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CLASSICAL AND CONTEMPORARY CAUSES OF JANU SANDHIGATA VATA

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ABSTRACT

The treatment of any disease is initiated by knowing the relevant present and past history. This gives us insight into the causes and symptoms concerned. Though there are innumerable causes specific to the person/age /gender/habitat and the like in Ayurveda, a generalized idea about all the factors involved is necessary for the ideal diagnosis and treatment. The topic in context is *Janu Sandhigata Vata* wherein a clinical study was carried out with 40 patients. *Janu Sandhigata Vata* is one among the *Gatavata* mentioned under the context of *Vatavyadhi* in the *Samhittas*. It can occur due to *Dhatukshaya* or *Avarana*. It is correlated to Osteoarthritis (OA) due to the similarities in manifestation. Osteoarthritis (OA) is a very common chronic disease that affects all joint tissues, causing progressive irreversible damage and finally, the failure of the joint. The various Nidana or causes ruled in favour for the formation of the disease are being considered for the study in this article.

KEYWORDS: Nidana, Janu Sandhigata Vata, Osteoarthritis, Menopause, Obesity.

INTRODUCTION

Janu Sandhigata Vata is one among the Gatavata mentioned under the context of Vatavyadhi in the Samhittas. It is a Shoola and Shotha Pradana Vata characterised by clinical features Vyadhi like Shoola(Pain), Atopa(crepitus), Vata Poorna Druthi Sparsha (palpable like air filled bag), Shotha(swelling) and Prasarana Akunchana Vedana (pain during movements).^[1] It is similar to that of Osteoarthritis mentioned in contemporary literature. Osteoarthritis is the second most common rheumatologic problem and it is the most frequent joint disease with a prevalence of 22% to 39% in India. Osteoarthritis (OA) is a very common chronic disease that affects all joint tissues, causing progressive irreversible damage and, finally, the failure of the joint as an organ.^[2] Characteristic pathological changes in OA not only include joint cartilage degeneration but also subchondral bone thickening, osteophyte formation and synovial inflammation, all of which are associated with capsule laxitude and decreased muscle strength.^[3] The pathological changes that occur in OA are the result of the action of biomechanical forces coupled with multiple autocrine, paracrine and endocrine cellular events that lead to a breakdown of the normal balance in tissue turnover within the joint.^[4,5]

To understand the nature and extent of causation, the study was conducted. The patients diagnosed with *Janu Sandhigata Vata* (Osteoarthritis knee joint) approaching the OPD and IPD of SKAMCH&RC, Bengaluru were selected and the clinical relevance of the nidana (causes) are discussed. In the present study, 40 subjects were registered of which 20 subjects were registered under Group A with Sahacaradhi Kashaya,^[6] as internal medication and 20 subjects were registered under Group B with *Maharasnadhi Kashaya*,^[7] as internal medication. *Jatamayadhi Upanaha Sweda*,^[8] was selected as the *Bahya Upakrama*. The assessment of the various *Nidana* to that of the risk factors is being discussed in this article.

OBSERVATIONS

Age

In Group A, out of 20 subjects, maximum number of subjects (11 in number i.e., 55.0%) belonged to the age group of 50 -59 years, 7 subjects (35.0%) belonged to the age group of 60-70 years and 2 subjects (10.0%) belonged to the age group of 40-49 years.

In Group B, out of 20 subjects, maximum number of subjects (7 in number i.e., 35.0%) belonged to the age group of 60-70 years, 6 subjects (30.0%) belonged to the age group of 50-59 years, 4 subjects (20.0%) belonged to the age group of 30-39 years and 3 subjects (15.0%) belonged to the age group of 40-49 years.

Age	Group A	Group B	Total
30-39	0	4 (20.0%)	4 (10.0%)
40-49	2(10.0%)	3 (15.0%)	5(12.5%)
50-59	11 (55.0%)	6 (30.0%)	17(42.5%)
60-70	7 (35.0%)	7 (35.0%)	14 (35.0%)
Total	20 (100.0%)	20 (100.0%)	40

Gender

In Group A, out of 20 subjects, maximum number of subjects (15 in number i.e., 75.0%) belonged to female gender and 5 subjects (25.0%) belonged to the male gender.

In Group B, maximum number of subjects (14 in number i.e., 70.0%) belonged to the female gender and 6 subjects (30.0%) belonged to the male gender.

Gender	Group A	Group B	Total
Female	15 (75.0%)	14 (70.0%)	29 (72.5%)
Male	5 (25.0%)	6 (30.0%)	11 (27.5%)
Total	20 (100.0%)	20 (100.0%)	40

Habitat

In Group A, out of 20 subjects, maximum number of subjects were from *Sadharana Desha* (18 in number i.e., 90.0%) and 2 subjects were from *Anupa Desha* (10.0%).

In Group B, out of 20 subjects, maximum number of subjects were from *Sadharana Desha* (19 in number i.e., 95.0%), 1 subject (5.0%) was from *Jangala Desha*.

Habitat	Group A	Group B	Total
Sadharana	18 (90.0%)	19 (95.0%)	37 (92.5%)
Jangala	-	1 (5.0%)	1 (2.5%)
Anupa	2 (10.0%)	-	2 (5.0%)
Total	20	20	40

Occupation

In Group A, out of 20 subjects, maximum number of subjects were housewives (11 in number i.e., 55.0%), 7 subjects (35.0%) had other occupations (carpenter, construction worker, criminal lawyer, delivery boy, garment factory worker, shopkeeper and teacher) and 2 subjects were tailors (10.0%).

In Group B, out of 20 subjects, maximum number of subjects were housewives (10 in number i.e., 50.0%), 4 subjects were teachers (20.0%), 4 subjects (20.0%) had other occupations (Technician, software engineer, government staff and retired manager) and 2 subjects were shopkeepers (10.0%).

Occupation	Group A	Group B	Total
Housewife	11 (55.0%)	10 (50.0%)	21 (52.5%)
Teachers	-	4 (20.0%)	4 (10.0%)
Tailors	2 (10.0%)	-	2 (5.0%)
Shopkeepers	-	2 (10.0%)	2 (5.0%)
Others	7 (35.0%)	4 (20.0%)	11 (27.5%)
Total	20	20	40

Family History

In Group A, maximum number of subjects had family history (11 in number i.e., 55.0%) and 9 subjects (45.0%) had no family history.

In Group B, maximum number of subjects had family history (13 in number i.e., 65.0%) and 7 subjects (35.0%) had no family history.

Family history	Group A	Group B	Total
With history	11 (55.0%)	7 (35.0%)	18 (45.0%)
Without	9 (45.0%)	13 (65.0%)	22 (55.0%)
history			
Total	20	20	40

Fresh or treated cases

In Group A, maximum number of subjects were those who had taken intervention previously (15 in number i.e., 75.0%) and 5 subjects were fresh cases (25.0%).

In Group B, maximum number of subjects were those who had taken intervention previously (14 in number i.e., 70.0%) and 6 subjects were fresh cases (30.0%).

Fresh or Treated cases	Group A	Group B	Total
Fresh cases	5 (25.0%)	6 (30.0%)	11 (27.5%)
Treated cases	15 (75.0%)	14(70.0%)	29 (72.5%)
Total	20	20	40

Joints Involved

In Group A, maximum number of subjects had bilateral involvement (16 in number i.e., 80.0%) and 4 subjects had unilateral involvement.

In Group B, maximum number of subjects had bilateral involvement (15 in number i.e., 75.0%) and 5 subjects had unilateral involvement.

Bilateral or Unilateral involvement	Group A	Group B	Total
Unilateral involvement	4 (20.0%)	5 (25.0%)	9 (22.5%)
Bilateral involvement	16 (80.0%)	15 (75.0%)	31 (77.5%)
Total	20	20	40

Chronicity

In Group A, maximum number of subjects had chronicity of 2-3 years chronicity (5 in number i.e., 25.0%), 4 subjects had chronicity of 4-5 years (20.0%), 4 subjects had chronicity of < 1 year (20.0%), 3 subjects had chronicity of > 5 years (15.0%), 2 subjects had chronicity of 1-2 years (10.0%) and 2 subjects had chronicity of 3-4 years (10.0%). In Group B, maximum number of subjects had chronicity of > 5 years (6 in number i.e., 30.0%), 5 subjects had chronicity of < 1 year, 4 subjects had 3-4 years chronicity (20.0%), 2 subjects had chronicity of 4-5 years, 2 subjects had chronicity of 2-3 years and 1 subject had chronicity of 1-2 years (5.0%).

Chronicity	Group A	Group B	Total
>5 years	3 (15.0%)	6 (30.0%)	9 (22.5%)
4 - 5 years	4 (20.0%)	2 (10.0%)	6 (15.0%)
3-4 years	2 (10.0%)	4 (20.0%)	6 (15.0%)
2-3 years	5(25.0%)	2 (10.0%)	7 (17.5%)
1-2 years	2 (10.0%)	1 (5.0%)	3 (7.5%)
< 1 year	4 (20.0%)	5 (25.0%)	9 (22.5%)
Total	20	20	40

BMI

In Group A, maximum number of subjects belonged to normal BMI (16 in number i.e., 80.0%) and 3 subjects were overweight (10.0%) and 1 subject was obese (5.0%).

In Group B, maximum number of subjects belonged to normal BMI (15 in number i.e., 75.0%), 3 subjects were overweight (10.0%) and 2 subject was obese (5.0%).

BMI	Group A	Group B	Total
Normal BMI (18.5-24.9)	16 (80.0%)	15 (75.0%)	31 (77.5%)
Overweight (25-29.9)	3 (15.0%)	3 (15.0%)	6 (15.0%)
Obese (above 30)	1 (5.0%)	2 (10.0%)	3 (7.5%)
Total	20	20	40

Nidana

Aharaja Nidana

In Group A, 12 subjects (60.0%) had *Vishamashana*, 6 subjects (30.0%) had *Akalabhojana* and 2 subjects (10.0%) had *Adhyashana*.

In Group B, 16 subjects (80.0%) had *Vishamashana*, 3 subjects (15.0%) had *Akalabhojana* and 1 subject (5.0%) had *Adhyashana*.

Nidana	Group A	Group B	Total
Vishamana	12 (60.0%)	16 (80.0%)	28 (70.0%)
Akalabhojana	6 (30.0%)	3 (15.0%)	9 (22.5%)
Adhyashana	2 (10.0%)	1 (5.0%)	3 (7.5%)
Total	20	20	40

Viharaja Nidana

In Group A, 17 subjects (85.0%) had *Dukhashayya*, 15 subjects (75.0%) had *Dukhasana*, 12 subjects (60.0%) had *Atiyaana*, 8 subjects (40.0%) had *Bharaharana*, 6 subjects (30.0%) had *Divaswapna*, 6 subjects (30.0%) had *Vegadharana* and 3 subjects (15.0%) had *Ratrijagarana*.

In Group B, 15 subjects (75.0%) had *Dukhashayya*, 15 subjects (75.0%) had *Dukhasana*, 14 subjects (70.0%) had *Atiyaana*, 11 subjects (55.0%) had *Bharaharana*, 6 subjects (30.0%) had *Divaswapna*, 5 subjects (25.0%)

had Veghadharana and 4 subjects (20.0%) had Ratrijagarana.

Nidana	Group A	Group B	Total
Dukhashyya	17 (85.0%)	15 (75.0%)	32 (80.0%)
Dukhaasana	15 (75.0%)	15 (75.0%)	30 (75.0%)
Atiyaana	12 (60.0%)	14 (70.0%)	26 (65.0%)
Bharaharana	8 (40.0%)	11 (55.0%)	19 (47.5%)
Diwaswapna	6 (30.0%)	6 (30.0%)	12 (30.0%)
Vegadharana	6 (30.0%)	5 (25.0%)	11 (27.5%)
Ratrijagarana	3 (15.0%)	4 (20.0%)	7 (17.5%)

Prakruti

In Group A, maximum number of subjects had *Vatakapha Prakruti* (13 in number i.e., 65.0%), 6 subjects (30.0%) had *Vatapitta Prakruti*, and 1 subject (5.0%) has *Pittakapha Prakruti*.

In Group B, maximum number of subjects had *Vatakapha Prakruti* (11 in number i.e., 55.0%), 6 subjects (30.0%) had *Vatapitta Prakruti*, and 3 subjects (15.0%) have *Pittakapha Prakruti*.

Prakruti	Group A	Group B	Total
Vatakapha	13 (65.0%)	11 (55.0%)	24 (60.0%)
Vatapitta	6 (30.0%)	6 (30.0%)	12(30.0%)
Pittakapha	1 (5.0%)	3 (15.0%)	4 (10.0%)

Vaya

In Group A, 13 subjects (65.0%) were of *Madhyama Vaya* and 7 subjects (35.0%) were *Vridha*.

In Group B, 13 subjects (65.0%) were of *Madhyama Vaya* and 7 subjects (35.0%) were *Vridha*.

Vaya	Group A	Group B	Total
Madhyama	13 (65.0%)	13 (65.0%)	26 (65.0%)
Vridha	7 (35.0%)	7 (35.0%)	14 (35.0%)

DISCUSSION

Contemporary sciences consider several factors that increase a person's chances of developing osteoarthritis. These include:

Heredity: Several Osteoarthritis are related to alterations in HLA-A1B8,^[9] certain haplotypes of HLA-B8,^[10] and with diverse isoforms of α 1 antitrypsin,^[9] however not all studies show the same association.^[11] These genes are responsible for making cartilage. This causes defective cartilage, which leads to more rapid deterioration of joints. People born with joint abnormalities are more likely to develop osteoarthritis, and those born with an abnormality of the spine (such as scoliosis or curvature of the spine) are more likely to develop osteoarthritis of the spine.

Obesity: Obesity increases the risk for osteoarthritis of the knee, hip, and spine. The increase in the prevalence of OA is directly attributable to the rise in obesity. Obesity is characterized not only by excessive joint loading, but also by an abnormal lipid profile: dyslipidaemia. Obesity-related dyslipidaemia is characterized by high plasma levels of TGs, low levels of HDL cholesterol (HDL-c), often slightly increased levels of LDL cholesterol (LDL-c), and increased levels of FFAs.^[12] Moreover, HDL function is impaired in obesity.^[13] Various studies have shown that lipid metabolism can play a role in OA. High serum cholesterol is associated with generalized OA, indicating that cholesterol might be a systemic risk factor for OA.^[14] Hence maintaining ideal weight or losing excess weight may help prevent osteoarthritis of these areas and decrease the rate of progression, once osteoarthritis is established.

Injury: Individuals who sustain a joint injury are known to be at substantially increased risk of developing OA compared with uninjured persons. Osteoarthritis that develops after joint injury is deemed posttraumatic OA (PTOA). Biomechanical changes in ACL-deficient and -reconstructed individuals may change the regions where tibiofemoral joint contact occurs, thereby loading areas of cartilage that were previously unloaded and decreasing loads to areas of cartilage normally experiencing higher loads during weight bearing.^[15] Similar biomechanical alterations have been observed after meniscectomy that may contribute to PTOA development.^[16]

Menopause: With menopause, women enter an estrogen deficient phase in their lives, which accelerates the ageing process. There is increasing evidence that estrogens fulfill a relevant role in maintaining the homeostasis of articular tissues and, hence, of the joint itself. The dramatic rise in OA prevalence among postmenopausal women, which is associated with the presence of estrogen receptors (ERs) in joint tissues,^[17] suggests a link between OA and loss of ovarian function. This association indicates a potential protective role for estrogens against the development of OA.

Failure of estrogen production at menopause is associated with a relevant loss of muscle mass and, therefore, significant impairment of muscle performance and functional capacity¹⁸. Diminished strength of the quadriceps in women but not men predict knee OA,^[19] and peri- and postmenopausal women also seem to have less lean body mass when compared with premenopausal women.^[20] In addition, varus-valgus laxity has more frequently been described in women than in men.^[21]

Wnt5b and other genes involved in osteoclast function are differentially expressed between male and female OA

bone.^[22] Furthermore, aggrecan production, as well as SOX9, type II collagen and parathyroid hormone-related protein mRNA expression was inhibited in sclerotic but not non-sclerotic osteoblasts (OBs), while expression of matrix metalloproteinases MMP-3 and MMP-13 and osteoblast-specific factor 1 by human OA chondrocytes was augmented in a co-culture system. Thus, sclerotic osteoarthritic subchondral OBs may contribute to cartilage degradation and chondrocyte hypertrophy.^[23]

Joint overuse: Overuse of joints increases the risk of developing osteoarthritis. For example, people in jobs requiring repeated bending of the knee are at increased risk for developing osteoarthritis of the knee.

Other diseases: People with rheumatoid arthritis, the second most common type of arthritis, are more likely to develop osteoarthritis. In addition, certain rare conditions, such as iron overload or excess growth hormone, increase the chance of developing OA.

In Ayurveda

The general *Nidana*,^[24-29] stated in the classics and other *Nidana* which can be considered are.

Aharaja Nidana: Anna which is Ruksha, Sheeta, Alpa and Laghu; Rasa like Amla, Lavana, Katu; Kshara, Sushka Shaka, Sushka Mamsa, Tila, Palala, Pishtanna, Viruda, Navasuka, Navasamidhanya, Virudhahara, Asatmya Ahara, Abhishyanda Bhojana, Klinna Bhojana, Guru Bhojana, Puti Bhojana and Paryushita Bhojana. All these factors along with Gramyahara lead to Vata Pradhana Dosha Prakopa (Samana Vata, Pacaka Pitta and Kledaka Kapha) especially that of Samana Vata. The derangement in Samana Vata gets associated with Vyana Vata which carries its effect all over the body.

Viharaja Nidana: Vyavaya, Atiprajakara, Langana, Plavana, Atiadva, Ativyayama, Atichesta, Dukkhasayyasana, Diwaswapna, Vegasandharana, Ama, Abhighata, Abhojana, Marmabhigata, Yanapatamsana, Vishamasana, Adhyasana, Strinitya, Madyanitya and Vishama Vyayama. These vitiate the Vyana Vata, Pacaka Pitta and Sleshaka Kapha which leads to its accommodation at the Khavaigunya Sthana (Janu Sandhi), if any.

Anya

Vardhakya – Old age is dominanted by *Vata* which is further aggravated due to *Dhatu Kshaya* that takes place as the age advances. As mentioned in *Susruta Samhitta*, though *Jara* starts after 60 years, degenerative changes are mentioned to commence after 40 years which is termed as *Parihani Avastha* of *Madhyama Vaya* wherein the individual develops the tendency to suffer from *Vata Vyadhi* on exposure to *Vata Prakopaka Nidana*.

Bharaharana – Repetitive use of joints through any activity may lead to excessive strain leading to erosion and joint damage.

Sthoulya – Sthoulya is a prominant cause in the present scenario for Janu Sandhigata Vata. Even in Susruta Samhitta, Commentator Dalhana opines Stoulya to be a cause for Vata Vikaras. Obese persons have a high risk of developing Osteoarthritis due to overload on the weight bearing joints, mainly the Knee Joint. The whole weight of the obese patient is carried by the knee and any physical activity causes increased strain on the supporting structures of the knee joint and may reduce the joint space leading to Osteoarthritis.

CONCLUSION

Understanding the various Nidana in reverence to its present scenario is necessary in defining the treatment. This helps in defining the diagnosis and treatment and also in improving the quality of life in an individual.

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