

ANATOMICAL CHANGES IN JANUSANDHIGATA VATA W.S.R TO KNEE OSTEOARTHRITIS

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ABSTRACT

Asthi (bone) is the main seat of *vata dosha* and *sandhi* (joint) is the main seat of *kapha dosha*. *Vata dosha* present in the body responsible for different body movements like *prasarana* (extension) and *akunchana* (flexion) of *sandhi*, whereas *Shleshak kapha* present in *Sandhi* is meant for nutrition, protection and to minimize friction during movement. The *shleshak kapha* is white, *pichhil* (slimy), *snigdha* (unctuousness), and *sthira* (immobile) in nature. These characters are similar with the characters of synovial fluid present within the joints. Due to aggravation of *vata dosha* in *Sandhi* result a diseased condition known as “*Sandhigat vata*”. When *Janu sandhi* is affected the condition is known as “*Janusandhigata Vata*”, here various symptoms are produced like *vatapurna driti sparsa* (feeling of air filled balloon), *prasarana akunchane vedana* (pain during extension and flexion), *shopha* (swelling), *stabdata* (stiffness), *sandhishool* (pain in joints), and *sandhishosha* (atrophy or degeneration of bone) etc. These symptoms are somehow similar to the symptoms of “knee Osteoarthritis”. Osteoarthritis is a joint failure, where all structures of a joint have undergoes pathological changes like hyaline cartilage loss, sclerosis of subchondral bony plate, growth of osteophytes at the joint margin and weakness of the muscles bridging the joint. Whenever Knee joint is affected called as “knee osteoarthritis”. Due to immobilization, misuse and overuse of joint there is an injurious static compression state, can stimulate the reduction of proteoglycans from the superficial zone (layer) of articular cartilage and damage the collagen network. This condition leads to fibrillation/damage of hyaline cartilage present in joint forming surfaces of bones. Chondrocytes present in the articular cartilage can respond to direct biomechanical perturbation by modulating synthetic activity or by increasing the production of inflammatory cytokines. On the other hand, underlying subchondral bone is affected by altered bone remodeling processes leading to sclerosis. Osteophytes are also produced like this. In Osteoarthritis the viscosity of synovial fluid is decreased because of decreased production and polymerization of hyaluronic acid, which is probably the major lubricant of synovial fluid due to water binding ability. This condition greatly affects the joint movements followed by pain and crepitus.

Keywords: *Janu Sandhi, Janu Sandhigata Vata*, cartilage loss, subchondral sclerosis.

INTRODUCTION

Janu Sandhi

The term “*Janu*” is described as one amongst two hundred ten (210) *Sandhi*¹ as well as among hundred seven (107) *Marma*², situated in *adhashakha* (lower extremity) in between *Uru* (thigh) and *Jangha*³ (leg) in human body. *Janu Sandhi* is formed by three *asthi* (bone) like *urbarthi* (femur), *antah prakosthathi* (tibia) and *bahir prakothasthi* (fibula). It is a type of kora variety of *Sandhi*. *Kora* denotes shape of hinges of door.

Janu marma is described under *Sandhi marma*⁴, where involvement of multiple joints seen. *Janu marma* is also classified under *vaikalyakar marma*⁵ because injury to this leads to *vaikalyata* (disability) and *khanjata*⁶ (lameness), and injury to its nearer tissue result *ruja*⁷ (pain). It measures about three (3) *angula* (finger) length⁸. *Janu marma* is made up of predominantly of *soumya mahabhuta*⁹, so its injury is not fatal. Anatomically it can be compared with Knee joint.

Sandhi is defined as “*Asthi sanyog sthanam*”¹⁰ that means *sandhi* is formed by junction of two or more *asthi* (bones). *Vyana Vayu*¹¹ present in all *sandhi* responsible for various movements of joint like *prasarana* (extension) and *akunchana* (flexion). *Shleshak kapha*¹² situated in internal structure of a *Sandhi* meant for lubrication to minimize friction during movements, give nutrition, protection and to stabilize the Joint. *Janu sandhi* can be compared with Knee joint.

Anatomy of knee joint

Knee Joint is the strongest weight bearing joint of body, its various movements are responsible for day to day activities like walking, running, and sitting etc. It is a synovial hinge joint formed between three bones like femur, tibia and patella. These bones make the knee by three joints that are two condylar joints between the condyles of femur and tibia, and one saddle joint between femur and patella. The joint forming surfaces of each bone are covered by a thin layer of hyaline cartilage, which give smooth surface and protect from damage called as articular

cartilage. The other supporting structures of Knee joint are fibrous capsule, ligamentum patellae, tibial collateral/medial ligament, fibular collateral/lateral ligament, oblique and arcuate popliteal ligament, anterior and posterior cruciate ligament, medial and lateral menisci, transverse ligament and bursae¹³.

Janusandhigata Vata

There is nothing exactly like *Janusandhigata Vata* described in *Ayurvedic* literature. But *Sandhigata vata* is described as one of the *Vata vyadhi*¹⁴. *Vayu mahabhuta* feels the spaces within the *asthi dhatu*. There will be subsequently increase in the amount of *Vata dosha* and decrease in the amount of *kapha dosha* in old age. The aggravated *vata* diminishes the moisture of *asthi dhata* leading to “*kha vaigunya*”. On the other aspect all *dhatu*s (*rasa, rakta, mansa, meda* etc.) undergoes degeneration with increasing age. As *kapha* is stated as *mala* of *rasa dhatu*¹⁵, there is also reduction in normal quantity of *kapha dosha* in body as well as from the joints. All above factors indicate that aggravated *vata dosha* and diminished *kapha dosha* combinedly affect the bony joints (*sandhi*) lead to *Sandhiagata vata*. Whenever *Janusandhi* is affected it is named as *Janusandhigata vata*, because it is a most complex weight bearing joint.

During progression of *Janusandhiagata vata* (*sandhigata vata*) the symptoms produced like *sandhi shosha* and *hanti sandhi*¹⁶. Both symptoms indicate dehydrated or damaged state of cartilage/bone. There is also a decreased amount of *shleshak kapha* in *sandhi* which hinders its nourishment and lubrication, resulting pain and crepitation during movements, the symptoms produced like *sandhi soola*¹⁷ refers to pain, and *atopa*¹⁸ denotes crepitation etc. Other symptoms described by different *Acharyas* are *vatapurna dritisparsa*¹⁹ and *shopha*²⁰ somehow indicate there is inflammation. All the above symptoms are mostly equal to the symptoms of knee Osteoarthritis.

Comparable symptoms

Symptoms	<i>Janu sandhigata Vata</i>	Knee Osteoarthritis
Pain and Tenderness	✓	✓
Joint stiffness	✓	✓
Loss of flexibility	✓	✓
Grating sensation	✓	✓
Bone spurs	X	✓

Knee osteoarthritis

Osteoarthritis is most common form of arthritis, affecting large section of population with significant disability and impaired quality of life. It affects mainly mild, moderate and severe degree. OA is joint failure, a disease in which all structures of the joint have undergoes pathological changes, that is hyaline cartilage loss, and sclerosis of subchondral bony plate by outgrowth of osteophytes at the joint margin by stretching of the articular capsule, by mild synovitis in many affected joints and by muscles bridging the joint²¹. Frequently affected sites include hips, knees, hand and spines. When considering the pathogenesis of Osteoarthritis, it is important to review the contribution of bone in addition to cartilage and synovium. The Knee is very commonly affected because it is a weight bearing joint. The condition is known as “Knee osteoarthritis”.

Causes

Etiology of Osteoarthritis is multifactorial and broadly divided into genetic and nongenetic factors. Nongenetic factors like age (12% of aged ≥ 60 yrs, and 6% of aged ≥ 30 yrs²⁴), gender (females are more prone), obesity, inactive life style, joint injury and occupation. Genetic factors like hereditary and altered gene expression pattern of cartilage and subchondral bone tissue.

Anatomical changes in knee Osteoarthritis

The articular surface plays a vital role in load transfer across joint and there is enough evidence that conditions leading to increased load transfer and/or altered patterns of load distribution can accelerate initiation and progression of OA. Anatomical progression of OA can be represented by structural alteration in articular cartilage leading

to narrowing of the joint space. On the other hand, underlying subchondral bone is affected by altered bone remodeling processes leading to sclerosis and formation of osteophytes. In joint, the close physical relationship between the cartilage and subchondral bone has introduced the concept of biochemical and molecular crosstalk across the affected region. In knee OA, chondrocytes can respond to direct biomechanical alarm by modulating synthetic activity or by increasing the production of inflammatory cytokines²².

Onset of progression of Knee osteoarthritis

A diarthrodial joint has articular cartilage, subchondral bone, synovial membrane, ligaments, menisci and bursae. The articular cartilage histologically made up of 4 layers (zones) like superficial, middle, deep and calcified zone. The superficial layer is composed of a highly structured network of extracellular matrix proteins, proteoglycans, collagen and non-collagenous proteins and it act to maintain high water content. Middle layer composed of randomly concerned collagen fibers within which larger chondrocytes are present. The deep stratum is arranged in vertical columns separated by collagenous fibrils. The last layer is composed of calcified cartilage within which hypertrophic chondrocytes are situated. Between the calcified zone and the deep zone there is a thick bundle of collagen fibrils called as tidemark. The subchondral bone is situated just under the articular cartilage. It is the zone of epiphyseal bone consists of two layers deeper one is subchondral bone plate and underlying trabecular bone²².

Alteration in Cartilage

Articular chondrocytes and the subchondral bone cells have capacity to receive persistent acute as well as chronic pressure and react so. Generally biomechanical stress and strain are necessary for joint homeostasis. At the time of immobilization, misuse and overuse of joint, there is rapid loss of proteoglycans in joints like in injurious static compression state can stimulate the reduction of proteoglycans from its superficial zone and damage the collagen network. These events decrease the synthesis of cartilage matrix proteins. The Cartilage cannot withstand high tension at the edges of the joint contact regions for a long time as a result the cartilage is at a state of unbearable condition leads to fibrillation. Chondrocytes of adult cartilage matrix, having a very low metabolic activity, requires low oxygen and less blood supply. Chondrocytes have receptors to respond against biomechanical changes in the surrounding cartilage matrix as well as extrinsic growth factors, cytokines and other inflammatory mediators. Several integrins (function-attachment of the cell to the ECM and signal transformation from cell to ECM) on activation can stimulate the production of matrix degrading proteinases and inflammatory cytokines and chemokines in chondrocytes. Significant modulation of chondrocytes by increasing synthesis of fibronectin, type 2 collagen and aggrecan (responsible for maintenance of structure of a cartilage) just after onset of disease suggests that articular chondrocytes try to repair the damaged matrix. This repair process eventually appears to fail leading to irreversible cartilage generation²².

Changes in Synovial fluid

Synovial fluid present within the synovial membrane of joints made up of hyaluronic acid, lubricin, proteinases and collagenases. The hyaluronic acid forms very viscous solutions, which are probable the major lubricants in synovial joints because of its ability to bind water. In knee osteoarthritis the viscosity of synovial fluid is decreased because of

decreased production and polymerization of hyaluronic acid. Such less viscous fluid hinders the free movements of knee joint followed by pain and crepitus.

CONCLUSION

Sandhi (joint) is defined as the junction place of two or more *asthi* (bone). *Asthi* is the major seat of *Vatadosha* and *sandhi* is the major seat of *kapha dosha* (*slesak kapha*). Either due to aggravation of *vata dosha* in old age or due to intake of *vatic ahara vihar* (vata aggravating diet and regimen), there is aggravation of *Vata dosha* which reduces *Sneha* (moisture) of *asthi dhatu* at joint places. This condition leads to *kha vaigunya*, which ultimately result manifestation of a disease called *Sandhigata vata*. *Janu sandhi* is very commonly affected by *Sandhigata vata* because of its complexity, called as *Janusandhigata Vata*. The symptoms of *Janusandhigata vata* are similar to the symptoms of knee osteoarthritis of modern medical science. Broadly in Osteoarthritis there is degeneration of hyaline cartilage because of decrease in the amount of proteoglycan from its superficial zone. Same as in case of *janusandhigata vata* there will be the reduction in *sneha* of *asthidhatu* due to increased *rukshya* character of *Vata dosha*, which produces symptoms like *sandhishosha* and *hanti sandhi* which implies there is damage of *taruasthi* (cartilage). There is also reduction of hyaluronic acid, major constituent of synovial fluid within the synovial cavity in knee OA which hinders its viscosity. This may correlate with the decreased state of *sleshak kapha* (*kapha* is basically derived from *rasa dhatu*). This condition is suggestive of *dhatukshyaya* in old age. This decreased state of *shleshak kapha* manifests the symptoms like *prasarana akunchane vedana*, and *sandhi shoola* which suggests pain during movement of joints. Other symptoms like *atopa*, suggests there is crepitation during joint movement etc. The above discussion can be

concluded that the knee osteoarthritis is nothing but the *Janu sandhigata vata*.

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