

INTERVERTEBRAL DISC PATHOLOGY AND AYURVEDA**Dr. R. Nithin Krishnan¹, Dr. S. Gopi Krishna²**¹PG Scholar, ²Associate ProfessorPG studies Department of Roganidana, ShriDharmasthalaManjunatheshwara
College of Ayurveda, Hassan, Karnataka, India**ABSTRACT**

Intervertebral discs are fibrous cartilage located between adjacent vertebrae in the vertebral column. They act as shock absorbers between each of the vertebrae in the spinal column by keeping the vertebrae separated when there is impact from activity. They also serve to protect the nerve that runs down the middle of the spine to intervertebral discs. But, evolving life style without caring spine resulted in significant damage to the spine including the intervertebral discs ending in deterioration, herniation or other dysfunction of the same. 60 to 70% prevalence of low back pain arise out of intervertebral disc pathologies, peaking between the age of 35 and 55. The symptoms of intervertebral disc disorders such as low back pain is the ready cause of activity limitation and work absence throughout much of the world, imposing a high economic burden on individuals, families, community industries and governments. This article covers different dimensions of intervertebral disc pathology with reference to its structure and integrating its understanding with the principles of Ayurveda.

Keywords: Intervertebral disc, Pathology, Herniation, Ayurveda**INTRODUCTION**

An intervertebral disc or intervertebral fibrous cartilage lies between adjacent vertebrae in the vertebral column. Each disc forms a fibrous cartilaginous joint (a symphysis) to allow slight movement of the vertebra and act as a ligament to hold the vertebra together. Their role as shock absorbing in the spine is crucial. Intervertebral disc disorder is a condition that involves deterioration, herniation or other dysfunction of an intervertebral disc. The common etiologies ending in the spectrum of intervertebral disc disorders mainly include age related degeneration or induced by trauma or due to influence of both. Other etiologies may be charted as an incorrect weight bearing and heavy strain

(including pregnant women), deformation of the spine accelerating wear and tear, contact sports such as football and other games such as gymnastics, tennis, squash, swimming, skiing, etc.¹

It is difficult to estimate the incidence of intervertebral disc disorders manifesting into symptoms such as low back pain as the incidence of first ever episodes of low back pain is already high by early adulthood and symptoms tend to recur over time. The life time prevalence of non-specific (common low back pain) is estimated at 60% to 70% in industrialized countries. The prevalence rate for children and adolescents is lower than that seen in adults but is rising. Prevalence increases

and peaks between the age of 35 and 55. As the world population ages, low back pain will increase substantially due to the deterioration of the intervertebral discs in older people. The symptoms of intervertebral disc disorders such as low back pain is the ready cause of activity limitation and work absence throughout much of the world, imposing a high economic burden on individuals, families, community industries and governments².

General anatomy and mechanics of spine:

Vertebral column is an amazingly complex structure that must meet the seemingly contradictory demands of mobility and stability of the trunk and extremities, providing protection for the spinal cord. They are a chain of bones; connected by discs of deformable substance developed around and largely replaced the notochord. Notochordal vestiges occur in vertebrae of many fish, amphibians and reptiles and centrally in mammalian intervertebral discs. Vertebral column gives ten times more strength and support for carrying weight capacity than when it is absent.

Smaller functional unit of vertebra is called as the “mobile segment”. Each mobile segment constitutes two vertebrae with intervertebral disc and all the soft tissues that serve them together. There are 33 vertebrae which comprises of seven cervical vertebrae, twelve thoracic, five lumbar, five fusiform sacrum and four coccygeal vertebrae. The vertebral size increases from cervical to lumbar and decrease from sacral to coccygeal. They comprise of 23 intervertebral discs stacked between the vertebral bodies³.

The vertebrae can be differentiated into body, pedicles, laminae, transverse process and annulus fibrosus. Body of the vertebra helps to resist compressive forces and transmits compressive forces to vertebral

end plates. Pedicles of the vertebra transmit bending forces exerted by muscles attached to the spinous and transverse process to the vertebral bodies. Laminae resist and transmit forces that are transmitted from spinous and zygapophyseal articular processes to pedicles. It serves as attachment sites for muscles and ligaments. Transverse processes serve as attachment sites for muscles and ligaments. Spinous-process resists compression and transmits forces to laminae and serves as attachment sites for ligaments and muscles. Zygapophyseal facets resist shear compression forces to vertebral endplates and translate vertical compression forces into circumferential tensile forces in annulus fibrosus. Annulus fibrosus resists tensile, torsional and shear forces⁴.

Structure of intervertebral discs:

Intervertebral disc also known as intervertebral fibrocartilage lies between adjacent vertebrae in the vertebral column. Each disc forms a fibro cartilaginous joint or a symphysis, with the sole function of increasing available motion and transmitting load from one vertebral body to other. Therefore, size of the intervertebral disc depends upon the load of the disc and range of motion. The height of intervertebral discs varies from 3mm in the cervical region to 9mm in the lumbar region. The ratio of thickness of the disc to the vertebral body thickness determines the available motion from the joint. If more – then range of movement also will be more, if less then range also will be reduced. Therefore, inter vertebral disc thickness to the vertebral body thickness is more in cervical and lumbar but least in thoracic⁵.

Intervertebral disc consists of three components – nucleus pulposus, annular fibrosus and vertebral end plate. Nucleus pulposus is rich in water and proteoglycans. That is 70 to 90% water (depends on age

and time of day), 65% of dry weight constituted by proteoglycans which are hydrophilic. 15 to 20% dry weight by collagen, other constituents includes many cells, elastin, proteins, proteolytic enzymes, chondrocytes and other types of collagen. Nucleus pulposus has two types of collagen – Type 1 and type 2. Type 2 is predominant as it bears more amount compressive weight. Hence, nucleus pulposus can be frequently told as a balloon filled with water.

Annulus fibrosus carries 60 to 70% water, 50 to 60% dry weight collagen and 20% dry weight proteoglycans. Rest is elastins, fibroblasts and chondrocytes. Type 1 and 2 collagen fibers are present in the annular fibrosus, among them type 1 is predominant because of more tensile strength. It attaches to the epiphyseal ring by inverted T fibres⁶.

Vertebral end plate is 0.6mm to 1mm height covering the superior and inferior vertebral bodies encircled by ring apophysis. They cover the entire nucleus pulposus and annular fibrosus – but not the entire vertebra. Vertebral end plate made up of water, proteoglycans and collagen fibers, where water and proteoglycans are present proximal to nucleus pulposus, collagen fibers present distal to nucleus pulposus or near to proximal vertebral body. Vertebral end plate is made up of a hyaline cartilage – i.e. found mainly in young disks. Fibro cartilage is present closest to the nucleus pulposus. With increased age, gradually the “fibrocartilage” replaces “hyaline cartilage” reflects the need to tolerate high compressive force⁷.

The intervertebral discs do not get nourishment from any major arterial branches, instead metaphyseal arteries from a dense capillary plexus in the base of the end plate cartilage and subchondrial bone deep to the end plate; also supply the outer surfa-

ceof the annulus fibrosus. For rest of the annulus fibrosus and nucleus pulposus it gets nourishment by the process of diffusion through these sources. Vertebral nerve, supply to one third to half of annular fibrosus. Sinovertebral nerve, supply to peri – discal connective tissue specific ligaments associated with vertebral column⁸.

Ayurvedic understanding of intervertebral disc and its impact with ageing:

The skeletal tissues, cartilage and bone are essentially specialized connective tissues and consist of the same component cells embedded in a matrix permeated by a system of fibers. But, physically matrices of skeletal tissues differ from those of general connective tissues in being solidified. Generally bones are considered as *asthi* formed from *medodhatu*. The compactness is brought about by the action of the *usma* present in the *meda* acting itself upon the *prithvi*, *jala* and *vayumahabhoota*. This action gives rise to *kharatva* which result in formation of *asthi* in human beings⁹. In the physical body, *asthidhatu* is formed as *posaka* (unstable) *medodhatu* which flows into the *purishadharakala* and is digested by the *asthiagni*. The *purishadharakala* is the membrane that holds the *asthiagni*. *Purisha* means feces. The term is also used to describe the large intestine as in the *purishavahasrotas*. The *asthidhatu* is produced by the transformation of *meda* into a compact form. Here it provides an important clue regarding the relationship between health of the large intestine and that of the bones. The large intestine is the site of *vata dosha*. The close relationship between these two tissues reveals the susceptibility of the skeletal system to disorders associated with *vata dosha*. The main function of *asthidhatu* is to maintain erect posture and bearing weight followed by nourishment of *majjadhatu*.

In Ayurveda there are two types of *dhatu*, i.e. *sthayi* and *poshakadhatu*. *Asthi* or bone can be considered as *sthayidhatu* and not the *asthidhatu* completely. *Asthidhatu* exist in liquid form called as *poshakaasthi* that traverse through the *asthivahasrotas* and nourishes the *sthayiaasthidhatu*. From the contemporary science, the nutrients that are responsible for the nourishment of the bone tissue such as calcium, phosphorus, magnesium etc. can be considered as the *poshakaasthidhatu*. Overlooking embryological development of axial endo – skeleton, first a notochord and then a vertebral column, is the basic feature of chordate and their subphylum, the vertebrata, including mankind. A stiff but flexible axis, in bilaterally symmetrical animals that show an early tendency to elongation, prevents telescoping of the body during waves of contraction in successive segmental muscles to produce the sinuous movements, especially in the tail, which are the basic mode of locomotion in aquatic vertebrates. A chain of bones connected by discs of deformable substance develop around and largely replaced the notochord. However, notochordal vestiges occur in vertebrae of many fish, amphibians and reptiles and centrally in mammalian intervertebral discs. This replacement is repeated in early vertebrate embryo¹⁰. Hence, intervertebral disc can be included under *asthidhatu*. *Asthidhatu* can precisely be termed as the edifice bars, on the systematic frame work of which human being stand. Therefore, *asthidhatu* facilitates *dharana*¹¹. Here, *asthidhatu* can be understood as both vertebra as well as intervertebral disc. Based on the principal of *ahrayaashrayibhava*, *vatadosha* is inversely related to *asthidhatu*. *Vatadosha* increases physiologically with escalating age or in the old age¹². Therefore age induced degenerative changes to intervertebral discs are

inevitable. There will be a significant reduction of *kaphadosha* with predominant *apmahabhoota* due to the *rookshaguna*¹³ induced by *vatadosha* predominant with *akasha* and *vayumahabhoota*¹⁴. Degenerative type of pathological conditions in human body can be understood under the category of *vata vyadhi*. One among the condition affecting the *sandhi* by *vata dosha* – *sandhigata vata* is included under it. The term *gata* is derived from the root “*gam*” which means gone to, arrived at, situated in, directed to, hence, it can be revealed that the word *gata* is related with site (here *asthi* and *sandi*), in which vitiated *vata dosha* get lodged and manifest symptoms of *asthi* and *sandhigata vata*. In this condition due to exposure to etiological factors, aggravated *vata* accumulates in *rikta srotas* to give rise to various generalized and localized diseases. Here, in this context *vata dosha* is vitiated in vertebral joints (especially intervertebral disc), it leads to *sandhigata vata*. One among the common manifestation of intervertebral disc pathology is *gridrasi*. *Gridrasi* is a disease having its origin in *pakwashaya* and seat as *sphik* and *kati* i.e. lumbar spine. Cardinal features include distribution of pain that begins in the back and radiates through the posterior aspect of the thigh and calf to outer foot. Herniation and degeneration of the disc are the commonest causes.

Over the life span, the vertebral column is exposed to recurrent loads that change the morphology of the column. However, normal age related changes also occur in the structures of the vertebral column. The vertebral bones undergo changes in the amount and form the trabeculae. The numbers of both horizontal and vertical trabeculae decrease with age, and the horizontal trabeculae become significantly thinner. This loss can decrease the loads that the

vertebrae are able to withstand before failure. Each of the structures of the intervertebral disc undergoes changes that include loss of the amount of proteoglycans and change in the specific type of proteoglycan, with resultant loss of water content. In addition, there is an increase of collagen in these structures and loss of elastin. This results in a loss of the ability for the disc to transfer loads from one vertebra to another as the swelling ability of the nucleus decrease. The overall disc height will also decrease somewhat. The vascularization of the disc also changes. *In utero*, blood vessels can be demonstrated within the fibers of the annulus fibrosus. By the end of the second year of life, these have predominantly degenerated. Thus, the disc relies on the diffusion of nutrients through the vertebral end plate. The vertebral endplate, with ageing, gradually becomes more collagenous, and the process of diffusion is hindered. The fibers of the annulus fibrosus in the cervical spine of adults normally demonstrate lateral fissures that subdivide the disc into two halves at the uncovertebral joints. These fissures can first be observed in children at approximately 9 years of age. After formation of these fissures, joint pseudocapsules develop with vascularized synovial folds. The formation of these fissures appears to be load – related and is located predominantly in the regions of C3 to C5¹⁵.

With large and or repetitive loads, further changes occur in the discs. The discs demonstrate a dramatic decrease in their elasticity and proteoglycans. Eventually, the intervertebral disc will become so dry that it begins to crumble. In the lumbar region, the inner layers of the annulus fibrosus begin to buckle outward, and the lamellae separate. Fissures and tears can occur within the annular fibers, which can decrease the ability of the disc to provide

stiffness during movement. The vertebral end plates may become ossified. The adjacent spongy bone of the vertebral body can begin to sclerose. On occasion, blood vessels grow into the discs and trigger ossification. The disc can prolapse or protrude as a result of the pressure of the nucleus and the lack of ability of the annulus fibrosus to sustain it. Schmorl's nodes are formed when the nuclear material prolapses through the vertebral end plate and into the cancellous bone of the vertebra. This material may cause an autoimmune response when it comes in contact with the blood supply in the cancellous bone. This is typically labeled as degenerative disc diseases. In this case of degenerative disease, there is a more substantial loss of disc height, which causes all ligaments to be placed on slack. The ligamentous pre – stress normally provided by the ligamentum flavum will decrease, which in turn will impair spinal stiffness. This can also allow the ligament to buckle on itself with movement, potentially compressing the spinal cord. In addition, the ligamentum flavum begins to calcify with age, and this occasionally leads to ossification, which can also potentially cause compression of the spinal nerve in the vicinity of the zygapophyseal joints or the spinal cord within the canal¹⁶.

The zygapophyseal joints can also demonstrate age related changes and eventual degradation. Some authors have argued that these changes in the zygapophyseal joints must be secondary to disc degeneration, as a substantial amount of weight – bearing through these joints must occur to cause deterioration. This increase in weight – bearing may be due to the loss of disc height. However, this is always not the case. There have been descriptions of degenerative zygapophyseal joints without disc degeneration. The mechanism of this

is not as well understood. If, however, the discs degenerate and a substantive decrease in height occur, what follows is hypermobility as a result of slackened capsules and longitudinal ligaments. The vertebra may also slip forward or backward on the vertebra below (listhesis or retrolisthesis). There will be excessive shear forces generated, and the zygapophyseal joints will also become subject to more load – bearing. The result of these changes becomes the same as with what happens to the larger joints of the extremities: damage of the cartilage, including fissures and cysts, and osteophyte formation. These changes can lead to localized pain or pressure on spinal nerves or the central canal or, in the cervical region, compression of the vertebral artery in the transverse foramen.

The uncovertebral joints are frequent sites for age – related and degenerative changes. Osteophytes on the uncinated processes occur predominantly in the lower segments C5/ C6 or C6/ C7. The motion of lateral bending becomes extremely limited when these osteophytes manifest¹⁷.

Vatadoshaprakopakarana and lakshana with reference to *asthi* (vertebra and intervertebral disc):

Consumption of etiologies like *tikta* (bitter), *kashaya* (astringent), *katurasa* (pungent), *ruksha* (dry), *laghu* (light), *sheeta* (cold) food, *anasana* (abstaining from food completely), *vishamasana* (irregular intake of food), *adhyasana* (excess intake of food), *parinate cha anne* (after digestion), *aparane* (evening), *ativyayama* (excess physical activity), *prapatana* (fall), *angabhanga* (injury), *kshaya* (wasting), *atishucha* (excess cleansing), *adhyayana* (continuous reading), *langhana* (fasting), *plavana* (swimming), *jagarana* (awake during night), *bhashana* (excess speech), *vegodeerana – dharana* (voluntary

initiation and suppression of urges), *bhaya* (fear), *shoka* (worry), *chinta* (excess mental activities), etc. lead to *vatadoshaprakopa*; further resulting in a spectrum of disorders¹⁸. The commonest of the etiologies include a history of trauma as twisting of the spine or carrying heavy objects or exposure to cold. Trauma can induce injury to *snayu* (ligaments), *sira* (vasculature), *kandara* (tendon), *asthi* (bone) and *sandhi* (joint). In severe cases, it may cause severe intensity to these structures causing *sandhi chyuti* (dislocation) or *asthi bhagna* (fracture). Classical literature mentions different diseases affecting *kati pradasha* (low back – where most IVD pathologies are manifesting). They mainly present with pain (local or radiating) or stiffness as one of the presenting symptoms in *kati pradasha*. To be precise, intake of *katu rasa* results in *rookshana*, intake of *tikta rasa* lead to *shoshana* of *dhatu*s and *upadhatu*s. *Kashaya rasa* consumption in major proportions lead to *karshana*. Consuming reduced amount of food cause *uttarottara dhatu kshaya*, leading to *asthi majja kshaya* and *shosha* of *upadhatu*. Continuous travelling has the potential to produce minor injury or *shithilata* in *dhatu*, *upadhatu* and *sandhi* leading to various disorders including intervertebral disc disorders.

From the reference of Ashtangahrudaya, *sutrasthana*; aggravated *vayu* possess the following characteristic features, they are the following: a) *sramsas* (drooping down) b) *vyasa* (dilation) c) *vyatha* (cutting pain) d) *svapa* (loss of sensation) e) *sada* (weakness) f) *ruk* (continuous pain) g) *toda* (pricking pain) h) *bheda* (splitting pain) i) *sanga* (blockage) h) *angabhanga* (crushing pain) i) *sankocha* (contraction) j) *varta* (twisting) k) *harshana* (tingling) l) *tarshana* (thirst) m) *kampa* (tremor) n) *parushya* (roughness) o) *soushiryas* (cavitation) p)

shosha (dryness) q) *spandana* (pulsations) r) *veshtana* (covering) s) *stambha* (stiffness) t) *kashyarasata* (feeling the astringent taste in this mouth) u) *shyavavarna* (bluish hue) v) *arunavarna* (reddish hue)¹⁹.

The above mentioned characteristic features with respect to the *vataprakopalakshanas* can also be extrapolated with reference to *asthi* (both vertebra and intervertebral disc – a part of mobile segment) as following: *Balabhrmsha*– Laxity of adjacent structures of mobile segments or laxity of annulus fibrosus, *Vartana* – Replacement of proteoglycans by collagen fibres, *Shosha&Soushrya* – Disappearance or degeneration of horizontal and vertical trabeculae, *Tarshanam* – Intervertebral disc desiccation or dehydration, *Angabhanga* – Friction of mobile segments, *Parushya* – Drying or degenerative changes of intervertebral disc due to reduced proteoglycans, *Sankocha* – Reduced disc spacing or compressed or compromised vasculature, *Spandana*, *Veshtana* and *Stambhana* – Degree of restriction or stiffness, *Vyadha*, *Ruk*, *Toda* and *Bheda* – Neurogenic or neuropathic pain due to stimulus to various adjacent structures of mobile segments and itself, *Svapa* – Compromised neural functioning (numbness), *Sanga* – Impact of neural functioning deficit over other systems including evacuation of bladder, bowel, etc.

Classification of disc pathology:

Normal Disc: Disc appears to be normal in development and there are no signs of disease, trauma or aging. **Annular tear - Disc herniation:** **Annular tears** are also called annular fissures and are separations between annular fibres, avulsion of fibres from their vertebral body insertions, or breaks through fibres involving one or many layers of the annular insertions, or breaks through fibres involving one or

many layers of the annular lamellae. The terms 'tear' or 'fissure' does not imply that the lesion is consequent to trauma. In case of a traumatic event the term 'rupture' is appropriate. **Disc herniation:** is displacement of disc material beyond the limits of the intervertebral disc space. A herniated disc can be contained (covered by outer annulus fibrosus) or uncontained.

Focal herniation: is a herniated disc less than 90° of the disc circumference.

Broad based herniation: is a herniated disc in between 90°-180° of the disc circumference. **Bulging Disc:** is the presence of disc tissue 'circumferentially' (180°-360°) beyond the edges of the ring apophyses and is not considered a form of herniation.

The nucleus pulposus is covered by the intact annulus fibrosus. **Protrusion:** indicates that the distance between the edges of the disc herniation is less than the distance between the edges of the base. **Extrusion:** is present when the distance between the edges of the disc material is greater than the distance at the base. **Migration:** indicates displacement of disc material away from the site of extrusion, regardless of whether sequestered or not. **Sequestration:** is used to indicate that the displaced disc material has lost completely any continuity with the parent disc. **Axial localizations -Central or medial (orange):** Since the PLL (posterior longitudinal ligament) is at its thickest in this region, the disc usually herniates slightly to the left or right of this central zone. **Para median or lateral recess (blue):** Because the PLL is not as thick in this region, this is the prime region for disc herniation to occur in. **Foraminal or subarticular (red):** It is rare for a disc to herniate into the intervertebral foramen. Only 5% to 10% of all disc herniation occur here or farther out.

When herniation do occur in this zone, they are often very troublesome for the patient.

This is because a super-delicate neural structure called the 'Dorsal Root Ganglion' (DRG) lives in this zone resulting in severe pain, sciatica and nerve cell damage.

Extraforaminal or lateral (green): Disc herniation in this region is uncommon²⁰.

Recent advances in understanding IVD pathology:

It has been recently discovered that, the spinal discs have 24 hour body clock that can maintain its balance in structure and function; when in imbalance resulted in pain. The cells from IVD were taken and tagged with bioluminescent genes which “pulse” in time with the circadian rhythms that govern the body’s 24 hour clock. The cells within the discs had their own clock. The cells within the discs had their own “clocks” which were regulated by temperature. When they designed cells without cellular clocks, their discs become damaged much faster than those of normal mice.

A study was conducted to look at the molecular and genetic activity within cells, to understand effect of a circadian rhythm to IVD, to identify a cellular clock in IVD, their change with age, temperature and chemical mediators. For identification of response to temperature, the cells were made luminescent to track the activity within them, in line with daily rhythms. Checking response against chemical mediators performed by using Interleukin B and Tumor Necrosing Factor. In a separate experiment live mice were bred without 24 hour clocks in their IVD cells and were monitored for disc degeneration, compared to normal mice of the same age. The work showed both mice and human cells had their own internal 24 hour clocks, demonstrated by the regular emission of pulses of

light. The cells became desynchronised when subjected to temperature changes at different times, suggesting that body temperature may be what “sets” the cells’ clocks. Cells from older mice had a weaker 24-hour pattern than those from younger mice, reflecting the way that body clocks are known to weaken with age. The cells’ body clocks were disrupted by interleukin B, suggesting that long-term inflammation could also cause body clock problems. The discs of engineered mice without body clocks in these cells degenerated much faster than those of normal mice. Images of the discs after 12 months showed they were much thinner, had bony growths into the cartilage and signs of fibrosis in the tissue around the edges.

CONCLUSION

Intervertebral discs are one among the key component in human body that are least taken care with the evolving life style. It leads to tremendous damage resulting in spectrum of intervertebral disc pathologies. The condition has become a quite common phenomenon in the world population causing a reduced quality of life. In contemporary knowledge intervertebral disc is a mixture of proteoglycans including its branches of glycosaminoglycan and collagen fibres with predominance in water. Overlooking the embryology from contemporary science and Ayurveda, intervertebral disc can be designated as *asthi*. But, considering its location and mechanics, intervertebral disc act as joints that aid in mobility and act as shock absorber. The component predominant in intervertebral disc is *shleshakakapha* with predominance of *apmahabhoota*. With the physiological increase in age or due to ageing process there will replacement of proteoglycans by collagen or *shoshana* of *kaphadosha* takes place due to *rookshaguna* of *vataadosha*. The impact of *vata-*

dosha due to ageing and other traumatic events also cause an impact on the mobile segments itself or to adjacent structures supporting them structurally and functionally. This leads to spectrum of intervertebral disc pathology depending upon the duration and intensity of aetiology associated with other morbid factors for the manifestation of varied set of signs and symptoms. The recent advancement aid in for a better discerning of IVD pathology by revalidating the impact of erratic lifestyle causing disruption to circadian rhythms, effect of ageing and impact of inflammation on health of intervertebral disc.

REFERENCES

1. A. J. Cole, S. A. Herring; The low back pain handbook – A guide for the practicing clinician, 2nd Edition; Hanley and Belfus Medical publishers, Ch. 3; Low back pain . Definition. P.27.
2. World Health Organization, Priority diseases and reasons for inclusion, Ch 6.24, Low back pain, Background.
3. P. K. Levangie, C. C. Norkin; Joint structure and function: A comprehensive analysis, 4th Edition; Jaypee Brothers Medical Publishers, Ch. 2; Anatomy. P.97
4. P. K. Levangie, C. C. Norkin; Joint structure and function: A comprehensive analysis, 4th Edition; Jaypee Brothers Medical Publishers, Ch. 2; Articulation and Kinematics. P.98
5. L. h. Bannister, M. M. Berry, P. Collins, M. Dyson, J. E. Dussek, M. W. J. Ferguson; Gray's anatomy, 38th Edition; Churchill livingstone (Harcourt Publishers); Ch. 6. Axial skeleton, Intervertebral discs. P. 515.
6. L. h. Bannister, M. M. Berry, P. Collins, M. Dyson, J. E. Dussek, M. W. J. Ferguson; Gray's anatomy, 38th Edition; Churchill livingstone (Harcourt Publishers); Ch. 6. Axial skeleton, Annulus fibrosus. P.513.
7. P. K. Levangie, C. C. Norkin; Joint structure and function: A comprehensive analysis, 4th Edition; Jaypee Brothers Medical Publishers, Ch. 4; Vertebral endplate. P. 157 – 158.
8. P. K. Levangie, C. C. Norkin; Joint structure and function: A comprehensive analysis, 4th Edition; Jaypee Brothers Medical Publishers, Ch. 4; Vertebral nutrition. P.160
9. Agnivesha, CharakaSamhita with Ayurveda Dipika Commentary, Ed. Acharya Y.T, ChaukhambhaSurbhara-tiPrakashan, Varanasi, 2009 (Reprint), Chikitsa Sthana.15/30
10. L. h. Bannister, M. M. Berry, P. Collins, M. Dyson, J. E. Dussek, M. W. J. Ferguson; Gray's anatomy, 38th Edition; Churchill livingstone (Harcourt Publishers); Ch. 6. Axial skeleton, Vertebral column. P.511.
11. Vagbhata, AshtangaHrudaya with SarvangaSundara Commentary, Ed. Acharya H.P, ChaukhambhaOrientalia, Varanasi, 2005 (Reprint), Sutra Sthana.11/4. P.507.
12. Vagbhata, AshtangaHrudaya with SarvangaSundara Commentary, Ed. Acharya H.P, ChaukhambhaOrientalia, Varanasi, 2005 (Reprint), Sutra Sthana.1/8. P.6.
13. Vagbhata, AshtangaHrudaya with SarvangaSundara Commentary, Ed. Acharya H.P, ChaukhambhaOrientalia, Varanasi, 2005 (Reprint), Sutra Sthana.11/26. P.509.
14. Vagbhata, AshtangaHrudaya with SarvangaSundara Commentary, Ed. Acharya H.P, ChaukhambhaOrientalia, Varanasi, 2005 (Reprint), Sutra Sthana.1/11. P.9.
15. P. K. Levangie, C. C. Norkin; Joint structure and function: A comprehensive analysis, 4th Edition; Jaypee Brothers Medical Publishers, Ch. 4; Vertebral nutrition. P.160

- sive analysis, 4th Edition; Jaypee Brothers Medical Publishers, Ch. 4; Age related changes. P.187.
16. Ibid
17. Ibid
18. Madhava. Ch 1. In: Y. Upadhyaya (eds.)MadhavaNidana. 1st ed. Varanasi: Chaukhambha Sanskrit Sansthan; 1998. P.5.
19. Vagbhata, AshtangaHrudaya with SarvangaSundara Commentary, Ed. Acharya H.P, ChaukhambhaOrientalia, Varanasi, 2005 (Reprint), Sutra Sthana.13. P.211.
20. Radiology Assistant, *Robin Smithuis*, Radiology department of the Rijnland Hospital, Leiderdorp, the Netherlands. Cited 28th July, 2016. Available on <http://www.radiologyassistant.nl/en/p423d18702d2bd/spine-disc-nomenclature.html>.

CORRESPONDING AUTHOR

Dr. R. Nithin Krishnan

Post Graduate Scholar

Department of Roganidana,

ShriDharmasthalaManjunatheshwara

College of Ayurveda,

Hassan, Karnataka, India

Email: nithinkris1989@gmail.com

Source of Support: Nil

Conflict of Interest: None Declared