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COMPARATIVE CLINICAL STUDY OF VRIKSHAMALA (GARCINIA INDICA) AND KATUKI (PICRORRHIZA KURROA) IN THE MANAGEMENT OF METABOLIC SYNDROME

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ABSTRACT

Metabolic Syndrome is a set of risk factors that includes abdominal obesity, Insulin resistance, Hypertension; Dyslipidemia (raised triglycerides and low HDL-cholesterol). Parallel to this s in Ayurveda Sthaulya is the condition. Material and Methods, Diagnosed 110 patients on basis of inclusion and exclusion criteria, study design under demographic profile, clinical profile and laboratory profile (hematological, biochemical test), were selected for the present clinical work. The patient were divided into four Groups I, II, III and IV in terms of the nature of given treatment. Group I, II, III and IV comprised of 27, 27, 28 and 28 patients of metabolic syndrome respectively. Results study showed the significant changes over all the parameters of the syndrome. Conclusion: Study concluded that its Preliminary study conducted as a part of education research programme and further more clinical and experimental studies are necessary to establish the above observations. We sincerely hope that the present study would be pioneer as an ideal research work in the field of Metabolic Syndrome and would provide useful lead for coming generations and future research workers.

KEYWORDS: Metabolic Syndrome, Ayurveda, Katuki, Vrkshamala.

INTRODUCTION

Metabolic syndrome in its path physiological perspective mainly deals with the regulation of insulin resistance, atherogenic dyslipidemia, hypertension, impaired glycemia, pro-inflammatory state or endothelial dysfunction, pro-thrombotic state, abnormal fat metabolism, fatty liver, abnormal ovarian androgen secretion.

Prevalence of the metabolic syndrome varies across the globe, in part reflecting the age and ethnicity of the populations studied and the diagnostic criteria are applied. In general, the prevalence of metabolic syndrome increases with age. The highest recorded prevalence worldwide is in Native Americans, with nearly 60% of women ages 45–49 and 45% of men ages 45–49 meeting National Cholesterol Education Program, Adult Treatment Panel III (NCEP:ATPIII) criteria. An evaluation of data that represents the entire U.S. population found that about 25% of adults have M.S. In India, disease profile is changing rapidly. The WHO has identified India as one of the nation that is going to have most of the lifestyle diseases in near future. The population at risk has shifted from 40+ to 30+ or even younger. Life style diseases are an important cause of premature mortality. The epidemiological transition has resulted in decrease in deaths due to infection to a concomitant rise in cardiovascular disease. According to WHO reports 2003 an estimated 16.7 millions of total global deaths result from cardio vascular disease. Obesity has reached epidemic proportions globally with >1 billion individuals overweight, is a major contributor to global burden of chronic diseases. Thus, in view of this upcoming challenging problem of Metabolic Syndrome which is slowly engulfing the country's potential it was decided to screen out the patients of Metabolic Syndrome from daily OPD and IPD of SSH, and to make an effort to reduce the global burden of CVD by making patients aware of future consequences. As a journey of thousands miles begins with a single step, thus the present study launched with the broad based.

Due to rapid urbanization and industrialization the incidence of metabolic disorders particularly diabetes mellitus, hypertension, obesity is increasing worldwide to an alarming rate. Due to remarkable risk profile of modern synthetic anti-diabetic, anti-hypertensive, anti-obesity and hypolipidemic agents there is an urgent need to develop eco-friendly, bio-friendly plant based products to replace synthetic chemicals particularly. India has a rich national heritage in the form of plant based remedies. Vrikshamala and Katuki have shown pharmacological therapeutic potentials in the prevention and management of various mental

and physical diseases. It is already mentioned that Ayurveda has listed a number of medicinal plants with their anti-diabetic, anti-obesity and hypolipidemic properties. It is pertinent to mention here that we have long experience based knowledge but we are lacking with evidence based scientific documentation required for global acceptance of these natural products. Recently World Health Organization has provided guidelines for validation of this plant origin.

It possess Cholagogue property, cholorectic, anticholestatic (Shukla B *et al.,* 1991) action which causes excretion of bile and decrease the absorbtion of fat from intestine. It also possess Sheeta veerya which helps in correction of hypertension.

Katuki also has Antioxidant (Russo A et al. 2001 and Ray A et al. 2002), Free Radical Scavenging activity which prevent from cardiovascular disease.

Both trial drug have antioxidant activity. Many studies support that antioxidant improves the insulin sensitivity (Paolisso *et al.,* 1996). Considering the above facts, the above drugs have been selected for the therapeutic and experimental trial in the present study so that this alarming syndrome can be checked from progression.

Keeping in view the above concept, the present research work was carried out at OPD and IPD of Kayachikitsa, Sir Sunderlal Hospital, I.M.S., B.H.U., Varanasi.

OBJECTIVES

- To compile and understand concept of Metabolic Syndrome in Ayurvedic perspective
- To study the effect of the selected trial drugs on the basis of different subjective and objective parameters in Metabolic Syndrome.
- To evaluate the clinical safety for the trial drugs in the management of Metabolic Syndrome.
- To evaluate the effect of trial drug in HFD and dexamethasone induced metabolic syndrome in animal model.

MATERIAL AND METHODS

Following material & methods were adopted for conducting the present clinical trial.

Selection of Metabolic Syndrome patients

Patients were selected from Kayachikitsa OPD/IPD of the S. S. Hospital, IMS, BHU, Varanasi and LD50 evaluation in pairs of Charles foster albino rats and they were obtained from institutional animal house IMS, BHU, Varanasi and the suitable statistical methods were employed to analyze the results.

Patients fulfilling the diagnostic criteria were included in the present study. In total 130 patients were enrolled for the present study, out of which 20 patients discontinued the treatment during trial. So, they were dropped out from the study.

Diagnostic criteria of M. S.

For diagnosis of M.S. patients 2001 NCEP/ATP III Criteria was adopted.

Inclusion criteria for M.S. patients

All patients fulfilling 2001 NCEP/ATP III the criteria to define metabolic syndrome (presence of any three of the following five traits) for M.S were taken.

- Age between: 20-60 years of either sex.
- Waist ≥ 102cm (men), > 88cm (women)
- Hypertension > 130/85 or taking T/t
- Triglycerides ≥ 150 mg/dl or taking T/t
- HDL-cholesterol < 40 mg/dl
- Fasting plasma glucose ≥ 100mg/dl
- Patients having clinical signs and symptoms of Sthaulya.
- Patients willing for trial.

Exclusion criteria for M.S. patients

- Patients who were not willing for trial.
- Patients below the age of 20 years and above 60 years.
- Obese and overweight patients with complicated and chronic disorder like Paralysis, cancer, renal failure, hepatic failure & IHD.
- Patients with genetic and endocrinal disorder.

Details of procedures & methods used in the study

Availability of trial drug: Standardized aqueous extract of trial drugs Vrikshamala *(Garcinia indica)* and Katuki *(Picrorrhiza kurroa)* were purchased from Konark Herbal Pvt.

Ltd., Gujrat (GMP certified well reputed extract provider company) after permission of DRC and Head of the department. After that capsuling of 500mg strength of each drug were done and packed in the bottle.

Clinical study

Total No. of 130 cases were registered and were randomly divided into 4 groups.

- **Group 1 (n=27)** Standardized aqueous extract of Vrikshamala *(Garcinia indica)* 500mg TDS PO. In this group total 32 patients were registered and out of them 27 completed the course.
- **Group 2 (n=27)** Standardized aqueous extract of Katuki*(Picrorrhiza kurroa)* 500mg TDS PO. In this group total 32 patients were registered and out of them 27 completed the course.
- **Group 3 (n=28)** Standardized aqueous extract of Vrikshamala *(Garcinia indica)* 250 mg TDS and Standardized aqueous extract of Katuki *(Picrorrhiza kurroa)* 250 mg TDS PO. In this group total 33 patients were registered and out of them 28 completed the course.
- **Group 4 (n=28)**. To this group, the Pathya Ahar-Vihar was given and out of 33 registered patients 28 completed the course. General scheme of Pathya was advised to the patients. This Pathya varied from patient to patient according to his Age, Agni, Koshtha, Satmya, Abhyavaran Shakti, Jaranshakti. Following chart represents the general scheme of Pathya which was advised to the patients of this group.

Table 1: Specified low caloric diet in Pathya group.

Time	Food stuff	Amount	Weight
7.00 Am.	Warm water	1 Glass	200 ml.
8.00 Am.	Tea (Cow's milk without sugar)	1 Cup	150 ml. (75 ml milk+ 75 m water)
12.00 Pm	Wheat, *jwar* (sorghun), *bazara* (pearl millet), *makka* (maize) flour roti (without oil and ghee)	4 chapati	60 gm.
	Vegetables of bottle guard, brinjal, cabbage, drum-stick, patol (ivy gaurd), ridge guard, spinach, tori, sahajan (drum-stick)	1 Bowl	100 gm.
	Green gram pulses or red gram pulses	1 Bowl	100 gm
	Salad-cabbage, tomato, cucumber, reddish white	1 small plate	25 gm
4.00 Pm.	Coconut water/*Yav sattu/Manda* (gruel)	1 Cup	200 ml
7.00Pm	Mudga yush (green gram water)/ *Takra* (butter milk) (or)	2 Bowl	300 ml.
	Tomato+Spinach soup/ *Takra* (butter milk) (or)	2 Bowl	300 ml.
	Tomato + Drumstick soup/ *Takra* (butter milk)	2 Bowl	300ml
	Wheat flour or barely or maize roti + Vegetable (or)	3 1 Bowl	45 gm. 100 gm.
	Wheat flour+*Bajra* (pearl millet) flour *roti*+Vegetable	3 chapati 1 Bowl	45 gm 100 gm

The calorific value of this diet was estimated. It provides 800-1100 kcal/day with 9g Fibers, 40g fat, 50g protein and 100g carbohydrate.

Energy consuming practices in Pathya groups

Patients were advised to practice regular exercise. Ability to bear the strain of exercise varies from person to person according to their Bala (Strength). Exercise in open air or to continue it after sweating in healthy individuals were discouraged by Ayurveda because of its excessive slimming action therefore, one should practice exercise in open air and after sweating also. Here are some specific energy consuming practices in Pathya group (as per the Ayurvedic texts).

Table 2: Specific energy consuming practices in Pathya group.

Morning	**Jogging or skipping for 30 minutes or Stepping up and down for 30 minutes *Yogasana* for 30 minutes or Fast walking for 1 hour Unction with barley or *Bajra* powder**
Lunch	After lunch walking for 15 minutes
Dinner	After dinner slow walking for 15 minutes

The energy consumed by this practice was estimated. It consumed approximately 100 Kcal/day.

Patients were advised to sleep only 5-6 hours during night and avoid sleep during day time. Patients were totally prohibited not to take sweet and salty items, fried items, fast food, *Chole, Rajma Urad*, meat, milk products, cold drinks, chocolate, alcohol substances, excessive sweetened fruits, dry fruits, curd, pickles, *papad*, potato, sweet-potato, bread, butter, *Paneer*, fermented items etc.

Total duration of study

Total duration of therapy was 3 months with 3 follow ups of 1month each.

Assessment Criteria for Clinical Study

Assessment of effects of the therapy was done on the basis of various subjective and objective criteria For the purpose of assessment, a detailed research Performa incorporating various parameters like Dashvidha pariksha, Ashtavidha pariksha etc. Assessment was done every 30 days during the entire study period. Following subjective and objective criteria were adopted for the purpose of assessment.

a) Subjective parameters
b) Objectives parameters or biophysical parameters
c) Laboratory investigation based parameters

(a) Subjective parameters

As per Ayurvedic classics clinical features of (MS)Sthaulya were considered under subjective parameters and to asses the overall effect of therapies a special scoring method was adopted as follows:-

1. Angachalatva

Grade 0 Absence on movement.

Grade 1 Movement visible on running or fast walking.

Grade 2 Movement visible on normal gait.

Grade 3 Movement also visible even with breathing.

2. Kriccha vyavaya

Grade 0 Normal Sexual performance.

Grade 1 Mild difficulty in performing sexual act due to postural or pathological reasons.

Grade 2 Moderate difficulty in performing sexual act due to postural or pathological reasons.

Grade 3 Severe difficulty in performing sexual act due to both postural and pathological reasons.

3. Atikshudha

Grade 0 Feel hunger, before 1 hour at next Annakala.

Grade 1 Feel hunger, before 2 hour at next Annakala.

Grade 2 Feel hunger, before 4 hour at next Annakala.

Grade 3 Feel hunger, before >4 hour at next Annakala.

4. Nidradhikya

Grade 0 Sleeps 6-8 hrs. at night, without day sleep.

Grade 1 Sleeps 8-10 hrs. at night, without day sleep.

Grade 2 Sleeps 10-12 hrs. at night, without day sleep.

Grade 3 Sleeps > 12 hrs. at night, with day sleep.

5. Kshudraswasa

Grade 0 No swasa.

Grade 1 Swasa on hard work and relieve after 10 min. of rest.

Grade 2 Swasa on light work and relieve after 10 min. of rest.

Grade 3 Swasa on light work and relieve after > 10 min. of rest.

6. Atipipasa

Grade 0 No increasing pipasa.

Grade 1 Up to 1 liter excess intake of water.

Grade 2 Up to 2 liter excess intake of water.

Grade 3 More than 3 liter of water.

7. Angagaurava

Grade 0 No heaviness in body.

Grade 1 Feels heaviness in body but it does not hamper the routine work.

Grade 2 Feels heaviness in body but it hampers the routine work.

Grade 3 Feels heaviness with flabbiness all over the body which causes distress to the person.

8. Daurbalya/Alpavyayam

Grade 0 can do routine exercise.

Grade 1 Can do moderate exercise without difficulty.

Grade 2 Can do mild exercise with very difficult.

Grade 3 cannot do even mild exercise.

9. Daurgandhya

Grade 0 Absence of bad smell in the body.

Grade 1 Persistent bad smell limited to close areas difficult to suppress with deodorants.

Grade 2 Persistent bad smell felt from long distance not suppressed by deodorants.

Grade 3 Persistent bad smell felt from long distance even intolerable to the patient himself.

10. Swedadhikya

(At normal condition at comfortable zone i.e. temperature of 27 deg. C., 65 % humidity)

Grade 0 No sweating.

Grade 1 Sweating after moderate work.

Grade 2 Sweating after normal routine work.

Grade 3 Sweating even in resting condition.

11. Alasya

Grade 0 Normally active.

Grade 1 Hesitate to start work but once started completing it.

Grade 2 Starts work but does not complete it.

Grade 3 Does not have any desire in work, does under compulsion.

The assessment was done before starting the treatment and at each 3 follow ups of 30 days and the improvement was assessed on the basis of percentage relief and statistical evaluations.

(b) Objective or biophysical parameters

Objectives or biophysical parameters were assessed by B.W.(Body Weight), B.M.I. (Basal Metabolic Index), W.C.(Waist circumference), P.R (Pulse Rate), R.R.(Respiratory Rate) and B.P. (Blood Pressure) before trial and at every follow up.

1. Assessment of blood pressure

Blood pressure has been classified into following categories on the basis of Fifth Report of the Joint National Committee on Detection Evaluation and Treatment of High Blood Pressure.

Table 3: Classification of Blood Pressure.

Category	Systolic BP	Diastolic BP
Normal	<130	> 85
High normal	130- 139	85-89
Hypertensive	Average of 2 visit	
Mild	140 – 159	90 – 99
Moderate	160 – 179	100 – 109
Severe	180 – 209	110 – 119
Very severe	> 210	>120

2. Assessment of Basal Metabolic Index (B.M.I.)

Basal metabolic index is calculated by measuring a person weight in kilograms and then dividing by that person's height in meter square.

BMI = Weight (kg) / Height (m^2)

W.H.O. revised obesity criteria was adopted. Internationally accepted range of BMI in adult is shown in below table:

Table 4: Classification of Basal Metabolic Index.

Classification	**BMI(kg/m^2)**	
	Principal cut-off points	**Additional cut-off points**
Underweight	**<18.50**	**<18.50**
Severe thinness	<16.00	<16.00
Moderate thinness	16.00 - 16.99	16.00 - 16.99
Mild thinness	17.00 - 18.49	17.00 - 18.49
Normal range	**18.50 - 24.99**	**18.50 - 22.99**
		23.00 - 24.99
Overweight	**≥25.00**	**≥25.00**
Pre-obese	25.00 - 29.99	25.00 - 27.49
		27.50 - 29.99
Obese	**≥30.00**	**≥30.00**
Obese class I	30.00 - 34.99	30.00 - 32.49
		32.50 - 34.99
Obese class II	35.00 - 39.99	35.00 - 37.49
		37.50 - 39.99
Obese class III	≥40.00	≥40.00

Source: Adapted from WHO, 1995, WHO, 2000 and WHO 2004.

But for India, guidelines were released jointly by the health ministry. The diabetes foundation of India, the All India institute of medical science [AIIMS], Indian council and other health organization.

As per M.O. [Management of obesity]

- Underweight <18.4 kg/m^2
- Normal 18.5-22.9 kg/m^2
- Overweight >23-24.9 kg/m^2
- Obese >25 kg/m^2

Assessment of abdominal obesity

Assessment of Waist circumference: It shows the abdominal adiposity or we can say adhikaya gurutvam. To find Waist circumference, waist is measured at level of umblicus with abdomen relaxed **(Peter. T *et al.*, 2006)** Abdominal adiposity has also been reported among those with BMI >23 kg/m^2. In India abdominal adiposity is significantly more than white Caucasian. According to **IDF*** (International diabetes federation) waist circumference ≥80 cm is risk factor for metabolic disorders.

Laboratory Investigation based parameters:- Standard Laboratory methods were used for the following investigations

- Complete Blood Count (C.B.C.)
- Blood Sugar Fasting (FBS.)
- Blood Sugar Postprandial (PPBS)
- Serum Cholesterols
- Serum Triglycerides (T.G.)
- High Density Lipoproteins (H.D.L.)
- Low Density Lipoproteins (L.D.L.)
- Very Low Density Lipoproteins (V.L.D.L.)
- S. Adiponectin
- S. Leptin

Statistical Analysis

The data collected were transferred on master chart showing various items/variables in columns and subjects in rows. The analysis of data was done using statistical software SPSS version 16.0.

The items on demographic profile and personal characteristics were summarized using univariate and bivariate frequency tables, percentage, graphs and for continuous variables like age, weight, height BMI etc. mean and standard deviation (SD) were also determined.

The formulae for mean and standard deviation are as below:-

$$\text{Mean} = \frac{\bar{x}}{n} = \frac{\text{sum of the observations}}{\text{No.of the observations}}$$

$$\text{Standard Deviation SD} = \sqrt{\frac{\Sigma x^2 - \frac{(\Sigma x)^2}{n}}{n-1}}$$

Intra-group (within the group) comparison

To test the significance of mean of difference of paired observations (BT versus AT) paired t test was applied

$$\text{Paired t} = \frac{\text{Mean of difference}}{\text{SE of difference}}$$

$$\text{SE = Standard Error} = \frac{SD}{\sqrt{n}}$$

n = no. of cases (sample size) and d.f. = degree of freedom = n-1

Inter-group comparison (Between the Group)

In case of more than two independent groups, one-way ANOVA (Analysis of Variance) was applied and value of F test was determined. Wherever F test resulted statistically significant, post–hoc test was applied for multiple comparison, identifying significant pairs of groups. A non-parametric test in such situation applicable was Kruskal-Wallis test.

$$F = \frac{\text{Mean Sum of Square between Group}}{\text{Mean Sum of Square within Group}}$$

E = Expected frequency

Chi-square test

- For nominal and ordinal variables like signs and symptoms (daulbalya, alasya etc.) cross tables showing number and percent before treatment and at different follows according to groups were shown.

- For intra group (at various follow-ups) comparison of ordinal variable and dichotomous variable Friedman chi-square test and Cochran's Q test had been applied respectively. Pearson chi-square test was used for intergroup comparison.
- Pearson's Chi-square = $\sum \frac{(O-E)^2}{E}$

Where, O = observed frequency, E = expected frequency

Wherever expected frequencies came less than 5 Pearson Chi-Square had been calculated after suitably pooling the rows/columns.

- Friedman Chi-square = ([12/nk(k + 1)]*[SUM(T_i^2] - 3n(k + 1)) where, n is the number of subjects, T_i is the sum of ranks for i^{th} follow-up and k is the no. of follow-ups.
- Cochran $Q = (k \times (k - 1) \times \sum_{j=1...k}\{(T_j - Tbar)^2\}) / (k \times \sum_{j=1...k}\{u_i\} - \sum_{j=1...k}\{u_i^2\})$

Where k is the number of samples, T_j is the sum of 1's in the jth column, $Tbar$ is the mean of the T_j's, and u_i is the number of 1's in the ith row. Under the null hypothesis this has an approximate chi-square distribution with (k-1) degrees of freedom.

Statistical Significance

$p < 0.05$ was considered statistically significant and

$p < 0.01$ or $p < 0.001$ as statistically highly significant

$p > 0.05$ as not statistically significant

METHOD OF STUDY

(i) Consent

A written consent was taken from all the patients before inclusion in the trial.

(ii) Performa

A detailed Performa (case history sheet form) was prepared and filled to note down all the details of patients and the disease starting from demographic profile that includes age, sex, religion, occupation etc. to show the pattern and incidence of disease. After recording demographic profile, detailed history was taken, followed by physical and systemic examination. In addition to it, Srotas pariksha, Ashtavidha pariksha and Dashavidha pariksha was carried out and findings related to Nidana, Rupa, Upadrava, Dosha, Dushya were also noted in the proforma. Laboratory investigations done at the time of inclusion of patients in trial were also recorded in the Performa for the sake of comparison of these investigations before, and after treatment.

(iii) Instructions to the patients

- For proper observation and evaluation, the patients were advised to come for follow up after every 30 days or as per requirement.
- Patients were explained possible benefits of the treatment and strictly advised not to discontinue the trial drug till the trial was over.
- They were advised not to take any medication during the trial except most essential drugs.
- Patients were advised to follow all instructions advised to them.

OBSERVATIONS AND RESULTS

In the current study, present series of Metabolic Syndrome (MS), it has been noted that majority of the patients were of 41-50 years of age group (36.92%) followed by 51-60 years of age group (28.46%) reason may that MS increases with the age and mostly patients were females of menopausal age therefore, at this age hormonal imbalances are quite common. In Marital status, 76.92% of them were married. By the time person is middle aged she/he gets married, then after marriage, stress and anxiety increases which may be the triggering factor for MS.

The study of constitutional profile and deha Prakriti reveals that maximum patients (46.15%) were of Kaphavata Prakriti. This indicates that persons with Kapha Vata predominant trait Prakriti are predisposed to MS because due to visham ahar and vihara Kapha and Vata get vitiated. Kapha causes Srotoavrodha, passage of Vata gets obstructed it moves in Kostha, then person feel increased hunger (Atishudha) he overeats leading to Sthaulya. Generally patients having Madhyam Satva were (56.92%), Madyama Sara (60%), Madhyam Samhanana (55.38%), Madhyam Satmya (53.84%).

Observations from clinical study show that most of patient were involved in Madhura(46.9%), Amla (32.3%) and Lavana (19.2%) rasa pradhan diet. These rasas are kaphavardhak which is an etiological factor for Sthaulya. Sweet enriched diet is rich in high calories which causes obesity, diabetes, dyslipidemia. It is also stated in Charak Samhita in Astaninditya purush chapter that Madhur Rasa is cause of MS (Sthaulya).

Overindulgence of the patients in the diet having Payasa vikar (74.6%), Navanna (73.8%), Bhojannotara Jalapana (66.1%), Santarpana (50%), Viruddhahara (50%), Ati-Madhura (51.5%), Adhyasana (44.6%), Guru Ahara (43.1%), Ati-Snigdha (40%), Mamsa (Meat)

(36.9%), Atimatrasana (36.1%), Samshana (33.1%), Madya (30.7%), Vishmashana (22.3%) and Ati-Lavana (19.2%).

The patients selected for trial had complaint of Chala sphika udara stana (83.84%) and those patients were found to be suffering from Angagauravata.

RESULTS OF THERAPEUTIC TRIAL

Table 5: Mean change in Weight in 110 patients of Metabolic Syndrome.

Groups	**BT**	**Fu1**	**Fu2**	**Fu3**	**Within the group comparison Paired 't' test BT vs AT Mean±SD**
Group I (n=27)	80.40±12.73	76.92±12.52	72.55±12.46	67.07±12.72	13.33±4.07 t = 16.99 p < 0.001
Group II (n=27)	77.85±12.28	75.46±12.25	73.18±12.31	71.63±12.16	5.95±1.54 t = 20.07 p < 0.001
Group III (n=28)	77.86±10.85	74.92±10.64	71.64±10.55	68.18±10.42	9.60±1.82 t = 28.09 p < 0.001
Group IV (n=28)	75.76±9.15	74.73±8.98	74.08±9.07	74.04±9.07	1.73±1.04 t = 8.80 p < 0.001
Between the group comparison One Way Annova	F = 0.77 P=0.50	F = 0.21 P =0.88	F = 0.23 P =0.86	F = 2.23 P =0.08	

The above table depicts that mean weight decreased at consecutive follow ups in all four groups and the difference in mean weight at FU3 compared with initial was statistically highly significant in all four groups.

The intergroup comparison of mean weight was not statistically significant initially as well as at every follow up.

Table 6: Mean change in BMI in 110 patients of Metabolic Syndrome.

Groups	BT	Fu1	Fu2	Fu3	Within the group comparison Paired 't' test BT vs AT Mean±SD
Group I (n=27)	33.00±4.56	31.39±4.29	29.63±4.30	27.51±4.49	5.42±1.43 t = 19.69 p < 0.001
Group II (n=27)	31.54±3.24	30.60±3.18	29.68±3.25	29.01±3.15	2.52±0.78 t = 16.67 p < 0.001
Group III (n=28)	32.27±3.61	31.55±3.58	30.17±3.49	28.6±3.61	4.10±0.88 t = 24.63 p < 0.001
Group IV (n=28)	31.74±3.49	31.31±3.42	31.04±3.46	30.9±3.47	00±.44 t = 9.05 p <0.001
Between the group comparison One Way Anova	F = 1.01 P =0.396	F = 0.350 P =0.789	F = 0.350 P =0.789	F = 4.07 P =0.009	

The above table shows that the decrease in mean BMI at III follow up compared to initial was 5.42, 2.52, 4.10 and 00 in Group I, II, III, IV respectively. This decrease in mean BMI was statistically highly significant in all four groups.

The intergroup comparison of mean BMI was not statistically significant initially as well as at every follow up.

Table 7: Mean change in Waist Circumference in 110 patients of Metabolic Syndrome.

Groups	BT	Fu1	Fu2	Fu3	Within the group comparison Paired 't' test BTvsAT Mean±SD
Group I (n=27)	106.14±5.49	102.48±7.31	98.57±5.55	95.29±5.70	10.84±4. 09 t = 13.76 p < 0.001
Group II (n=27)	104.08±7.68	102.48±7.31	100.17±7.06	99.80±7.32	4.20±1.97 t = 11.04 p < 0.001
Group III (n=28)	102.03±8.11	100.17±8.37	98.57±8.18	94.02±8.81	8.01±4.46 t = 9.45 p < 0.001
Group IV (n=28)	99.35±9.13	98.48±9.02	98.50±9.15	98.60±9.07	0.75±1.18 t = 5334 p =0.002
Between the group comparison One Way Anova	F = 3.86 P =0.011	F=1.74 P=0.161	F=0.615 P=0.607	F=3.08 P=0.011	

The above table explains that mean waist circumference decreased at successive follow ups in all four groups and the difference in mean waist circumference at FU3 compared with initial was statistically highly significant excluding Group IV.

The intergroup comparison of mean weight was found not statistically significant initially as well as at every follow up.

Table 8: Mean change in Systolic Blood Pressure in 110 patients of Metabolic Syndrome.

Groups	BT	FU1	FU2	FU3	Within the group comparison Paired 't' test BT vs AT Mean±SD
Group I (n=27)	141.48±12.48	134.15±5.02	127.56±6.03	123.48±7.85	18.00±8.54 t = 10.95 p< 0.001
Group II (n=27)	138.67±10.72	129.41±8.48	122.96±5.77	117.93±5.69	20.74±11.76 t = 9.12 p< 0.001
Group III (n=28)	138.36±11.04	129.64±8.00	122.93±4.91	117.14±4.33	21.21±11.74 t = 9.56 p<0.001
Group IV (n=28)	138.21±9.21	130.57±6.70	123.93±5.92	122.07±5.90	16.14±7.11 t =12.00 p<0.001
Between the group comparison One Way Anova	F=0.735 P=0.533	F=2.53 P=0.61	F=4.04 P=0.009	F=7.163 P<0.001	
Post Hoc Test Sig. pairs of groups (p< 0.05)				(1,2) (1,3)	

The above table shows the decrease mean of SBP when initial compared to FU3 and the mean change was 18.00, 20.74, 21.21 and 16.14 in Group I, II, III, IV respectively. This decrease in mean SBP was statistically highly significant in all groups. The intergroup comparison of mean SBP was also statistically highly significant at FU3. The Post hoc analysis showed the significant pair of groups (1,2) (1,3) at FU3.

Table 9: Mean change in Diastolic Blood Pressure in 110 patients of Metabolic Syndrome.

Groups	BT	FU1	FU2	FU3	Within the group comparison Paired 't' test BT vs AT Mean±SD
Group I (n=27)	95.19±5.92	87.70±4.82	82.44±4.55	79.78±5.41	15.40±6.41 t = 12.47 p<0.001
Group II (n=27)	91.93±6.08	86.37±4.93	80.52±2.26	75.50±4.42	16.22±7.11 t = 11.85 p < 0.001
Group III (n=28)	91.07±5.31	84.86±5.87	81.29±5.31	77.29±4.62	13.78±8.26 t = 11.64 p<0.001
Group IV (n=28)	89.43±3.42	87.50±5.56	84.50±5.37	83.07±5.14	6.35±5.55 t =6.05 p <0.001
Between the group comparison One Way Anozva	F=4.82 P=0.003	F=1.66 P=0.176	F=3.98 P=0.010	F=11.78 P<0.001	
Post Hoc Test Sig. pairs of groups(p< 0.05)	(2,4)			(2,4) (3,4)	

The above table shows decreased mean of DBP when initial compared to FU3 and the mean DBP was 15.40, 16.22, 13.78 and 6.35 in Group I, II, III, IV respectively. This decrease in mean DBP was found statistically highly significant in all four groups.

The intergroup comparison of mean DBP was also statistically highly significant at FU3.

The Post hoc analysis showed the significant pair of groups (2,4) (3,4) at FU3.

Table 10: Mean change in FBS in 110 patients of Metabolic Syndrome.

Groups	BT	AT	Within the group comparison Paired 't' test BT vs AT Mean±SD
Group I (n=27)	104.55±17.59	83.48±7.75	21.07±18.86 t =5.80 p< 0.001
Group II (n=27)	111.33±15.30	82.39±6.60	28.93±19.04 t = 7.89 p < 0.001
Group III (n=28)	105.53±17.42	81.80±7.58	23.37±16.71 t = 3.98 p< 0.001
Group IV (n=28)	100.87±12.36	89.75±12.11	11.12±6.68 t = 8.80 p < 0.001
Between the group comparison One Way Anova	F=2.05 P=0.110	F=4.82 P<0.05	

The above table shows the decreased mean of FBS when initial compared to final treatment was 21.07, 28.93, 23.37 and 11.12 in Group I, II, III, IV respectively This decrease in mean FBS was found statistically highly significant in all four groups.

The intergroup comparison of mean FBS was statistically significant at after the treatment ($p<0.05$).

Table 11: Mean change in FBS in 110 patients of Metabolic Syndrome.

Groups	**BT**	**FU1**	**FU2**	**AT**	**Within the group comparison Paired 't' test BT vs AT Mean±SD**
Group I (n=27)	104.55±17.59	99.19±14.25	90.95±9.08	83.48±7.75	21.07±18.86 t =5.80 p< 0.001
Group II (n=27)	111.33±15.30	104.50±11.70	91.66±9.01	82.39±6.60	28.93±19.04 t = 7.89 p < 0.001
Group III (n=28)	105.53±17.42	100.25±14.85	89.06±11.21	81.80±7.58	23.37±16.71 t = 3.98 p< 0.001
Group IV (n=28)	100.87±12.36	97.56±11.26±	94.93±10.76	89.75±12.11	11.12±6.68 t = 8.80 p < 0.001
Between the group comparison One Way Anova	F=2.05 P=0.110	F=1.39 P=0.249	F=1.64 P=0.183	F=4.82 P<0.05	

The above table shows the decreased mean of FBS when initial compared to final treatment was 21.07, 28.93, 23.37 and 11.12 in Group I, II, III, IV respectively This decrease in mean FBS was found statistically highly significant in all four groups.

The intergroup comparison of mean FBS was statistically significant at after the treatment ($p<0.05$).

Table 12: Mean change in PPBS in 110 patients of Metabolic Syndrome.

Groups	BT	FU1	FU2	FU3	Within the group comparison Paired 't' test BTvsAT Mean±SD
Group I (n=27)	120.41±43.46	107.33±29.24	95.65±15.00	91.29±18.02	29.11±31.15 t =4.85 p< 0.001
Group II (n=27)	121.11±33.93	111.26±30.21	96.22±10.56	90.00±13.55	31.11±27.24 t = 5.93 p < 0.001
Group III (n=28)	147.55±55.45	132.25±43.23	113.20±22.12	110.20±26.83	36.79±46.17 t = 4.21 p< 0.001
Group IV (n=28)	109.86±28.59	104.35±20.5	102.42±10.2	102.89±12.46	6.96±9.11 t = 4.04 p < 0.001
Between the group comparison One Way ANOVA	F=4.01 P=0.009	F=3.910 P=0.007	F=3.88 P=0.001	F=5.59 P<0.001	
Post Hoc Test Sig. pairs of groups (p< 0.05)				(1,3)	

The above table depicts that mean PPBS decreased in all four groups when BT compared with AT and the difference in mean PPBS was statistically highly significant in all four Groups.

The intergroup comparison of mean PPBS was seen statistically significant on final treatment.

The Post hoc analysis reported the significant pair of groups (1,3).

Table 13: Mean change in S. Triglycerides in 110 patients of Metabolic Syndrome.

Groups	BT	FU1	FU2	FU3	Within the group comparison Paired 't' test BT vs AT Mean±SD
Group I (n=27)	185.89±63.24	164.12±49.12	143.72±36.64	130.9±32.52	54.95±42.78 t = 6.77 p < 0.001
Group II (n=27)	179.54±47.78	154.10±35.53	127.96±23.44	109.63±19.16	69.91±44.67 t =7.81 p< 0.001
Group III (n=28)	227.15±75.44	198.51±73.30	171.80±68.71	140.2±59.77	86.92±37.15 t = 12.38 p< 0.001
Group IV (n=28)	155.56±31.26	155.31±31.49	155.16±31.52	155.61±31.56	0.05±2.55 t = 0.11 p =0.912
Between the group comparison One Way Anova	F=7.64 P<0.001	F=4.80 P<0.05	F=5.00 P<0.05	F=6.70 P<0.001	
Post Hoc Test Sig. pairs of groups (p< 0.05)	(3,4)	(1,3)	(1,3)	(1,4)	

The above table depicts that mean S. Triglycerides decreased at successive follow up in all groups except fourth group and the difference in mean S. Triglycerides compared with initial was statistically highly significant in all groups excluding Group IV (p= 0.912).

The intergroup comparison of mean S. Triglycerides was statistically significant before treatment as well as at successive follow ups.

Post Hoc Test for pair wise comparison revealed significant difference group (3,4) at BT and (1,4) (1,3) (1,3)and(1,4) respectively at every follow up.

Table 14: Mean change in S.HDL in 110 patients of Metabolic Syndrome.

Groups	BT	FU1	FU2	FU3	Within the group comparison Paired 't' test BT vs AT Mean±SD
Group I (n=27)	33.88±4.64	37.56±4.01	41.60±4.56	45.24±5.42	11.35±5.45 t =10.81 p< 0.001
Group II (n=27)	33.41±5.27	37.10±3.50	40.76±3.34	44.76±4.76	11.91±5.99 t = 10.32 p < 0.001
Group III (n=28)	34.36±6.53	37.40±4.75	40.20±4.13	42.57±0.78	8.21±6.31 t = 6.88 p< 0.001
Group IV (n=28)	33.37±5.64	34.33±3.04	35.16±3.13	36.35±3.45	2.50±3.09 t = 4.42 p < 0.001
Between the group comparison One Way Anova	F=0.162 P=0.922	F=4.27 P=0.007	F=21.73 P<0.001	F=21.73 P<0.001	
Post hoc test sig. pairs of group (p< 0.05)			(1,4) (2,4) (3,4)	(1,4) (2,4) (3,4)	

The above table shows the increase in mean S.HDL when FU3 compared to initial was 11.35, 10.91, 8.21 and 2.50 in Group I, II, III, IV respectively. This increase in S.HDL was statistically highly significant in all four groups.

The intergroup comparison of mean S.HDL was statistically highly significant at consecutive follow up when compared with initial.

The Post hoc analysis showed the significant pair of groups were (1,4) (2,4) (3,4) in FU2 and FU3.

Table 15: Mean change in S.LDL in 110 patients of Metabolic Syndrome.

Groups	BT	AT	Within the group comparison Paired 't' test BT vs AT Mean±SD
Group I (n=27)	107.49±37.26	100.76±26.96	6.72±15.45 t =2.26 P= 0.032
Group II (n=27)	112.37±26.73	105.25±17.65	7.12±20.76 t = 1.78 P=0.086
Group III (n=28)	109.11±36.76	89.95±21.86	19.15±15.26 t = 3.24 P=0.003
Group IV (n=28)	111.49±31.98	120.32±32.73	2.83±6.27 t = 2.39 P= 0.024
Between the group comparison One Way Anova	F=0.48 P=0.696	F=6.83 P<0.001	
Post Hoc Test Sig. pairs of groups (p< 0.05)		(3,4)	

The above table depicts that mean change in S.LDL BT vs AT in all groups except IV group and the difference in mean AT compared with BT was statistically not significant in all four groups. The intergroup comparison of mean S.LDL was statistically significant after the treatment. The Post hoc analysis showed the significant pair of groups (3,4) on AT.

Table 16: Mean change in S.VLDL in 110 patients of Metabolic Syndrome.

Groups	BT	AT	Within the group comparison Paired 't' test BT vs AT Mean±SD
Group I (n=27)	35.43±8.85	29.05±5.97	6.37±7.67 t =4.32 p< 0.001
Group II (n=27)	35.37±14.18	29.55±5.97	5.82±11.99 t = 10.32 p < 0.001
Group III (n=28)	38.03±14.39	29.48±8.76	8.55±14.81 t = 3.05 p< 0.001
Group IV (n=28)	35.57±13.77	36.86±13.65	1.28±3.06 t = 2.22 p < 0.001
Between the group comparison One Way Anova	F=0.27 P=0.845	F=4.63 P<0.05	

The above table shows that the decrease in mean S.VLDL when BT compared to AT and the mean was 6.37, 5.82, 8.55 and 1.28 in Group I, II, III, IV respectively. This decrease in

S.VLDL was statistically highly significant in I group out of all four groups. The intergroup comparison of mean S.VLDL was statistically significant after the treatment (P<0.05).

Table 17: Mean change in S.Urea and S.Creatinine in 110 patients of Metabolic Syndrome.

Groups	Components	BT	AT	AT~BT	Within the group comparison Paired 't' test BTvsAT Mean±SD
Group I (n=27)	S.Urea	28.26±6.17	27.93±4.78	0.33±8.47	t =0.05, P= 0.840
	S.Creat.	0.68±0.19	0.64±0.17	0.04±0.20	t =1.04, P= 0.305
Group II (n=27)	S.Urea.	0.68±0.19	0.64±0.17	0.04±0.20	t =1.04;P=0.487
	S.Creat.	0.68±0.16	0.63±0.17	0.05±0.19	t = 1.39; P=0.174
Group III (n=28)	S.Urea	27.50±5.71	27.71±4.86	0.64±7.22	t = 0.46;P=0.644
	S.Creat.	0.70±0.17	0.66±0.19	0.04±0.20	t = 1.19;P=0.244
Group IV (n=28)	S.Urea	28.50±7.02	28.21±4.30	0.28±7.52	t = 0.20;P= 0.842
	S.Creat.	0.72±0.16	0.65±0.16	0.06±0.12	t = 2.87;P<0.05

Variables	Between the group comparison One Way Anova	
	BT	AT
S.Urea	F = 0.368, p=0.776	F =0.084, p=0.969
S.Creatinine	F=0.312, p=0.817	F=0.106, p=0.956

The above table reveals that no mean change in S. Urea BT vs AT in all four groups was noted and mean difference of before and after treatment in S.Urea was also found statistically not significant in all four groups.

Similarly the mean of S. Creatinine remained more or less same before and after treatment in all four groups and this mean difference in before and after treatment in S.Creatinine was also not significant statistically in all four groups.

Table 18: Mean change in Hb% and ESR in 110 patients of Metabolic Syndrome.

Group	Components	BT	AT	AT~BT	Within Group comparison Paired 't' test
Group I (n=27	Hb%	12.24±0.61	12.05±0.58	0.19±0.57	t=1.73,P=0.094
	E.S.R.	12.25±1.62	12.82±1.82	0.14±2.39	t=1.00,P=0.750
Group II (n=27)	Hb%	11.28±0.86	11.40±0.68	0.11±0.63	t=0.94,P=0.355
	E.S.R.	11.96±1.74	13.22±1.60	1.29±2.33	t=2.73,P=0.721
Group III (n=28)	Hb%	11.83±0.95	11.95±0.54	0.32±0.69	t=0.24,P=0.809
	E.S.R.	13.18±1.62	12.80±1.82	0.36±2.91	t=0.32,P=0.322
Group IV (n=28)	Hb%	11.85±0.77	11.65±0.61	0.20±0.65	t=0.65,P=0.105
	E.S.R.	13.50±1.63	12.80±1.70	0.69±1.87	t=0.69,P=0.060

Variables	Between the group comparison One Way Anova	
	BT	AT
Hb%	F =6.47, P<0.05	F =5.34, P<0.05
ESR	F =5.91, P<0.05	F =1.07, P=0.364

The above table shows that mean of Hb% remain more or less same in all four groups on AT vs BT and mean difference of AT vs BT was statistically not significant in all four groups.

Similarly the no change in mean of ESR was seen on before and after treatment in all four groups and mean difference in before and after treatment in ESR was not significant statistically in all four groups.

Table 19: Mean change in Liver Function Test in 110 patients of Metabolic Syndrome.

Group	Components	BT	AT	AT~BT	Within Group comparison Paired 't'test
Group I (n=27)	S. Bilirubin	0.74±0.16	0.67±0.10	0.06±0.23	t=1.40,P=0.171
	SGOT	35.70±3.27	30.61±5.33	2.45±3.27	t=3.37,p<0.001
	SGPT	36.25±3.04	33.05±2.05	3.20±2.93	t=4.88,p<0.001
	S. Alk.Phos	62.10±16.17	60.15±15.06	1.95±2.81	t=3.09,p<0.001
Group II (n=27)	S. Bilirubin	0.73±0.21	0.69±0.12	0.02±0.29	t=0.85,P=0.399
	SGOT	35.94±3.01	30.61±5.32	5.33±5.14	t=4.40,p<0.001
	SGPT	30.61±5.64	29.78±5.28	0.83±3.53	t=1.00,p<0.001
	S.Alk.Phos	67.33±21.82	64.44±18.31	2.88±5.58	t=2.19,p<0.001
Group III (n=28)	S.Bilirubin	0.73±0.17	0.69±0.14	0.04±0.26	t=0.85,P=0.399
	SGOT	33.47±6.40	29.17±4.83	4.29±4.68	t=3.78,p<0.001
	SGPT	31.47±5.15	28.59±4.70	2.88±4.63	t=2.68,p<0.001
	S.Alk.Phos.	65.23±18.28	62.94±15.71	2.26±5.53	t=1.78,P=0.086
Group IV (n=28)	S.Bilirubin	0.68±0.17	0.69±0.13	0.00±0.25	t=0.075,P=0.94
	SGOT	31.20±7.10	27.45±5.96	3.75±5.38	t=3.11,P=0.016
	SGPT	31.50±5.81	30.00±5.09	1.50±2.03	t=3.29,P=0.171
	S.Alk.Phos.	65.21±18.28	62.94±15.72	2.26±5.53	t=1.78,P=0.086

Variables	Between the group comparison One Way Annova	
	BT	AT
S. Bilirubin	F =0.39, P=0.75	F =0.11, P=0.95
SGOT	F=0.69, P=0.61	F=0.63, P=0.66
SGPT	F=1.75, P=0.146	F=1.93, P=0.12
S. Alk. Phos.	F=0.41, P=0.81	F=0.41, P=0.83

The above table shows that decreased mean of SGOT, SGPT in all four groups on AT vs BT and mean difference of AT vs BT was statistically significant in all four groups.

The change in mean of S.Bilirubin, S.Alk.Phos. remained more or less same at before and after treatment in all four groups. On between the group comparison before and after treatment in S.Bilirubin, S.Alk.Phos, SGOT, SGPT P value was not also found statistically significant in all four groups.

Table 20: Mean change in S. Adiponectin in 110 patients of Metabolic Syndrome.

Groups	BT	AT	Within the group comparison Paired 't' test BT vs AT Mean±SD
Group I (n=27)	31.46±0.92	45.11±0.61	1.34±0.84 t = 8.23 p< 0.001
Group II (n=27)	36.72±0.99	45.48±0.51	8.75±0.94 t = 4.81 p < 0.001
Group III (n=28)	34.46±0.95	44.30±0.72	9.83±4.02 t = 4.03 p<0.001
Group IV (n=28)	37.30±1.10	37.84±0.99	5.35±.35 t =0.79 P= 0.433
Between the group comparison One Way Anova	F=1.770 P=0.157	F=6.615 P<0.001	

The above table depicts that mean S. Adiponectin increased at final treatment in all four groups and the difference in mean S. Adiponectin final compared with initial was statistically highly significant in I, II, III, group excluding IV Group(p>0.433).

The intergroup comparison of mean S. Adiponectin was statistically significant after the treatment.

Table 21: Mean change in S. Leptin in 110 patients of Metabolic Syndrome.

Groups	BT	AT	Within the group comparison Paired 't' test BT vs AT Mean±SD
Group I (n=27)	22.48±3.53	12.31±3.19	11.72±4.68 t = 13.01 p<0.001
Group II (n=27)	23.22±3.18	12.77±4.79	10.45±5.97 t = 9.09 p < 0.001
Group III (n=28)	20.94±4.78	13.25±3.69	7.69±6..72 t = 6.05 p<0.001
Group IV (n=28)	22.60±2.81	22.67±2.80	0.00±0.52 t =2.58 P=0.016
Between the group comparison One Way ANOVA	F=1.94 P=0.127	F=50.13 P=0.000	
Post hoc test sig. pairs of group (p< 0.05)		(1,4) (2,4) (3,4)	

The above table shows that the decrease in mean S. Leptin at III follow up compared to initial was 11.72, 10.45, 7.69 and 00 in Group I, II, III, IV respectively. This decrease of mean in S.Leptin was statistically highly significant in all groups except group IV.

The intergroup comparison of mean S.Leptin was statistically highly significant after the treatment as compare to before treatment.

The Post hoc analysis revealed the significant pair of groups (1, 4) (2, 4) (3, 4).

Table 22: Based on presence /absence of 10 symptoms before treatment and FU3; the No. of symptoms reduced at FU3 is given in following table according to group.

No.of Symptoms absent after treatment	GROUPS			
0	2(7.4%)	0(0%)	0(0%)	10(35.7%)
1-3	11(40.7%)	3(11.1%)	5(17.9%)	15(33.6%)
4-6	11(40.7%)	12(44.4%)	17(60.7%)	3(10.7%)
7-9	3(11.1%)	12(44.4%)	6(21.4%)	0(0%)
10	0(0%)	0(0%)	0(0%)	0(0%)
Pearson χ^2 test	χ^2=57.33; p<0.001			

Assuming the seriousness of all the 10 symptoms are equal then the presence and absence of symptom were added and p<0.001 value found was statistically highly significant.

DISCUSSION

As per the descriptions available in Ayurvedic classics, therapeutic effect of drug depends on certain physiochemical properties in that substance. According to Ayurveda, five such physiochemical property categories, have been elucidated namely, Rasa, Guna, Virya, Vipaka and Prabhava. The drugs having these properties act on agni or dosha or dushya or on both of them or Srotas which demolish the pathogenesis (Samprapti Vightana).

Most of the drugs which have mentioned in the Texts for Sthaulya, have Katu, Tikta and Kashaya rasas. These Rasas have the tendency of reducing Kapha and Medas. Katu rasa removes the obstruction of channels and normalizes the flow (Srothovivarana, Kaphahara (A.H.Su.10/17-18). Katu rasa also possess Lekhana guna (Ch.Su.26/43) that scraps out excessive Kapha and Meda from Srotas. Both trial drugs also have got Katu and Tikta Rasa which does this action.

In addition to Lekhana, Ruksha Guna of Vrikshamala and Katuki also has the property of Soshana (Ch.Su.26/44), which absorbs the excessive fluids and lipid substances which lead to

hypercholesterolaemia. So, it is apparent that by virtue of their property, these drugs act as Kaphahar, Medohara which helps in management of obesity, diabetes, hyperlipidemia the components of MS.

Lagu Guna acts as Kaphahara, reduces the tissue weights (Langana) and clears the channels of the body (Srothoshodhana) which helps in prevention of atherosclerosis which further helps in prevention of hypertension.

With regard to Virya, Vrikshamala has Ushna Virya. By its sheer energetic processes, they oppose any increment of Kapha and Medas. In recent times, Upadhyay, *et al.*, in 1979 have showed that substances having Usna Virya are accountable for the incensement of basal metabolic rate, oxygen consumption and accelerated breakdown of fat at mitochondrial level. All these actions are very much beneficial in obesity, hyperlipidemia while Katuki has Sheeta veerya acts against the vitiated Pitta hence Rakta eventually helps in lowering down of Blood Pressure.

Related to Vipaka, Vrikshamala has Amla Vipaka which is said to be hridya, and Amla Vipaka is Laghu in Guna this property works on obesity. Katuki has Katu Vipaka which enhances the Jatharagni and Dhatvagni and normalize the metabolic process. It also helps to reduce the Kapha and Medas and correct the vitiated medodhatvagni.

In Sthaulya derangement of Agni is seen. Mainly Jatharagni is increased but Medodhatvagni is diminished, so it causes excessive accumulation of Meda in body & results in Ati Sthaulya. Both the trial drugs are Rochana, Deepana, Pachana therefore corrects the deranged Agni at different levels.

Maximum patients complained of Angauravta, Angachalatva, Atikshuda, Alasya, Daurbalya, Kshudrashwasa, Krichavyavaya, Nidradhikya, and Swedadhikya these were the main symptoms of Sthaulya as per classics. Decrease in these symptoms have seen more in group I, then in group III, then in group II and lastly in group IV.

Considering the most important component of MS i.e. 92.30% patient reported to had increased body weight in between 75-90Kg and regarding BMI, most patients 66.2% reported were of class I Obesity who fall into high risk group because maximum patients visit hospitals when conditions become beyond tolerance and as maximum patients were female and they have habit of tolerating the things this might be the cause. Drug therapy showed

highly significant reduction in body weight, waist circumferences and BMI in Group I, then in Group III then in II and in IV Group. Result of trial drug was due to the fact that Vrikshamala has been reported as antiobesity agent, inhibit lipid oxidation, de novo lipogenesis whereas Katuki acts on hyperlpidemia, by the virtue of its cholorectic and anticholestasis property, both the trial drug ultimately help in lessening of the weight, waist circumference, BMI.

Hypertension is the one of the diagnostic component of MS. Trial drug also has reported significant fall in systolic BP and diastolic BP in all groups. Statistically group I, II and III showed more or less same result and group IV also showed good result. Fasting blood sugar is also one of the prime constituent of MS. In this series maximum result was seen in the Group I then in group III then group II and lastly in Group IV and in PPBS all group showed the equal result.

Lipid profile is also an important biochemical investigation for obesity, HTN, diabetes. Out of this S. Triglyceries and S.LDL are important component of MS. Food is the main source of triglycerides. Firstly, decrease food intake and decrease fat absorption produces decrease in triglycerides and/or increased excretion of cholesterol via bile and sweat. Secondly, the trial drug by virtue of their Kapha and Medohara properties, don't allow Meda to be deposited in the body and also scrap out the excess fat from the body. Thus result is low TG and rise of S.HDL level. In order of group II & III then group I significant decline in S.TG level was seen. Significant increase in S.HDL was seen in I&II, and then in group III then in group IV. During trial study significant decline in level of S.LDL, S.VLDL and total cholesterol was also observed in order of group III then in group I and II. Group IV has also showed significant result in this series.

In biochemical parameters, mean increase in S.Adiponectin was more in group I then in group III then in II and decrease in S.Leptin has been observed and effect was more in group I then in group II and then in group III and no effect on level of S.Adiponectin and S.Leptin was noticed in group IV.

In other haematological profile like in Hb%, ESR, LFT, B.Urea, S.Creat. no change in their values were noted with the trial drug in all four groups.

Probable mechanism of action of trial drugs

Probable mode of action of Vrikshamala *(Garcinia indica)*

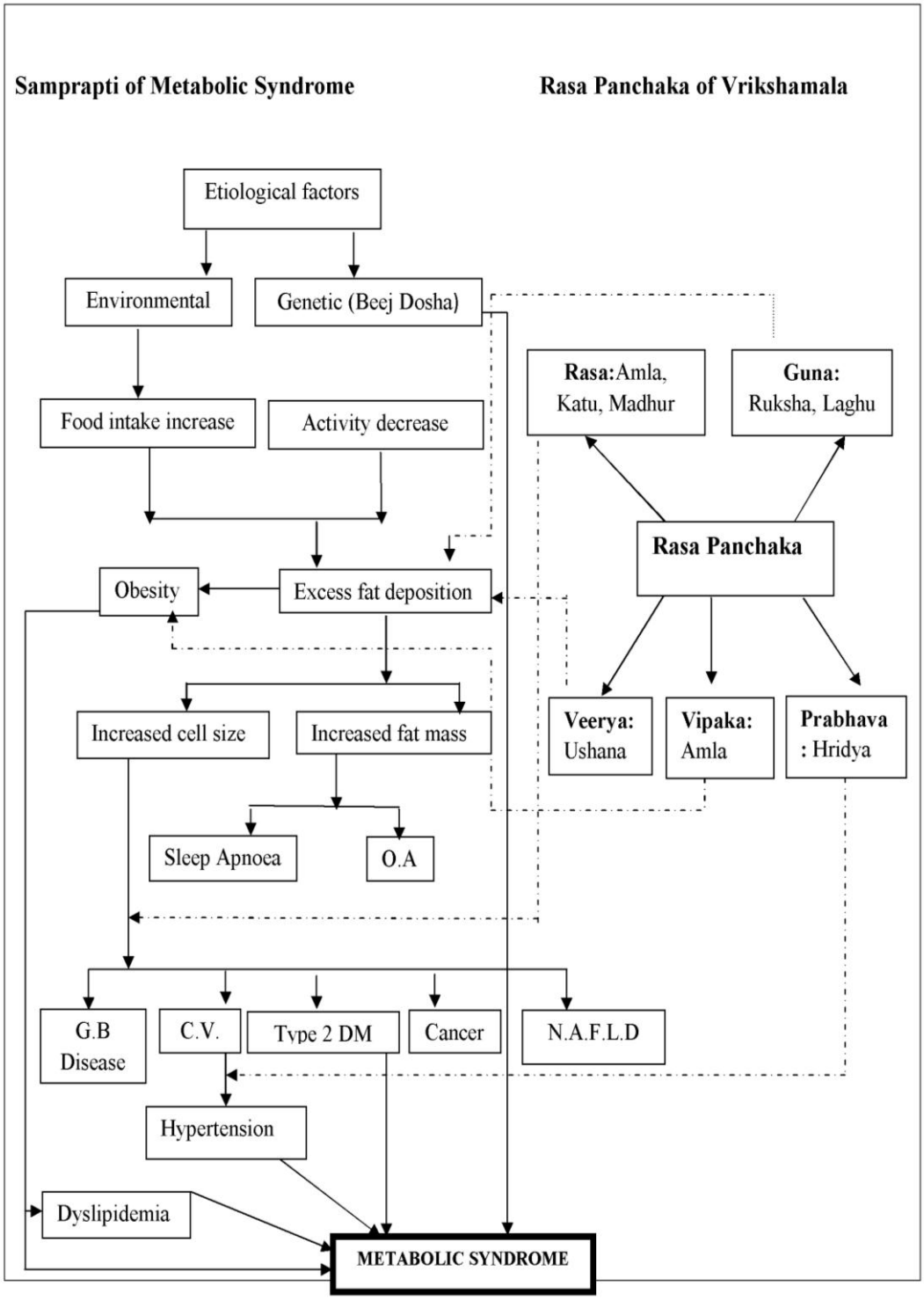

Figure 1: Samprapti Vighatana through Rasa Panchaka of Vrikshamala.

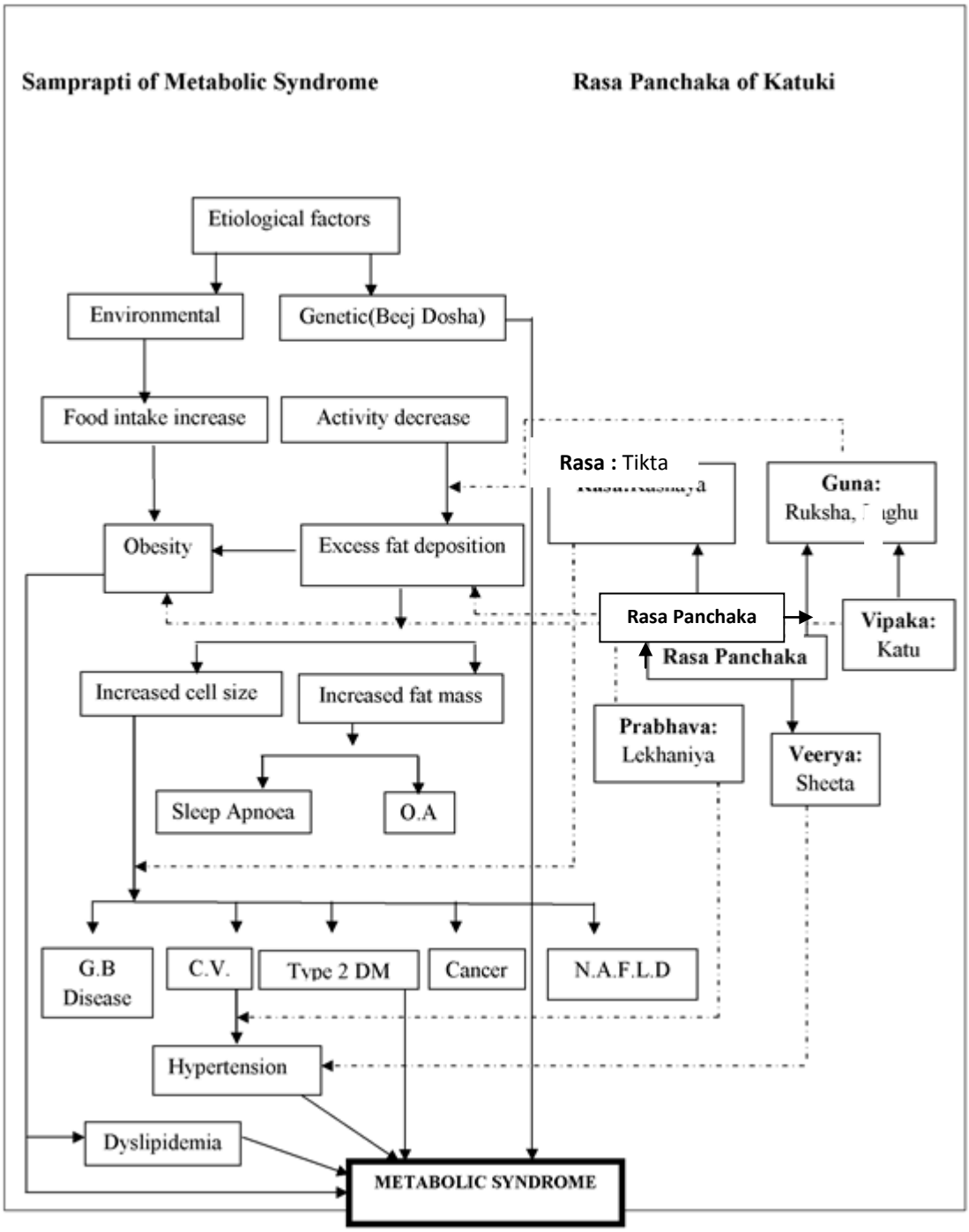

Figure 2: Samprapti Vighatana through Rasa Panchaka of Katuki Probable mode of action of Katuki *(Picrorrhiz a kurroa).*

Since unhealthy eating habits combined with limited activity are a major contributor to obesity and its related metabolic syndrome. It is clear that the management of Metabolic Syndrome is to treat individual component. Ayurveda gives more importance for the prevention of the disease rather than cure The evidence from various previous works show that the patient of metabolic syndrome usually suffer from cardiac illness in the latter stage. Hypertension is considered as a part of the disease cluster. As per Ayurvedic view, Tri-

marmas are affected from the very beginning to end stage of the disease (death of the patient). Keeping this view in mind, drugs like Vrikshamala and Katuka were selected. The main aim of the study was to correct deranged Medaagni with drugs having effect on meda either by its lekhana karma or to recover derranged metabolic chain either by channeling the Agni (Medagni) or clearing the channel of Medovaha srotas from any type of obstruction. In case of M.S. derangement of the Agni is seen in different level and is obviously enhanced at the Jatharagni level. On the other hand at the level of Dhathavagni, the diminished Agni of the Medagni leads to improper digestion of meda which causes only formation of meda dhatu. So aim is to correct the diminished agni to control diabetes, obesity.

When calorie intake exceeds the energy expenditure then fat gets deposited in the body which results in obesity, Guna present in Vrikshamala viz Ruksha and Laghu and its Usna veerya property inhibits the fat deposition and melts the deposited fat leading to weight loss and therefore helps in improvement of hyperglycemia and O.A, sleep apnoea like associated condition.

Vrikshamala also has got amla vipaka which is laghu in property as stated by Acharya (Ch.Su. 26/62) (Su.Su. 40) in texts. Ruksha and laghu guna have scraping property which furthermore, pacify the obstructed channel of Medovaha Srotas, prevents from dyslipidemia and from metabolic syndrome. Its Yakruttejaka property also helps in correction of dyslipidemia.

Hridya prabhava and Kapha-Vata shamak (Ch.Su. 27) property of Vrikshamala reduce the atherosclerosis also helps in relieving HTN which may progress to cardiovascular disease in course of time.

Its katu rasa property scrapes out the excess meda dhatu which further helps in reduction of cell size and further inhibit the diseases like GB disease, CVD, Type 2 diabetes, NAFLD later checks the disease like HTN therefore prevent the person to suffer from metabolic syndrome.

As Vrikshamala is katu and amla in rasa and amla in vipaka and usna in veerya all these properties corrects the vitiated agni at different levels, when agni gets corrected proper formation of further dhatus occurs instead of only meda dhatu we can say which is the main reason of MS.

HCA present in G.indica limits the initial steps of fatty acid and cholesterol biosynthesis during carbohydrate feeding. A more recent approach for determining fat metabolism by HCA was conducted by measuring urinary concentration of malondialdehyde (MDA), acetaldehyde (ACT), formaldehyde (FA), and acetone (ACON) of the tested subjects. The urinary excretion of these four metabolites was proposed to be a consequence of enhanced *β*-oxidation of fats in body tissues (H.G. Preuss *et al.*, 2004).

More recently, neuropeptide Y (NPY) had also been implicated in the appetite suppression of HCA. Preuss *et al.*, 2004, reported that HCA caused a significant reduction in appetite, weight loss, and plasma leptin level, concomitant with an increase in the serum serotonin level and a favorable lipid profile in human clinical trials. Similar results were also obtained in a study conducted by Asghar *et al.*, In 2006. Garcinol was also shown to up-regulated the gene expression of adiponectin as well as down-regulated the gene expressions of leptin and FAS, indicative of its anti-adipogenic effects for treating obesity-related conditions (Hsu. C. L., *et al.*, 2012).

In hyperglycemia, auto-oxidation of glucose increases the formation of free radicals beyond the capacity of defense system to neutralize it and cause oxidative stress (Kaneto H *et al.*, 2005). Garcinol which act as strong antioxidants check the oxidative stress. These strong antioxidants also helps in preventing the depletion of GSH under stress conditions. (Yamaguchi *et al.*, 2000). Many studies support that antioxidant improves the insulin sensitivity (Paolisso *et al.*, 1996). Isolated (-)-hydroxycitric acid has been reported to posses regulatory effect on carbohydrate and lipid metabolism (Jena BS *et al.*, 2002). It limits the availability of acetyl-CoA required for fatty acid synthesis and promotes glycogenesis, β-oxidation, etc. These metabolic pathways initiate the uptake of glucose by the muscle and thereby combat insulin resistance of type 2 diabetes (Grover JK *et al.*, 2002).

It has been reported that isomer of Garcinia (2*S*, 3*R*)-HCA inhibits pancreatic α-amylase and intestinal α-glucosidase, leading to a reduction in carbohydrate metabolism (Yamada T *et al.*, 2007).

Katuki is Tikta in Rasa, Ruksh and Laghu in Guna, Katu in Vipaka and lekhaniya in Prabhava all these properties present in the trial drug prevent the excess fat accumulation in body and again helps in scraping out of excess fat, decrease the cell mass and cell size hence improve and check the condition like HTN, CVD, gall bladder disease, sleep apnoea, type 2

diabetes. It can pacify the obstructed channel of Medovaha Srotas and might correct the atherosclerosis which progress to HTN at advance stage. Katu rasa effect scraps out the Kleda, Meda, Vasa therefore, prevent from obesity, DM and CVD. Especially sheeta veerya property of Katuki helps in correction of HTN and Laghu, Ruksha Guna by sheer of their property increase the Vata and further helps in vasodilation which result in lower down in BP. Katuki can also correct the derranged Agni with the help of Katu Vipaka property.

The liver synthesizes the various lipoproteins involved in transporting cholesterol and lipids throughout the body. When excess fatty food intake increases, cholesterol in body increases which get oxidized by the liver into a variety of bile acids, along with cholesterol itself, is excreted from the ***liver*** into the ***bile*** and reabsorbed from the intestines, Katuki possess cholagogue, cholerectic and anticholestatic drug (Shukla B *et al.*, 1991). These properties stimulate the bile secretion and excretion hence decrease the absorption of fat from intestine, inhibits from dyslipidemia, insulin resistance, obesity.

Katuki is the well-known hepatoprotective drug. Its extract has been reported to reverse the increased AST and ALT activities towards near normalcy (Lee HS *et al.*, 2006) which suggests prevention of cellular and tissue damages under diabetic conditions. Therefore, hepatoprotective activity of Katuki extract may be partially responsible for the observed antidiabetic activity. This plant has been shown to be hydrocholerectic in a biliary fistula model in dogs and humans (Pandey VN *et al.*, 1970).

Acharya BhavaPrakash (Haritkyadi Varga 6/152) has mentioned the katuki in Prameha. Currently various research has been done on Picrorhiza kurroa as hypoglycaemic plant. It controls the release of glycogen from liver thereby decreasing the insulin load. Liver is store house of glucose. Picrorhiza kurroa regulates the release of glucose from liver by controlling glycogenesis and glycogenolysis thereby reducing the requirement of insulin. It has been shown in various researches that its extract showed considerable improvement in β-cell density, the islet size was also larger. This may be an indication of the regeneration and rejuvenation of β-cells leading to increase in insulin production and secretion (Shivkumar Chauhan *et al.*, 2008).

Therefore, we can conclude that both the trial drug have the potential to correct all the components of metabolic syndrome.

CONCLUSION

Current study concludes that The trial drug are very useful for reduction of symptoms like angagauravta, Atishudha, Atipipasa, Kshudraswasa, Angachalatva, Swedadhikya, Alasya, Gatrasada and Nidraadhikya which were the chief complaints of the patients. The Vrikshamala group I showed the marked improvement in weight loss, lightness of body, blood sugar level, knee joints pain, and ankle joints pain, HTN. Katuki group II showed the significant results in improving TG, Cholesterol level and the HDL level. The trial drugs in this study have potential to act on components of metabolic syndrome i.e. HTN, dyslipidemia, hyperglycemia. The no unwanted effect with the trial drug was observed. Thus, highly encouraging results were found in all trial groups in patients who completed the study successfully.

Thus, we sincerely hope that the present study would be pioneer as an ideal research work in the field of Metabolic Syndrome and would provide useful lead for coming generations and future research workers and future recommendation are to conduct the clinical trial on large samples since the drugs are clinically safe and no side effect have been observed.

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