“CLINICAL EVALUATION OF VRIHAT PANCHMOOLBALAKSHEER VASTI IN PAIN MANAGEMENT OF SANDHIGAT-VATA VIS-À-VIS OSTEOARTHRITIS”

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ABSTRACT

The millions of people in the world are affected by the disease Sandhigat – Vata vis-à-vis osteoarthritis. The disease was considered as a simple degenerative disease from a long time, but recent studies have revealed that, this is not simple degenerative disease, it is an end result of various pathology. Now its etiopathogenesis is said to be intricate which bridges biomechanics and biochemistry. Most of the cases of Sandhigat – Vata are of primary type (Dhatukshay, as said by Ayurveda) and rest cases of secondary type (due to other disease pathology). According to Ayurveda, etiopathogenesis of Sandhigat Vata starts with vitiated Vata and Dhatukshay due to malpractice of Ahar and Vihar of Vat Vardhak Gunas. Dhatukshay results in development of the disease pathology whereas vitiated Vata is capable in producing severity of the disease symptoms. The symptoms are also due to pathology of disease (dhatukshay). Basically Vata and Kapha Doshas have opposite properties each other, hence if Vata vitiates, there is decrease functioning of Kapha and vice versa. In the disease Sandhigat –Vata there is focal cartilage changes of a affected joint by the vitiated Vata and decreased functioning of Kapha. The focal cartilage changes are due to imbalanced these two doshas because the Sandhies are specific site of Sleshak Kapha and Sandhies and Asthies are specific sites of Vata especially Vyan Vayu. The therapies of the present study were undertaken on focusing over the pathology and Doshas involved in the development of Sandhigat – Vata. Vrihat Panchmooolbalaksheer Vasti has capable in pacify Vata Doshas due to Vrihat Panchmoool Dravyas, checks Dhatukshay due to Bala and Ksheer and improves Sleshak – Kapha functions due to Ksheer in it. Present study has been divided into two groups based on the type of therapy to which patients were subjected;
Group 1; Treated with Vrihatpanchmoolbalaksheer Vasti therapies.
Group 2; Treated with luke hot plain water Vasti therapy only.

**KEYWORD:** The millions Vrihat Panchmoolbalaksheer Vasti (dhatukshay).

**INTRODUCTION**

Osteoarthritis is a degenerative form of arthritis that features the breakdown and eventual loss of the cartilage of one or more joints. Cartilage is a structure of protein substance that serves as a "cushion" between the bones of the joints. Before age 45, osteoarthritis occurs more frequently in males. After 55 years of age, it occurs more frequently in females. Globally as of 2010, approximately 250 million people had osteoarthritis of the knee (3.6% of the population). Hip osteoarthritis affects about 0.85% of the population. Together, knee and hip osteoarthritis had a ranking for disability globally of 11th among 291 disease conditions assessed Sandhigata-Vata is briefly described in Ayurvedic texts. In the ideal text of Ayurvedic medicine (the Charaka Samhita), Charaka has termed the ‘Sandhigat Vata’ as Sandhigat-Anila (Anila=Vata) in ‘Vat-Vyadhi-Chikitsa’ chapter 28 and described as-

‘Vata-Purnadritispash’ (soft swelling like air filled bag), ‘Shoth’ (inflammation, non-suppurative) and ‘Prasaranal/Kunchanyoh Pravrattisch savedna’ (painful joint movements especially of extension and flexion). Another Ayurvedic texts are also defined the term Sandhigat-Vata and their treatment but especially by Susrut; ‘Sandhi-Hanti’ (destruction in the articular structures of the joint) and ‘Asthishosh’ (osteoporosis) are also described. So, it is pertinent to point out that Sandhigat-Vata is almost similar disease as that of osteoarthritis (OA) in modern medical science.

The etiopathogenesis particularly for Sandhigat-Vata is not described independently in the Ayurvedic texts but on the basis of major etiological factor - vitiated Vata, various descriptions are mentioned. According to those descriptions; early pathology of Sandhigat-Vata starts with vitiated vata due to the practice of Vata-Vardhak Ahar and Vihar (excessive exercise and diet responsible for malnutrition and catabolism), Dhatukshay (degeneration) as by ageing process or Avarana of the Vata (neurological pathology due to metabolic and endocrinal disturbances). Accumulation of vitiated Vata in Sandhisthanas facilitated by two main factors-
1. Because Sandhi and Asthi are specific sites for Vata and
2. Khavaigunyat.
The present work entitled ‘Clinical evaluation of Vrihat Panchmoolbala Ksheer Vasti in the pain management of Sandhigat-Vata vis-à-vis osteoarthritis’ aim to undertake a critical literary and conceptual study, detailed demographic and clinical study and a study to evaluate the efficacy of different therapeutic regimen for the treatment of Sandhigat-Vata.

REVIEW OF DISEASE PATHOGENESIS

AYURVEDIC REVIEW

The disease Sandhigat-vata is briefly described in Ayurvedic texts and said to be a Vatik disorder (Vat-Vyadhi; ch.chik. 28/37) and also by Sushrut (su.nid. 1/38), Madhav (m.nid. 22/21). The Sandhigat-Vata is chiefly characterized by soft swelling as air-filled bag (Vatapurnadritisparsh), inflammation with degenerative changes, tenderness and stiffness (shoth), painful joint movements esp. during flexion and extension (prasaran akunchanh privritti svedna), destructive changes in the joint structures (hntisandhigath), pain in the joint especially of aching or tearing nature (shoool/bheda), may be with osteoporosis (asthishoshm), and heaviness (aatop) in the joint affected. Basically above mentioned signs and symptoms are seen when Sandhigat-Vata is in their progressive phase with chronicity. There are also some other clinical symptoms in Sandhigat-Vata due to specific with vitiated Vata in the joint (Ch.chik. 28/20).

The word Sandhigat-Vata is having two distinct vital parts-
1. Sandhi.
2. Vata.

The Sandhi in Ayurveda is a anatomical part implicated for that where two separate bones, muscles or other structures met. So in the case of Sandhigat-Vata, Sandhi means the joint or articulars. The Vata according to Ayurveda is a factor/component of the body responsible for physio-pathological changes. This is a major controlling system of the body up-to the cellular level and known as nervous system of the body.

All the aetiological factors are independently responsible for the vitiation of Vata. In Ayurvedic classics, there is no specific description of the pathogenesis of Sandhigat-Vata like some other diseases regarding Doshas, Dusyas, Srotas and their Dushti, Adhisthan etc, but there is much description regarding pathogenesis of Vatik disorders (Sandhigat-Vata also is a pure Vatik disorder; Ch. chik. 28/18). Vitiated Vata produce the above mentioned diseases by entering in hollow organs as like Asthi and Sandhi. Asthi and Sandhi are also
assumed as residing places of *Vata* (A.S.sut. 20/3). The occurrence and pathogenesis of the diseases due to *Vata* is due to *Kha*-vagunya (altered physiological changes) of that particular part/place of the body (Sh. Pur. 5/25). Vitiated *Vata* degenerate and absorbs *Sleshak-kapha* (*Sleshakkapha-ksyay*) is responsible for decreased stability (*Sthiratva*) of the joints, malalignment and remodeling of the joint contour due to *Sandhibhandhan- Vikriti* and decreased lubrication (increased friction in the articular surfaces) due to *Asingdhatva*. All the above pathology is the end result of degeneration in the articular cartilage and sub-chondral bone changes.

**SIGNS AND SYMPTOMS**

On the basis of description in the various *Ayurvedic* classics, signs and symptoms of *Sandhigat-Vata* are as follows (*Ch Chik. 28/37, Su.nid.1/38, Ma. Nid. 22/21, B.P.mad. khand 24/258*) -

- **Vatpurna-driti sparsh** (on palpation of the affected joint, there is a feeling of soft swelling as like air filled bag). This sign is especially mentioned for knee joint where soft swelling present on both lateral sides of the knee. In this type of swelling, there are no color changes, heat and pain. Pain and tenderness are of deep origin inside the joint and their articulations.

- **Shoth** (inflammation). This inflammation not a typical inflammation is due to changes in the joint articulations. It can be says that this is a degenerative inflammatory process.

- **Prasaran-akunchanyoh pravrittisch savedana** (painful joint movement especially of flexion and extension movements). It is due to degenerative changes which aggravate *Vata* also and friction between articular surfaces.

- **Sandhi-hantin** (destructive changes in the joints). These are in the form of osteophytes formation, mal-alignment between the articular bones and pseudocysts formation also. This is mentioned by *Sushrut* and found in late and severe stage.

- **Asthi-shosh** (osteoporosis and sub-chondral bone changes). It is also mentioned by *Sushrut* and found in late and old aged cases. Normally in old cases which have tendency to develop osteoporosis are more prone to the severity of disease.

- **Sandhi-shabd** (crepitus during flexion and extension). It is a cardinal symptom of *Sandhigat-Vata* but is not mentioned in the symptomatology of *Sandhigat-Vata* in *Ayurvedic* classics and is due to friction between articular surfaces.
• **Mans-ksaya** (muscle wasting). It is due to no use of the joint therefore muscles get weakened. Other factors responsible for weak and wasted muscles are vitiated *Vata* and degenerative changes.

• Other associated clinical features (*Ch.Chik*. 28/20)-
  - *Sankoch* (muscle spasm).
  - *Stambh* (stiffness).

These are due to vitiated *Vata* and responsible for narrowing of the joint space. Narrowing of the joint space is a morbid factor in the development of the disease *Sandhigat-Vata*. Pain is especially during and after use in the joint and it may be localized or spread to the adjacent structures or may be referred through the nerve root to the other parts of the body. Pain typically is provoked by use of the joint and relieved by the rest. Stiffness in the affected joints mainly occurs after rest or when first getting out of bed and is usually less than 15-20 minutes but more period may occur. Crepitus typically of snow-ball scrunching and grating type are audible by the use of stethoscope or may be felt by the palm of hand. Muscles are hypotonic and in long standing cases, wasted also but true myopathy is not present. Deformity in the joint is due to osteophytes formation, friction between the articular surfaces, mal-alignment of the articular bones and weakness of the muscles around the joint.

**MODERN REVIEW**

Osteoarthritis is a degenerative disease and these degenerative change occur mainly in the articular cartilage. It is classified as primary and secondary osteoarthritis in which pathogenesis of primary OA is still unknown and is believed that this is a ageing process, whereas pathogenesis of secondary OA is clearly understood with a recognizable local or systemic factor.

**Pathogenesis**

The pathogenesis of secondary OA depends on the causative changes in the degeneration. Due to the trauma i.e. fractures etc. there is mal-alignment of the articular bones at their surfaces and incongruity of the joints. Traumas play a important predisposing factor of premature OA. Developmental abnormalities are believed to be of major importance in the aetiology of hip OA. Collagen gene defects have been identified in a few families in whom familial, premature, polyarticular OA is associated with an epiphyseal or spondyloepiphyseal dysplasia. Abnormal articular surface contacts and weight –bearing alignments lead to
increased local mechanical stress and wear. Metabolic diseases lead to cartilage degeneration by different mechanisms. Alkaptonuria is with genetic defect of homogenetisic acid oxidase and results in accumulation of a pigmented polymer that binds to the collagen, rendering it brittle and prone to mechanical degradation. Crystals of calcium pyrophosphate dehydrate or hydroxyapatite if deposited in the joints there may be altered properties of cartilage matrix. Acromegaly is a consequence of joint incongruity due to bony changes following cartilage outgrowth. Other endocrinal diseases with disturbances in the endocrinal physiology result in a mechanically defective metabolism. Aseptic necrosis due to Paget’s disease, Gaucher’s disease and other various disease results stress on the overlying articular cartilage.

Schematic representation of the pathogenesis of OA is as follows-

1; Fatigue fracture of Collagen fibre network
   ↓
Increased hydration of The articular cartilage
   ↓
Unraveling and loss of proteoglycans into synovial fluid
   ↓
Collagen loss
   ↓
OA

2; Micro fractures of the sub-chondral bone
   (following repetitive loading)
   ↓
Fracture healing leads to loss of resilience of the subchondral bone
   ↓
Fibrillated cartilage surface and deep clefts appear
   ↓
Proliferative changes (at the Joint margins)
   with formation of osteophytes
   ↓
Loss of articular cartilage
   ↓
Hard and eburnated underlying bone
   ↓
OA

Osteoarthritis is the end result of multiple factors with the following changes-

1. Loss of proteoglycan.
2. Fracture of the collagen mesh network.
3. Metabolic changes and cell loss.
Interleukin-1 (IL-1), tumor necrosis factor alpha (TNF-α) insulin like growth factor – 1 (IGF-1) and transforming growth factor beta (TGF-β) have been found to play an important part in the articular cartilage metabolism, and in matrix protein synthesis and degradation.

Basic pathological changes:

- In the articular cartilage: The integrity of the articular cartilaginous surface represents a fine balance between ‘wear and tear’ losses and replacement by chondrocytes of the specialized matrix. There is marked surface loss of the articular cartilage. The earliest change in ageing and OA is in the chemical composition of the matrix which becomes softer, this is followed by progressive characteristic morphological changes. At the site of pressure, early changes are flaking and fibrillation of the cartilaginous surface but later there are areas of cystic degeneration and loss of cartilage. It results in exposure of bone which becomes hard and polished (eburnated). So there is osteophytes formation at the edge of the joint. Osteophytes are bone forming from cartilage or outgrowth of irregular bone forming a lip round the joint margin. Osteophytes may contribute to limitation of movements. The bone cysts are formed within the bone (subchondral) where synovial fluid is forced under pressure through minute minute cracks in the bone. In the synovial membrane, there is villous hypertrophy possibly response to ingestion of flakes of degenerated cartilage by phagocytosis of synovial membrane. Secondary acute synovitis occurs in inflammatory episodes due to degenerative changes. Due to degenerative changes during the period of rest, there is marked decrease secretion of the synovial fluid. When joint movements are initiated, there is proper secretion of synovial fluid, so after prolonged rest patient feels stiff joint which is relieved after movements. There is fibrosis and contraction in the capsular ligament.

- Biochemical changes in the cartilage due to degeneration are increased water content, decreased collagen, proteoglycan, monomer size, hyaluronate, keratin sulphate, chondroitin increased chondroitin ratio (4:6), increased collagen and proteoglycan synthesis and increased aggrecanase, stromelysin and collagenase.

- Heberden’s and Bouchard’s nodes are mucoid degeneration of capsule and soft tissues overlying joints form cysts containing pure hyaluronic acid, later cartilaginous and bony metaplasia.
DISTRIBUTION AND PROGRESSION

Most common affected joints by osteoarthritis are weight bearing joints or those which are prone to repetitive fractures i.e. the knee, hip, metatarsophalangeal or interphalangeal joints. Osteoarthritis can affect the following joints;

- Joints of the knee, hip, metatarsophalangeal, carpometacarpal or interphalangeal, cervical-dorsal-lumbar regions
- Generalized OA may occur

The course of OA is highly variable in long term studies; one third to two third of radiographic OA have been shown to progress but improvements are rare. Symptomatic OA may progress or improve or may even be arrested. The patients with multiple affected joints have more rapid progression of OA. Advanced age and obesity are also associated with amore rapid progression of OA.

CLINICAL FEATURES OF OA

Asymmetrical and individual or symmetrical and two or more joints can be affected by secondary osteoarthritis due to various distinguished factors. But primary generalized OA (PGOA) is of two type

1. Nodal and.
2. Non-nodal.

Nodal OA

- It occurs predominantly in middle-aged women.
- The onset is insidious but sometimes may be acute.
- It affects characteristically terminal end/or proximal interphalangeal joints.

Herberden’s nodes are gelatinous cysts or bony outgrowths on the dorsal aspects of the fingers affect terminal interphalangeal joints. Bouchard’s nodes are similar lesions may affects the proximal interphalangeal joints.

Clinical features are

- Pain.
- Swelling.
- Inflammation.
- Deformity may occur.
Non-nodal generalized OA

- The common age of presentation of disease is 40-70 years but early aged persons may also be affected by the disease.
- The onset is insidious but acute onset may occur with pain, stiffness and swelling in the joint especially during morning and working hours.
- Pain is the cardinal symptom, usually at first, it is intermittent localized deep ache and often aggravated by the joint use and relieved by rest. Pain is especially during and after use, it may be localized or spread to the adjacent structures or may be referred through the nerve root to the other parts of the body, i.e. knee pain can referred to the hip joint and vice versa. As the disease progresses, the pain becomes persistent and in advanced cases, pain worsens at night also and disturbs the sleep. In advanced cases, night pain worsens at the lateral positions or crossing the legs each other. Pain in the knee OA is provoked by using the stairs, laterines, rickshaws and autorickshaws, train and buses etc. Osteoarthritis of hip cause pain especially during movements and certain sitting postures whereas OA of the spine is associated with pain during forward and lateral bending and radiculopathy with cervical OA.
- Stiffness is found chiefly during morning and after prolonged rest and usually lasts less than 20 minutes. It is relieved after joint movements because of lubrication of the articular surfaces by the synovial fluid. During the period of rest, secretion of synovial fluid is markedly decreases. At this time further destruction in the cartilage is prevented by the associated muscle spasm. When movements of the joint are enhanced, secretion of the synovial fluid is increased which lubricates the joints, so stiffness is relieved. Stiffness is frequently found during the acute stage.
- Tenderness is present during the acute stage and progression of the disease. It can be elicited by pressing both lateral sides of patella and lateral and medial aspects of the knee joint, where Tibial and femoral ends meet. Tenderness is more elicited by flexing the knee joint. In acute and severe cases, when pressure is given to elicit tenderness, patient feels much pain.
- Swelling is found during acute stages of osteoarthritis particularly in advanced OA. Occasionally bony swelling due to osteophytes may be present. Swelling is due to inflammation of the synovial membrane or effusions. Sometimes swelling is huge on the both lateral sides of the knee joint above the midline of articulation.
• Crepitus or grating sensations are sound feel by the palm of the hand or auscultated by the stethoscope by the observer. Sometimes crepitus can be heard. These may be fine or course in nature and are typically of scrunching and grating type. These sounds are due to friction between the articular surfaces and are provoked by narrowing of the joint space.

• Restriction of movements is due to capsular fibrosis, osteophytes, malalignment of the articular bones or impaction of loose bodies. Joint movements are also restricted by severity of the joint pain, stiffness and swelling. The range of the movement purely depends on the above factor with severity and progression of the disease. During movement patient restricts the movements by voluntary contraction of the muscles to prevent increment of pain.

• Muscle wasting – There is wasted and hypotonic muscles due to their disuse whereas true myopathy is always absent as like in rheumatoid arthritis. Deformity in the joint is due to malalignment of the articular bones and also due to weakness of the muscles and fibrosed capsule.

MATERIAL AND METHODS
A series of 69 patients of Sandhigata – Vata vis-à-vis osteoarthritis were randomly selected for the present study from OPD and IPD of Panchkarma department, Rishikul Govt. Ayurvedic College Hospital, Haridwar. The cases were randomly selected regardless of their age sex and socioeconomic considerations, but fully satisfying the criteria of diagnosis of OA in conventional medicine and clinical features of Sandhigat–Vata as described in Ayurvedic medicine. Out of 69 patients, only 45 cases could complete their follow – up i.e. 3 months and 24 patients did not turn up for regular follow up. The following exclusion and inclusion criteria were adopted for the selection of cases of osteoarthritis:

Exclusion criteria: Major exclusion criteria was:
- History or active presence of other inflammatory or rheumatic diseases.
- Patients more than 80 years.
- Substantial abnormalities in blood, hepatic, renal or endocrinal diseases.

Inclusion criteria: Major inclusion criteria was:
- Patients fulfilling the diagnostic criteria of osteoarthritis
- Cases not violating exclusion criteria
- Patients aged over 30 years.
- Cases of primary osteoarthritis.

**Trial drug preparation**

In Vrihatpanchmoolbalaksheer Vasti, contents/drugs used are-Kwath, Bilwa, Agnimanth, Shyonak, Patla, Gambhari, Bala, Ksheer (milk), Maksik (madhu; honey), Lavana (saindhav salt), Sneha (oil); Mahanarayan Tail.

(a) Vrihat Panchmoolbalaksheer Vasti was prepared according to the method of preparation as described in Charak Siddhisthan chapter 3/23 and Astang Hridaya Sutrasthan chapter 19/45. So that series was Maksikam (honey), Saindhav/Lavanam (salt), Sneha (Mahanarayana Tail), Kalkam (paste of soya powder) and Kwatham (decoction of the selected drugs for Vasti therapy) with Ksheeram (milk).

**Dosage**

The trial Vasti therapy was used per rectally along with Anuvasana Vasti as Kala- Vasti. The dosage of individual ingredient and of prepared Vasti liquid was as follows –

* Maksikam = 100ml.
* Lavanam = 15gm.
* Sneham = 175ml.
* Kalkam = 60gm.
* Kwatham = 250ml. (with ksheer).

\[ \text{Total liquid} = 600 \text{ ml.} \]

\[ \text{/ Asthapana Vasti.} \]

**Method of study**

Present study has been divided into 2 groups based on the type of therapy to which patients were subjected:

**Group 1:** Vrihat panchmoolbalaksheer Vasti – this group of patients were treated with Vrihat Panchmoolbalaksheer Vasti.

**Group 2:** Plain luke warm water Vasti – this group of patients were treated with luke warm water.

<table>
<thead>
<tr>
<th>Group</th>
<th>No. of patients registered</th>
<th>No. of completed follow – ups</th>
<th>Treatment given with duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>28</td>
<td>15</td>
<td><em>Vasti</em> with <em>kwath</em> for 10 days – a month for 3 months</td>
</tr>
<tr>
<td>2</td>
<td>23</td>
<td>15</td>
<td><em>Vasti</em> with water --for 10 days – a month for 3 months</td>
</tr>
</tbody>
</table>
**Procedure of Vasti**

The vasti consisted of both *Anuvasana* (predominantly oil based) and *Asthapana* of *Niruh* (predominantly decoction based) measures. The ratio of *Anuvasana* and *Asthapana* has been provided as per classical referenced of *kala – vasti*. *Abhyanga* (massage) and *Swedana* (sudation) were carried out in all the patients for *Vasti* therapy as a preparatory procedure. Following the above procedures, patients was advised to lie down in a left lateral position with his left leg kept straight whereas right knee kept in a semi flexed position and *Vasti* was given. After that, patients were advised strictly to avoid taking *Asta – Mahadoshkar Bhavas*

- The contents of *Kwatham* total 50gm/day were taken and boiled with 800ml. of water and 200ml. of milk until ¼ (250ml) of the decoction is left.
- Total amount of *Vasti* liquid prepared was kept around 600ml.

*Anuvasana Vasti: Til Tail = 60ml.*

**Clinical assessment of disease**

Clinical assessment of symptoms and severity was objectively done in terms of gradation of pain score and other associated symptoms. The relative extent of all these criteria was recorded according to the rating scale in each patient at the initial stage and subsequent follow – ups.

These symptoms and their grading score procedures are as follows:

**Gradation of pain score**

0 = nil; no pain in the joints.

1 = mild pain; pain complained but tolerable.

2 = moderate pain; pain complained and disturbs routine work.

3 = severe pain; severe pain completely interrupting routine work.

Maximum distance walking.

- Without limits 0.
- More than 1km, but limited 1.
- More than 1km, about 15 minutes 2.
- From 500-900m (about 8-15min.) 3.
- From 300-500m 4.
- From 100-300 5.
- < 100 m 6.
Assessment of pain through visual analogue scale (VAS): For adequate treatment of pain assessment of the degree of pain, the patient feels is important. So in the present study, VAS was used to assess the degree of pain.

**Visual analogue scale (VAS)**

It is the easiest way to measure the intensity of pain and can be analyzed quickly. It provides simple efficient and minimally intrusive measures of pain intensity. The most common VAS consists of a 10 cm. horizontal (Huskisson 1983) or vertical (Sriwatanakul et al, 1983) line with the two end points labeled 0 = no pain and 10=worst pain ever. The patients are required to place a mark on the 10cm line at a points which corresponds to the level of pain intensity he/she presently feels. The distance in cm. form the low end of the VAS to the patient’s mark is used as a numerical index of the severity of pain.

- Visual analogue scale
  
  → 10 worst pain ever
  
  →
  
  →
  
  →
  
  →
  
  →
  
  →
  
  →
  
  →
  
  →

→ 0 no pain.

**OBSERVATION AND RESULT**

% distribution of severity of pain in group-1.

<table>
<thead>
<tr>
<th>Severity</th>
<th>BT</th>
<th>F1</th>
<th>F2</th>
<th>F3</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. %</td>
<td>No. %</td>
<td>No. %</td>
<td>No. %</td>
</tr>
<tr>
<td>Absent</td>
<td>0 0</td>
<td>1 6.67</td>
<td>4 26.67</td>
<td>9 60</td>
</tr>
<tr>
<td>Mild</td>
<td>2 13.33</td>
<td>5 33.33</td>
<td>7 46.67</td>
<td>4 26.67</td>
</tr>
<tr>
<td>Moderate</td>
<td>6 40</td>
<td>5 33.33</td>
<td>4 26.67</td>
<td>2 13.33</td>
</tr>
<tr>
<td>Severe</td>
<td>7 46.67</td>
<td>4 26.67</td>
<td>0 0</td>
<td>0 0</td>
</tr>
<tr>
<td>Total</td>
<td>15 100</td>
<td>15 100</td>
<td>15 100</td>
<td>15 100</td>
</tr>
</tbody>
</table>
The data reveals that the most of the cases were shifting towards reducing severity of pain. After completion of 3rd follow up, maximum shifting is into absent group (60%) whereas other cases shift into mild (26.67%) & moderate group (13.33%) from severe (46.67%), moderate (40%) & mild group (13.33%) with decreasing in severity of pain.

<table>
<thead>
<tr>
<th>% distribution of severity of pain in group-2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Severity</td>
</tr>
<tr>
<td>----------</td>
</tr>
<tr>
<td>Absent</td>
</tr>
<tr>
<td>Mild</td>
</tr>
<tr>
<td>Moderate</td>
</tr>
<tr>
<td>Severe</td>
</tr>
<tr>
<td>Total</td>
</tr>
</tbody>
</table>

The data reveal that there is minimum shifting of most of the cases towards reducing severity of pain. After completion of 3rd follow-up, minimum shifting is into absent group (6.67%) & remaining into mild (13.337%) & moderate group (66.67%) with no significant relief in severity of pain.

Table no.10: COMPARISION OF SEVERITY OF PAIN IN GROUP1&2.

<table>
<thead>
<tr>
<th>Group</th>
<th>Comparison</th>
<th>d</th>
<th>SD</th>
<th>SE</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>BT vs AT</td>
<td>1.8</td>
<td>0.6761</td>
<td>0.1746</td>
<td>10.311</td>
<td>0.01*</td>
</tr>
<tr>
<td>2</td>
<td>BT vs AT</td>
<td>0.3333</td>
<td>0.9759</td>
<td>0.252</td>
<td>1.3229</td>
<td>0.10**</td>
</tr>
<tr>
<td>1 vs 2</td>
<td>BT</td>
<td>0.2</td>
<td>1.0142</td>
<td>0.2619</td>
<td>1.3229</td>
<td>0.10**</td>
</tr>
<tr>
<td></td>
<td>AT</td>
<td>1.4667</td>
<td>0.7432</td>
<td>0.1919</td>
<td>7.6429</td>
<td>0.01*</td>
</tr>
</tbody>
</table>

* significant.
** Non significant

The above table shows that there is significant relief in severity of pain by treatment with Vihaat Panchmoolbalaksheer Vasti in comparison with the second control group. Comparison between groups is also suggestive for there is no significant difference in pain between two groups before treatment but there is significant difference between groups after treatment. It shows Vihaat panchmoolbalaksheer Vasti is highly effective in pain management of OA patients.

**DISCUSSION**

The Vasti therapy alone exhibit mild to moderate degree of clinical improvement in terms of reducing pain, swelling, tenderness, crepitus, restriction of movement and stiffness with some
clinical improvement in reducing the severity of crepitus. So even use of luke hot water as Vasti is mild effective in few cases.

- The effect of Vrihat panchmoolbalaksheer Vasti therapy in reducing the severity of disease is said to be very good. There is maximum shifting of cases into mild and absent group in group 1 rather than group 2 where the changes were minimum. All the seven patients of severe group start to move in upper group just after in first follow up.
- In group 2 one case from sever group shifted into moderate group and 2 patients of moderate group is shifted from moderate to mild group. One case of mild group is shifted from mild to absent group. And this movement is noticed after the third follow up which is after three month duration.

SUMMARY AND CONCLUSION

Over all therapeutic response of patients in group - 1

- The trial therapies ‘Vrihat Panchmoolbalaksheer Vasti’ exhibit over all better response in terms of reducing pain with some clinical improvement.
- The effect of trial therapies in reducing the severity of disease can also be presented as of moderate degree of clinical improvement. The maximum shifting of cases in terms of reducing severity was into absent and mild group then in moderate group. It can be says that Vasti therapy checks the degenerative changes and pacify vitiated Vata and treats the disease symptomatically on clinical parameters.

Over all therapeutic response of patients in group - 2

- The Vasti therapy alone exhibit mild degree of clinical improvement in terms of reducing pain even with luke hot water. But no significant effect is seen in reducing in majority of cases.
- The effect of plain water Vasti therapy in reducing the severity of disease is said to be mild. There is minimum shifting of cases between groups is evidence of it.

Over all response of Vasti therapy on pain management was significant in between two groups.

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