fire till the water evaporates leaving a solid salty white substance which is collected known as *Kshara*. (AFI)^[7]

500 g of *Kshara* is prepared from 20kg of leaves; 250 mg of *Kshara* was measured, and packed tightly in butter papers.

Medicinal *Hingu* (*Ferula narthex* Linn.) was purchased and identity tests were done in the lab for checking its genuineness. *Hingu* was purified by frying with *Ghritha* until it becomes dry and hard. Then it is powdered well for internal use and 125mg is packed air tightly in butter papers.

Inclusion Criteria

1. Patients of both sexes in age group of 30-60 years..
2. LDL level above 130mg/dl, non HDL-C level above 139 mg/dl.

Exclusion Criteria

1. Pregnant and lactating woman
2. Known cases of medication for coronary artery disease, cerebrovascular accidents, any type of malignancy, renal and hepatic disorders.
3. Those who are contraindicated for *Kshara*.

Sample Size

Total 33 patients were selected from the outpatient department, Dravyagunavijnana, Govt. Ayurveda college hospital, Trivandrum, having dyslipidemia confirmed by assessing lipid profile.

Dose

Dose of *Kshara* (R. T)-250 mg-1g.^[8]

Dose of *Hingu* (API) 10-125-500mg of drug.^[9]

An intermediate values of the above mentioned weights of *Kshara* and *Hingu* are opted as dosage for pilot study. Thus, 250mg of *Kshara* mixed with 125mg of

purified *Hingu* powder (in the ratio 2:1) and a total 375mg medicine will be given with *Mandam* twice daily.

Duration

1 month, and follow up for 1 month

Route of administration

Oral

Collection of data

Data was collected before the study, after the study and after 30 days follow up by interview and laboratory investigations according to the structured case proforma, a model of which is given in the appendix. The data includes personal data such as name, religion, occupation etc. , data related to disease such as chief complaints and its duration, history of present and past illness, menstrual history, history of obstetrics etc. General and systemic examinations were conducted.

Investigations

Dyslipidemia was assessed by checking serum lipid profile and non-HDL cholesterol of the patient before and after the study and after follow up. Lipid profile includes Total cholesterol, HDL, LDL, VLDL and Triglycerides.

Assessment Criteria

Laboratory findings- BT, AT and after follow up.

Total cholesterol, HDL, LDL, VLDL, Triglycerides, non-HDL-C.

Ethical consideration

Every patient was selected in the study after obtaining an informed consent from them. Before starting the study, No Objection Certificate was obtained from Institutional Ethical Committee (IEC) with ref. no IEC 135/28. 4. 2015.

Observation, Analysis and Results

Majority of the patients were in between age group 51-60 years. In this study 30% of patients were male and 70% were female. 94% belonged to middle class, 6% were poor, and 0% was rich. 45% were residing in rural area and 55% were residing in urban area. 6% were vegetarians and 94% were taking both vegetarian and non-vegetarian food. No patients had poor appetite, 97% had moderate and 3% had increased appetite. 6% had no exercise, 79% had mild exercise and 21% had moderate exercise.

Effect of Treatment on Lipid Profile

The average Lipid profile values before treatment, after treatment and after follow up are given below in the table.

Table 1: Lipid Profile Values

| Lipid profile values | BT | AT | AF |
|----------------------|---------|---------|---------|
| Serum cholesterol | 251. 27 | 241. 85 | 243. 09 |
| LDL | 181. 39 | 171. 76 | 172. 64 |
| VLDL | 22. 82 | 21. 24 | 21. 85 |
| Triglycerides | 117. 45 | 105. 82 | 107. 27 |
| HDL | 47. 12 | 48. 52 | 48. 09 |
| Non-HDL c | 204. 09 | 193. 33 | 194. 91 |

Statistical Analysis

The collected data were subjected to statistical analysis using SPSS v. 16 for WINDOWS. For categorical variables frequencies and percentages were used as summary measures. For continuous variables, mean and standard deviations were used as summary measures. For

all continuous variables, before-after treatment effect was assessed using paired sample t test. Hence for testing the effectiveness of treatment, statistical significance of before-after treatment effects was carried out using paired t test.

Table 2: Effectiveness of treatment on T. cholesterol

| | N | Total cholesterol (mg/dL) | | Effect | Paired Differences | | % of change | Paired t test | |
|----|----|---------------------------|-------|--------|--------------------|-------|-------------|---------------|------|
| | | Mean | Sd | | Mean | Sd | | t | P |
| BT | 33 | 251.27 | 24.67 | BT-AT | 9.42 | 18.03 | -3.75 | 3.002 | .005 |
| AT | 33 | 241.85 | 25.67 | AT-AF | -1.24 | 5.15 | 0.51 | 1.385 | .176 |
| AF | 33 | 243.09 | 26.66 | BT-AF | 8.18 | 19.31 | -3.26 | 2.434 | .021 |

The paired t test showed that the decrease in cholesterol after treatment is statistically significant ($t=3.002$, $p<0.05$) and there is significant effect in reducing cholesterol after follow up also ($t=2.434$, $p<0.05$). But there is no significant effect from after treatment to after follow up ($t=1.385$, $p>0.05$).

Table 3: Effectiveness of treatment on LDL

| | N | LDL (mg/dL) | | Effect | Paired Differences | | % of change | Paired t test | |
|----|----|-------------|-------|--------|--------------------|-------|-------------|---------------|------|
| | | Mean | sd | | Mean | Sd | | t | P |
| BT | 33 | 181.39 | 27.15 | BT-AT | 9.64 | 20.08 | -5.31 | 2.757 | .010 |
| AT | 33 | 171.76 | 23.87 | AT-AF | -0.88 | 3.83 | 0.51 | -1.318 | .197 |
| AF | 33 | 172.64 | 22.89 | BT-AF | 8.76 | 17.89 | -4.83 | 2.812 | .008 |

The paired t test showed that the decrease in LDL after treatment is statistically significant ($t=2.757$, $p<0.05$) and there is significant effect in reducing cholesterol after follow up also ($t=2.812$, $p<0.05$). But there is no significant effect from after treatment to after follow up ($t=1.318$, $p>0.05$).

Table 4: Effectiveness of treatment on VLDL

| | N | VLDL (mg/dL) | | Effect | Paired Differences | | % of change | Paired t test | |
|----|----|--------------|------|--------|--------------------|------|-------------|---------------|------|
| | | Mean | sd | | Mean | sd | | T | P |
| BT | 33 | 22.82 | 8.67 | BT-AT | 1.58 | 6.35 | -6.91 | 1.425 | .164 |
| AT | 33 | 21.24 | 6.11 | AT-AF | -0.61 | 1.80 | 2.85 | 1.932 | .062 |
| AF | 33 | 21.85 | 6.37 | BT-AF | 0.97 | 5.90 | -4.25 | .945 | .352 |

The paired t test showed that there is no significant effect in reducing VLDL level after treatment ($t=1.425$, $p>0.05$) and after follow up ($t=0.945$, $p>0.05$). There is no significant effect from after treatment to after follow up also ($t=1.932$, $p>0.05$).

Table 5: Effectiveness of treatment on Triglycerides

| | N | Triglycerides (mg/dL) | | Effect | Paired Differences | | % of change | Paired t test | |
|----|----|-----------------------|-------|--------|--------------------|-------|-------------|---------------|------|
| | | Mean | sd | | Mean | Sd | | T | P |
| BT | 33 | 117.45 | 42.70 | BT-AT | 11.64 | 29.77 | -9.91 | 2.245 | .032 |
| AT | 33 | 105.82 | 29.36 | AT-AF | -1.45 | 2.87 | 1.37 | 2.908 | .007 |
| AF | 33 | 107.27 | 29.71 | BT-AF | 10.18 | 28.03 | -8.67 | 2.087 | .045 |

The decrease in Triglycerides after treatment is statistically significant ($t=2.245$, $p<0.05$) and there is significant effect in reducing Triglycerides after follow up also ($t=2.087$, $p<0.05$). There is a significant effect from after treatment to after follow up also ($t=2.908$, $p<0.05$).

Table 6: Effectiveness of treatment on HDL

| | N | HDL (mg/dL) | | Effect | Paired Differences | | % of change | Paired t test | |
|----|----|-------------|------|--------|--------------------|------|-------------|---------------|------|
| | | Mean | sd | | Mean | Sd | | T | P |
| BT | 33 | 47.12 | 5.82 | BT-AT | -1.39 | 6.45 | 2.96 | 1.242 | .223 |
| AT | 33 | 48.52 | 5.24 | AT-AF | 0.42 | 1.95 | -0.87 | 1.248 | .221 |
| AF | 33 | 48.09 | 4.73 | BT-AF | -0.97 | 5.77 | 2.06 | -.965 | .342 |

The paired t test showed that the increase in HDL level after treatment is statistically not significant ($t=1.242$, $p>0.05$) and there is no significant effect in increasing HDL after follow up ($t=1.248$, $p>0.05$). There is no significant effect from after treatment to after follow up also ($t=.965$, $p>0.05$).

Table 7: Effectiveness of treatment on Non HDL-C

| | N | Non-HDL-C (mg/dL) | | Effect | Paired Differences | | % of change | Paired t test | |
|----|----|-------------------|-------|--------|--------------------|-------|-------------|---------------|------|
| | | Mean | sd | | Mean | sd | | t | P |
| BT | 33 | 204.09 | 24.51 | BT-AT | 10.76 | 18.68 | -5.27 | 3.309 | .002 |
| AT | 33 | 193.33 | 24.02 | AT-AF | -1.58 | 6.28 | 0.82 | -1.443 | .159 |
| AF | 33 | 194.91 | 25.31 | BT-AF | 9.18 | 19.67 | -4.50 | 2.681 | .011 |

There is a significant effect in reducing Non HDL-C level after treatment (t=3. 309, p<0. 05) and after follow up (t=2. 681, p<0. 05). But there is no significant effect from after treatment to after follow up (t=1. 443, p>0. 05).

DISCUSSION

Dyslipidemia can be correlated with *Medovridhi* or *Medovikara* described in Ayurvedic classics. Many signs and symptoms mentioned by the increase of *Medodhatu* in body. Quantitative increase in various forms of lipids may be correlated with these signs and symptoms of *Medovridhi*.^[10] It can also be comparable with the condition of *Athisthoulya* mentioned in Samhithas.^[11] A combination of *Erandapathrakshara* and *Hingu* is mentioned in Bhavaprakasa for curing *Medovridhi*.^[1] So a clinical study was conducted to assess the effect of this combination in *Medovridhi* by assessing the lipid profile values.

By analyzing sociodemographic data, 45% patients were in age group 51-60. This may be due to unsatisfactory food habits and degenerative changes of body cells. It reveals 70% patients were females which substantiates dyslipidemia is more prevalent among females. Majority of the patients are from middle class families (94%), indicates that it is a serious issue which affect majority of the public. 55% of patients from urban area which indicates that the disease is more prevalent in them due to taking fast food, junk food etc and may be due to polluted atmosphere.

By analyzing statistical data, it can be stated that the study drug is effective in reducing TC, LDL, Non HDL-C and triglycerides and the effect is sustained after follow up also. It is not effective in reducing VLDL and increasing HDL.

probable mode of action of the drug

Medovikara is a condition of increased *Medodhatu* associated with increased *Kaphadosha* and deranged *Kleda* in body. It is characterized by excessive accumulation and deposition of *Medodhatu* obstructing the normal function of *Medovahasrotas*. Aetiological factors of *Medovikara* includes excessive involvement of food having the qualities like *Snigdha*, *Guru*, *Picchila*, *Sheeta* and *Madhura rasa*, lack of activity, lack of exercise, habituate day sleep etc. It will lead to increase state of *Kapha*, *Meda* and *Kleda* in the body. *Medodhatu* formed in the body is in *Apakva* form due to *Dhathvagnimandya*. So the treatment should aim at the correction of *Agni* and *Lekhana* of the accumulated *Medas*.

In *Medovikaras*, due to *Medodhatvagnimandhya*-*Ashthayimedas* increases in quantity and enters the circulation. Due to its *Pichilaguna* (sliminess), it gets lodged in susceptible *Srotases* (physiological channels) causing *Margavarodha* (obstruction). So the drugs should have *Ruksha* (rough), *Chedana* (breaking), *Snehamedakleda visoshana* property (reduces) to produce *Srothovisodana* (purification of channels). Both *Kshara* and *Hingu* is mentioned under *Chedana* group of drugs^[12,13]. Both the drugs have *Katurasa* (pungent taste), *Ushnaveerya* (hot in potency), *Sukshma* and *Teekshna* properties.^[13,14] *Hingu* is with *Katuvipaka* also^[14]. The drug *Erandapathrakshara* is with *Katu*, *Tikta rasa* and *Ushnaveerya*^[15]. So there may be an effect of this combination in reducing fat molecules in the body. So it may act at *Dhatvagni* level,

correcting *Medodhatvagnimandya* and helps in formation of proper *Medodhatu* in the body.

The probable mode of action of a substance can be evaluated through its *Rasapanchaka* and *Panchabhautika* constitution. In the study, both the drugs i.e., *Erandapathrakshara* and *Hingu* are having *Katurasa*, *Katuvipaka* which are of *Vayu*, *Akasa* and *Agnimahabhoota* predominance. These are opposite to *Gunas* of *Prithvi* and *Jala*. Hence they will reduce *Bhujaladhikamedas*. *Ushnaveerya* of both drugs stimulates *Jatharagni* which in turn stimulates *Medodhatvagni* and ultimately reduces over production of *Medas*. Apart from that these drugs are also *Laghu* and *Teekshnagunas* which help in *Lekhana* karma. *Sharngadhara* included *Kshara* in *Chedana* group of drugs^[16]. So it may scratch out (*Unmoolayathi*) the adherent *Kapha* and other *Doshas* from their places due to its *Ushna*, *Laghu* and *Theekshna* properties.

It is mentioned that in *Kaphaja* diseases, drug should be used in an order of *Katu*, *Tiktha* and *Kashaya rasa*^[17]. As *Medodhatu* is having *Snigdha* (unctuousness), *Guru* (heaviness) and *Pichilaguna* (sliminess) it is moreover similar to *Kaphadosha* and *Medovridhi* can be considered as a *Kaphajavyadhi*. Both the drugs i.e. *Erandapathrakshara* and *Hingu* are having *Katurasa*, so they have a predominant role in curing *Kaphaja* disease. The *Katurasa*, which is used first, will subside the *Pichilathwa* (sliminess) and *Guruthwa* (heaviness) of *Kaphadosha* resulting in alleviation of disorders related to *Kaphadosha*.

Thus it can be concluded that the study drugs *Erandapathrakshara* and *Hingu* is effective in reducing total cholesterol, LDL, Triglycerides and Non HDL-C.

CONCLUSION

The study drug is found to be effective in lowering total cholesterol, LDL, Non HDL-c and triglycerides but not effective in increasing HDL level and decreasing VLDL level. No adverse effects were observed during study, so the study drugs can be used safely for clinical practice.

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