ORIGINAL ARTICLE

Comparative Study on the Effect of Vidangadya Churna and Lekhana Basti in Medodushti with Special Reference to Dyslipidaemia

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ABSTRACT:

Introduction- Cardiovascular diseases (CVDs) are the major causes of morbidity and mortality in our society, with dyslipidemia contributing significantly through atherosclerosis. All components of Dyslipidaemia get the most attention because of the link between plasma lipids & CVD, as it is a major risk factor for fatal diseases such as coronary artery disease (CAD). Sign & symptoms of Medodushti described in Ayurveda shows strikingly resemblance with Dyslipidaemia explained in contemporary medicine. Thus, while treating the Medodushti, selection of Dravya should have criteria that help in Lekhana of excessive Meda-Kapha without Vayu-Pra kopā & normalising the Agni both at the level of Jatharagni and Dhatwagni.

Aims and objectives- Present study was under taken to assess the efficacy of Lekhana Basti and Vidangadya Churna in the management of Dyslipidemia.

Material and methods- This study was carried out on 30 clinically diagnosed & investigated individuals of Dyslipidaemia. Patients were selected from the OPD & IPD of hospital of National Institute of Ayurveda Jaipur. Patients were randomly divided into two groups i.e. group A and group B having 15 patients each.

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Patients of group A were administered Vidangadya Churna (capsules) in dose of 3g/day with lukewarm water while patients of group B were given Vidangadya Churna (capsules) in dose of 3g/day with lukewarm water along with 15 Lekhana Basti.

INTRODUCTION:

Cardiovascular diseases (CVDs) have become the leading causes of death in India. This is largely because of the increase in the prevalence of CVDs and CVD risk factors in India. Almost 25% deaths in India are now because of CVDs with ischemic heart disease and stroke responsible for >80% of this burden. A more worrying fact is that the incidences of CVDs have gone up significantly for people between the age 25 and 69 to 24.8%, which means we are losing more productive people to these diseases. Excess calories in the body are converted into triglycerides and stored in fat cells throughout the body. Low-density lipoprotein (LDL) cholesterol is deposited in the walls of arteries and causes atherosclerosis. In general, lower LDL cholesterol numbers are better for vascular health. High-density lipoprotein (HDL) cholesterol protects against vascular disease by removing the “bad” cholesterol out of the walls of arteries. Total blood cholesterol is a measure of LDL cholesterol, HDL cholesterol and other lipid components. High triglycerides increase the risk of atherosclerotic CVD. Raised blood cholesterol increases the risk of heart disease and stroke. Globally, one third of ischaemic heart disease is attributable to high cholesterol. Case-control studies have reported that there is significant association of acute coronary events with raised apolipoprotein B, total cholesterol, LDL cholesterol and non-HDL cholesterol and inverse association with high apolipoprotein A and HDL cholesterol. To lower CVD mortality, aggressive and comprehensive management of its risk factors, including dyslipidemia, hypertension, diabetes mellitus, and smoking are crucial.

Medodushhti particularly medovridhdi occurs due to excessive indulgence of Kapha vadhakaahara and vihara e.g. sneha dravya; ghrita, taila, vasa, majja, milk products, sugar, jiggerly, tila, junk food, day time sleep, sedentary lifestyle etc. This causes rasa shatu vriddhi leading to kaphavridhdi and other dhatuvridhdi specially medovridhdi and medodushhti due to its resemblance with sneha dravya. Due the kapha vridhi srotus of other dhatu are obstructed and only medovridhi takes place along with poor nourishment of other dhatu. This is also responsible for avarana of samana vayu causing agni sadhukshana resulting in more eating. Excessive intake of ahara again produces samarasadhatu which further complicate the situation. Thus a various cycle is established which needs to be broken by the therapeutic intervention. While treating the Medodushhti, selection of Dravya should have criteria that help in Lekhana of excessive Meda-Kapha without Vayu-Prakopa & normalising the Agni both at the level of Jatharagni & Dhatwagni. Keeping these facts in mind Vidangadya Churna and Lekhana Basti were selected as therapeutic measures for the present clinical trial.

Aims and Objectives

Present Research work was undertaken with the following objectives-

- To evaluate clinical efficacy of Vidangadya Churna and Lekhanabasti in the management of Medodushhti (Dyslipidaemia).
- To compare the clinical efficacy of Vidangadya Churna and Lekhan Basti in management of Medodushhti (Dyslipidaemia) using various scientific parameters.

Materials and methods

The study was conducted on 30 clinically diagnosed & confirmed patients of Medodushhti and Dyslipidaemia on the basis of subjective & objective parameters. Patients were randomly selected from OPD and IPD of Arogyashala, P.G. Department of Kaychikitsa NIA, Jaipur. A regular record of assessment of all patients was maintained according to proforma prepared for the purpose.

A) Inclusion criteria:

a) Diagnosed & confirmed cases of Dyslipidaemia and Medo dushtion the basis of investigation.

b) Patients between the age group of 20-60 years in either sex.

c) Patients willing to sign the consent form.
d) Patients willing to undergo the process of Basti therapy.

B) Diagnostic Criteria:

Patients were diagnosed on the basis of laboratory investigations; mainly Lipid profile. Patients having alterations in any one or more component of the lipid profile as follows were included in present study-

- Serum cholesterol (200 mg/dl or more),
- Serum triglycerides (150mg/dl or more)
- Serum LDL (130 mg/dl or more)
- Serum VLDL (40 mg/dl or more)
- Serum HDL (40 mg/dl or less) were taken for the study.

C) Exclusion criteria:

Vidangadya Churna

Table No. 01: Showing the Contents of Vidangadya Churna

<table>
<thead>
<tr>
<th>Sl. No.</th>
<th>Name of drug</th>
<th>Latin name</th>
<th>Proportion</th>
<th>Part used</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Vidanga</td>
<td>Embeliaaribes</td>
<td>1 Part</td>
<td>Fruit</td>
</tr>
<tr>
<td>2.</td>
<td>Shunthi</td>
<td>Zingiber officinalis</td>
<td>1 Part</td>
<td>Rhizome</td>
</tr>
<tr>
<td>3.</td>
<td>Yava</td>
<td>Horskeum Vulgare</td>
<td>1 Part</td>
<td>Seed</td>
</tr>
<tr>
<td>4.</td>
<td>Amalaki</td>
<td>Emblica officinalis</td>
<td>1 Part</td>
<td>Fruit</td>
</tr>
<tr>
<td>5.</td>
<td>Yavakshara</td>
<td>-</td>
<td>1 Part</td>
<td>-</td>
</tr>
<tr>
<td>6.</td>
<td>Lauhabhasma</td>
<td>-</td>
<td>1 Part</td>
<td>-</td>
</tr>
<tr>
<td>7.</td>
<td>Madhu</td>
<td>-</td>
<td>1 Part</td>
<td>-</td>
</tr>
</tbody>
</table>

Method of Preparation: All the above Dravya-Vidanga, Shunthi, Amalaki, Yava, Yavakshara and Madhu were taken in equal proportion. First of all, initial 4 Dravya were powdered and added with sl. no. 5 and 6, then were mixed thoroughly & at last Madhu was mixed in it. The churna was formulated in the form of capsules; each of 500 mg. This formulation was prepared in Rasashala, i.e. pharmacy of National Institute of Ayurveda, Jaipur.

2. Lekhana Basti:-

Table No. -02: Showing the contents of the Lekhana Basti

<table>
<thead>
<tr>
<th>Sl. No.</th>
<th>DRUGS</th>
<th>ENGLISH/ LATIN NAME</th>
<th>PARTS USED</th>
<th>QUANTITY</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Saindhava</td>
<td>Sodium Chloride</td>
<td>8-10gms</td>
<td></td>
</tr>
</tbody>
</table>
### Method of Preparation of Basti Dravya:

*Madhu & Saindhava* are taken in above proportion and mixed thoroughly with *Yava kshara, Hingua, Shilajit* in fixed proportion then *Sneha Dravya* i.e. *Dashmoolataila* is mixed & stirred to make uniform mixture. As a *Kwath Dravya*, *Triphala kwath* is added and at last *Gomutra* is added in required quantity.

**Anuvasana Basti:** *Dashmoola Taila* was used for *Anuvasana Basti*. One *Anuvasana Basti* was administered on 1st day & then on every 4th day i.e. with the interval of 3 days of consecutive *Lekhana Basti*.

**Dose:** 60-80 ml of *Dashmoola Taila*.

### Study Design

Randomized, Control, Open, Interventional, Clinical study.

### Follow-Up Study

- Follow up of the patient was done fortnightly for a period of 30 days.
- Improvement in the symptoms, if any & other effects were noted down.
- Laboratory investigations were repeated after completion of treatment i.e. after 30 days.

### Criteria for assessment

Both subjective & objective parameters were employed for assessment of the impact of the treatment.

#### a) Subjective Criteria

The details of score adopted for the main signs and symptoms in present study were as follows:

**Assessment of Subjective parameters**

- **Assessment of Kshudhadhikya**
  0 – Becomes hungry after about 6hrs
  1 – Becomes hungry after about 4-5 hrs
  2 – Becomes hungry after about 3hrs
  3 – Becomes hungry after about 2-3hrs
  4 – Becomes hungry after about 2hrs

- **Assessment of Pipasadhikya**
  0 – Drinks about 8-10 glass of water daily
  1 – Drinks about 10-15 glass of water daily
  2 - Drinks about 15-20 glass of water daily
  3 - Drinks about 20-25 glass of water daily
  4 – unable to have a sound sleep for his thirst

- **Assessment of Kshudrashwas**
  0 – No shortness of breath
  1 – Mild dyspnoea after physical exertion relieved on rest
  2 – Moderate dyspnoea after physical exertion
  3 – Dyspnoea even after daily routine
  4 – Breathlessness even after changing posture
• **Assessment of Swedadhikya**
  0 – Normal perspiration
  1 – Mild perspiration after doing exertion
  2 – Increased perspiration after doing little exertion
  3 – Profuse perspiration after doing little exertion
  4 – Perspiration without exertion

• **Assessment of Atinidra**
  0 – 6-8 hours/day sleep
  1 – 8-10 hours/day sleep
  2 – 10-12 hours/day sleep
  3 – 12-14 hours/day sleep
  4 – >14 hours/day sleep

• **Assessment of Daurbalya**
  0 – Feeling of well being
  1 – Tired after doing strenuous physical activity
  2 – Tired after doing moderate physical activity but can perform daily activity
  3 – Perform daily activity with difficulty
  4 – Extremely tired to carry out daily routine activity

• **Assessment of Alasya**
  0 - Normally active
  1 - Hesitate to start work but once started complete it
  2 - Start work but does not complete it
  3 – Does not have desire, works under compulsion
  4 – Does not start work

• **Assessment of Angasada**
  0 - Absent
  1 - Occasional *angsada*
  2 - Continuous *angsada* but not interfere any activity
  3 - Continuous *angsada* and sometimes interfere daily activity
  4 - Continuous *angsada* which hampers daily activity and confined patient to complete rest

• **Assessment of Krichravyavayya**
  0 - Absent
  1 - Mild loss of libido
  2 - Moderate loss of libido
  3 - Severe loss of libido
  4 - Complete loss of libido

• **Assessment of Hriddrava**
  0 - No palpitation
  1 - Mild palpitation after physical/mental exertion
  2 - Palpitation after physical exertion
  3 - Palpitation even at rest
  4 - Palpitation present every time

• **Assessment of Krathana**
  0 - No snoring
  1 - Occasional snoring
  2 - Snoring in some parts of sleep
  3 - Snoring all the time of sleep
  4 - Interrupted sleep due to snoring and snoring accompanied by apnoea

• **Assessment of Gaurava**
  0 - No feeling of heaviness
  1 - Occasional feeling of heaviness
  2 - Continuous feeling of heaviness but patient does usual work
  3 - Continuous feeling of heaviness which hampers usual work
  4 - Unable to do any work due to heaviness

The assessment was done before starting the treatment and after 30 days of treatment i.e. at the completion of the treatment and the improvement was assessed on the basis of statistical analysis.

b) **Objective Criteria:**
   1) **Anthropometric Assessment:**
   • Weight of the Patient (in Kg)
   • B.M.I.-
   • Chest Circumference

2) **Biochemical parameter assessment:**
   • Routine Blood Investigation i.e. Hb%, TLC, DLC, ESR, FBS, Sr. Urea, Sr. Creatinine, SGOT, SGPT and Alk. Phosphate
   • Total Lipid profile
Demographic observations

In the present study majority of the patients were Hindu (73.33%), married (90%), females (67.67%) in 4th to 6th decades of their life (73.34%). Most of them were from middle class family (76.67%) and house wives (60%). Vata-Kaphaja Deha Prakriti (46.66%) and Rajasika Manas Prakriti (60%) along with Madhyama Sara and Samhanana (in 66.67%) were present in the majority of cases. Madhyama Sateva (in 66.67%) in spite of Pravara Abhyavaharanama Shakti (in 63.33%) was present in most of the cases. Most of the cases had Krura Koshitha (in 46.67%), Tikshnagni (in 40%) along with Atinidra (in 50%) and Avara Vyayama Shakti (in 70%). Higher incidence of various Nidana like Guruati Sevana (in 56.66%), Madhuratisevana and Diwaswapna (each in 66.66%), Kshiradsevana (in 63.33%), Navanna Sevana and Anupa and Jaliya Mamsa Sevana (each in 53.33%), Swapnasukha and Achinta (in 50%), Shatiya Sukha and Atibhojana (each in 46.66%), Ayuyama (in 43.33%) were found as etiological factors in patients of Medoroga. Maximum no. of cases i.e. 83.33% patients had negative family history for obesity and only 13.33% of the patients had family history of HTN. 60% of the registered cases had BMI in the range of 30-34.9 kg/m2 followed by 26.67% cases having the range of 25-29.9 and only 10% had BMI more than 35kg/m2.

RESULTS:

Table No 03:- Showing effect of Therapy in Subjective Parameters (B)

<table>
<thead>
<tr>
<th>Sign &amp; symptoms</th>
<th>Group</th>
<th>Mean D</th>
<th>AT(after 30 days)</th>
<th>Relief</th>
<th>% D</th>
<th>S.D</th>
<th>S.E</th>
<th>Kruskal-Wallis Statistic</th>
<th>p</th>
<th>R</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ksludraswaswa</td>
<td>A</td>
<td>2.87</td>
<td>2.00</td>
<td>0.87</td>
<td>30.23</td>
<td>0.74</td>
<td>0.22</td>
<td>6.59</td>
<td>0.036</td>
<td>S</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>3.20</td>
<td>1.33</td>
<td>1.87</td>
<td>58.33</td>
<td>0.676</td>
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</tr>
<tr>
<td>Ksludadthikya</td>
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<td>1.27</td>
<td>0.93</td>
<td>42.42</td>
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<td>0.0178</td>
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<td></td>
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<td>2.27</td>
<td>1.00</td>
<td>1.27</td>
<td>55.88</td>
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<td>12.37</td>
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<td>Pipasadthikya</td>
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<td>2.60</td>
<td>1.60</td>
<td>1.00</td>
<td>38.46</td>
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<td></td>
<td>B</td>
<td>2.27</td>
<td>0.93</td>
<td>1.33</td>
<td>58.82</td>
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<td>18.46</td>
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<tr>
<td>Daurbalya</td>
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<td>0.25</td>
<td>6.15</td>
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<td></td>
<td>B</td>
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<td>1.60</td>
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<td>46.67</td>
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<td>39.02</td>
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<td></td>
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<td>Krichhavyavayata</td>
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<td></td>
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<td>Hriddrava</td>
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<td>0.74</td>
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<td>11.01</td>
<td>0.004</td>
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<tr>
<td></td>
<td>B</td>
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<td>1.40</td>
<td>2.13</td>
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<td>Akasya</td>
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<td>0.23</td>
<td>24.55</td>
<td>&lt;0.0001</td>
<td>ES</td>
</tr>
</tbody>
</table>

(HS: Highly Significant, S: Significant, NS: Non Significant, ES: Extremely Significant)
**Table No 04:** Showing effect of Therapy in Anthropometric Parameters

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Group</th>
<th>Mean</th>
<th>Diff</th>
<th>% Relief</th>
<th>SD±</th>
<th>SE±</th>
<th>t value</th>
<th>p value</th>
<th>R</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body Weight (kg)</td>
<td>Gr.A</td>
<td>75.63</td>
<td>1.93</td>
<td>2.56</td>
<td>13.09</td>
<td>4.46</td>
<td>3.049</td>
<td>0.0087</td>
<td>S</td>
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<tr>
<td></td>
<td>Gr.B</td>
<td>74.40</td>
<td>3.83</td>
<td>5.15</td>
<td>10.31</td>
<td>3.16</td>
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<td>B.M.I. (kg/m²)</td>
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<td>1.17</td>
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<td>1.79</td>
<td>5.72</td>
<td>3.75</td>
<td>1.22</td>
<td>3.728</td>
<td>0.0023</td>
<td>S</td>
</tr>
<tr>
<td>Waist Circumference (cm)</td>
<td>Gr.A</td>
<td>111.17</td>
<td>4.50</td>
<td>4.05</td>
<td>6.97</td>
<td>2.65</td>
<td>3.125</td>
<td>0.0071</td>
<td>S</td>
</tr>
<tr>
<td></td>
<td>Gr.B</td>
<td>109.33</td>
<td>5.00</td>
<td>4.57</td>
<td>9.59</td>
<td>3.53</td>
<td>7.357</td>
<td>&lt;0.0001</td>
<td>ES</td>
</tr>
</tbody>
</table>

(HS: Highly Significant, S: Significant, NS: Non Significant, ES: Extremely Significant)

**Table No 05:** Showing effect of Therapy on Lipid Profile

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group</th>
<th>Mean</th>
<th>MeanDiff.</th>
<th>% Relief</th>
<th>SD±</th>
<th>SE±</th>
<th>t value</th>
<th>p value</th>
<th>R</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sr.TC (mg/dl)</td>
<td>Gr. A</td>
<td>209.57</td>
<td>13.44</td>
<td>6.41</td>
<td>25.18</td>
<td>8.82</td>
<td>1.524</td>
<td>0.0024</td>
<td>S</td>
</tr>
<tr>
<td></td>
<td>Gr. B</td>
<td>230.53</td>
<td>22.73</td>
<td>9.86</td>
<td>21.19</td>
<td>4.18</td>
<td>5.438</td>
<td>&lt;0.0001</td>
<td>ES</td>
</tr>
<tr>
<td>Sr.TG (mg/dl)</td>
<td>Gr. A</td>
<td>173.27</td>
<td>8.33</td>
<td>4.81</td>
<td>11.06</td>
<td>3.71</td>
<td>2.245</td>
<td>0.0042</td>
<td>S</td>
</tr>
<tr>
<td></td>
<td>Gr. B</td>
<td>174.13</td>
<td>12.60</td>
<td>7.24</td>
<td>13.80</td>
<td>4.79</td>
<td>2.632</td>
<td>0.0003</td>
<td>HS</td>
</tr>
<tr>
<td>Sr.LDL (mg/dl)</td>
<td>Gr. A</td>
<td>114.80</td>
<td>6.67</td>
<td>5.81</td>
<td>8.15</td>
<td>2.14</td>
<td>3.114</td>
<td>0.0045</td>
<td>S</td>
</tr>
<tr>
<td></td>
<td>Gr. B</td>
<td>112.23</td>
<td>10.63</td>
<td>9.47</td>
<td>12.36</td>
<td>2.70</td>
<td>3.937</td>
<td>&lt;0.0001</td>
<td>ES</td>
</tr>
<tr>
<td>Sr.VLDL (%)</td>
<td>Gr. A</td>
<td>53.58</td>
<td>5.65</td>
<td>10.54</td>
<td>11.41</td>
<td>3.99</td>
<td>1.414</td>
<td>0.0127</td>
<td>S</td>
</tr>
<tr>
<td></td>
<td>Gr. B</td>
<td>48.27</td>
<td>6.54</td>
<td>13.55</td>
<td>15.90</td>
<td>3.62</td>
<td>1.805</td>
<td>0.0007</td>
<td>HS</td>
</tr>
<tr>
<td>Sr.HDL (mg/dl)</td>
<td>Gr. A</td>
<td>60.33</td>
<td>1.47</td>
<td>2.43</td>
<td>8.83</td>
<td>3.26</td>
<td>0.450</td>
<td>0.0321</td>
<td>S</td>
</tr>
<tr>
<td></td>
<td>Gr. B</td>
<td>73.40</td>
<td>2.93</td>
<td>4.00</td>
<td>17.26</td>
<td>6.54</td>
<td>0.449</td>
<td>0.0002</td>
<td>HS</td>
</tr>
</tbody>
</table>

(Sr.TC-Serum Total Cholesterol; Sr.TG-Serum Triglycerides; Sr. LDL-Serum Low Density Lipoproteins; Sr. VLDL-Serum Very Low Density Lipoproteins; Sr. HDL-Serum High Density Lipoproteins FBS-Fasting Blood Sugar)

**Discussion:**

In *medohuhshti* there is production of either abnormal or excessive *meda* (fat) in the body. Both the conditions are pathological. Due to over production of *meda* other *dhatu* don’t get proper nourishment hence it get depleted that is reflected by their reduced action like *alpaatasa*, *alpasukra* etc. Overproduced *meda* circulates as *abaddhamedha* and deposits as *baddhameda* in the body. Due to this compactness of other *dhatu* are reduced e.g. *mamsa saithilya* etc. leading to easy fatigability as was encountered in the present study. Due to *Avarana* by *Meda & Kapha*, there is *Samana Vayu Prakopa* leading to *Agnisandhikshana* and *Krura Koshthia*. Overeating causes *sarbadoshprakopa* and especially formation of *Ama* which further causes *Apakva-Ama Rasa* & further impaired *Dhatuposhana*. Thusonly *medovriddhi* takes place and whatever other *Dhatu* are formed has *Shaithilya*. Due to *Kaphadhikya* patients have *Atinidra*
and also become less active. Mode of action of both the trial therapeutic measures to break this vicious cycle and pathogenesis of medodushti has been tried to explain as followings -

**Probable action of Vidangadya Churna:** This formulation selected in trial was chosen from Chakradatta Sthautyachikitsa 36/18 and contents of the Vidangadya Churna are Vidanga, Shunti, Yava, Amalaki, Yakvakshara, Lauha Bhasma and Madhuin equal proportion of each. This combination due to dominance of Katu Rasa Dravya, is having potential to break the basic pathogenesis that takes place in Medoroga showing an example of Vyadhpratyantika Chikitsa. Medoroga is a Kaphapradhana-Medopradoshaja Vikara and there is Medavrita-Vata (Cha.Su.21/5) also in it. So, to break the pathogenesis of disease, Dravya having properties opposite to Meda-Kapha & those which have Upchaya Upahanti, Shleshmanam Shamayati, Laghu, Ushna, Ruksa etc. properties are required. The Prabhava of Vidangadya Churanae Medo-Vatabhara (Cha.Su.25/40, A.H.U.40). Lauha Bhasma has the Lekhana property. In this formulation, out of 6 Dravya, there are 4 Dravya having dominant Katu Rasa, 2 Dravya having dominancy of Kashaya & Tikta Rasa. Katu, Tikta & Kashaya Rasa have potential to pacify the Kapha Dosha. Among this, Katu Rasa has potential of Agnisandipana and Mansavilekhanam, which helps to normalise the Jatharagni to form nutritional Amna Rasa as substrate which further gives qualitative nutrition to next Dhatu & helps in modification or normalisation of Dhatwagni. It also helps to scrap out the Abaddha Mamsha-Medo Dhatu from the body. Tikta Rasa also has properties of Deepana, Paachana, Kleda-Meda Shoshaka, Srotovishodhaka & potent in Lekhana property, thus helps to break the pathogenesis of Medoroga. Kashaya Rasa also has property of Sharira-Kleda Shoshhana. All these dominant Rasas in this formulation thus help in breakage of pathogenesis of disease. Besides this, there is dominancy of Laghu, Ruksa & Tikshna and Ushna Guña in the Vidangadya Churna which also helps in Kaphamedashamana and Kledamedakshoshana. 4 Dravya out of 6 in the formulation possesses Laghu & Ruksa Guña. This formulation also has 3 Dravyas with dominant Ushnavirya which also helps to pacify the Vata and Kapha Dosha. Along with these properties, Kaphanashaka property of Yakvakshara helps in Bhedana of Avarana of Samana Vayu. Vatamolomana-Varashamana property of some Dravya helps to normalise the Apana Vayu, thus controlling the Apana Vata itself along with other types of Vata.

**Probable action of Lekhana Basti**

Vata is very important Dosha to be managed during treatment of any disease as Acharya told that other Dosha are handicapped without Vatadosha, and Basti is very important therapy to manage Vatadosha, as its called as Ardhatikitsa or Purnachikitsa in any sort of diseases (Charak Siddhisthana 1/35). Sushruta also (in Chikitsasthana 35) mentioned that not only diseases of Vata, but Pittaja, Kaphaja, Raktaaja, Sansargaja, Samnipatika conditions can also be benefited by Basti. Acharya Sushruta compared Doshima nirharana potential of Basti with Sun; he told that Basti has potential to excrete all the Dosha from body even though it is staying in Pakvasaya (rectum and large intestine), in the same way that the sun has potential to suck all the Rasa from Prithvi (earth) even though it resides in sky.

As Medoroga is a Kapha-Medapradhana and with Medasavrattavata, Acharya Charak recommended use of Ushna-Tikshadravya for Basti. Acharya Sushruta mentioned Lekhanabasti for the management of Medoroga. Lekhana Basti has all its contents with dominant Ruksa,Tiksha, Laghuguna, Ushna Virya, Katu Vipaka and Kapha-Vatabhara properties. With the Samyakad introduction of Basti, there is Srotovishodhana along with Deepana and Pachana i.e. normalisation of Agni at the level of Jatharagni & Dhatwagni. Thus it helps in breaking the pathogenesis of this disease. All the properties of contents of Lekhanabasti administered in this trial, help in Kapha-Medaharana, Karshana of excess Meda from the body, Vatamoloma, and normalising the functions of Apana Vayu. Thus it controls the functioning of Vatadosha as a whole and helps to break the pathogenesis of Medoroga.

**CONCLUSIONS:**

Dyslipidaemia is entity comparable to Abadhdha Medavridhthi mentioned in Ayurveda. It is very prevalent in today’s society and is a potential risk factor for cardiovascular disorders mostly seen associated
with diabetes mellitus & Hypertension. It is reported that Dyslipidaemia is most common in 4th to 6th decade of life, & commonly seen in individuals having sedentary lifestyle, faulty dietary habits and emotionally disturbed individuals. Vīdagāḍya Churna and Lekhana Basti both produced statistically significant improvement in subjective as well as objective parameters but the effect of latter was more marked than that of the former. Thus, it can be concluded that Lekhana Basti is more effective than Vīdagāḍya Churna in management of the Dyslipidaemia. The Study also highlights the fact that Medoroga; considered as a Krichcha-sadhya Vyadhi by Acharya should be preferably treated with the help of Shodhana therapy.

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