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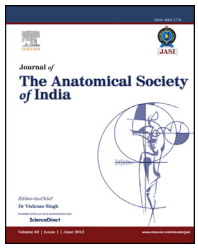

Review Article

Lumbago and associated morbid anatomy of lumbar spinal canal and facet joints☆

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ABSTRACT

Lumbago is the pain of variable duration in the lumbar region of dorsal spine. It is a leading cause of physical morbidity and disability. In recent times it has become a major medical concern across the globe specially in developed and industrialized countries. The ailment is of multifactorial origin but the degenerative changes are on the pinnacle. Out of the three major degenerative changes, viz: disc degeneration, spinal stenosis and facet joint arthrosis, the later two have been reviewed and analyzed in detail as a part of Ph.D thesis work of the second author under supervision of the first author. On analysis, it is found that: a. With the advent of recent imaging techniques there has been a major paradigm shift in the diagnosis and treatment of lumbago. b. Spinal stenosis with facet joint arthrosis is always associated with lumbago whereas spinal stenosis alone may or may not be. c. The kinesiology of the spine plays an important role in the degenerative process of the spine.

1. Introduction

The term Lumbago (L, lumb = loin) in early days was used to describe lower back pain (LBP). This term in real life is applied to the pain of variable duration on the back of body in the lumbar region. The low back pain in recent times has become so common that it has been an important health problem all over the world especially in industrialized nations.[1,2] It disables individuals ability to not only carry out their day-to-day activities but also affects their performance at the work-place. The work performance disability resulting from back pain is more common than any other disability in adults aged less than 45 years and second only to arthritis in people of 45–65 years age groups. It affects about 40% of people and occurs in almost similar proportions in different populations. Approximately 9–12% of people have LBP at any given point of time, and nearly one quarter (23.2%) of them report having it at some point over any one-month period.[3,4]

Low back pain occurs as a result of multiple etiologies. It has a variable magnitude for different ethnic and age groups.[5] The consequences of back pain are felt not only by the patient but also by family members, work-place managers and society as a whole.

Aging of the lumbar spine is an evolutionary and dynamic process that leads to degenerative changes, not only in the intervertebral disc but also in the bony canal and facet joints.

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The aim of the present article is to review in detail factors responsible for lumbar spinal stenosis (LSS) and facet joint arthrosis (FJA) in patients suffering from lumbago. The article deals with the work of earlier researchers using various imaging modalities to diagnose LSS and FJA. This kind of knowledge may be useful not only to physicians and surgeons but also to occupational and physiotherapy personnels in defining primary and secondary prevention plans/strategies. This will create an awareness in society of causes and risk factors associated with lumbago which may be of help in taking preventive measures.

2. History of lumbago

The history of low back pain is so old that it has even been described in the oldest Papyrus of surgical treatise- the Edwin Smith Papyrus (1500 BCE). Hippocrates (c. 460 BCE–c. 370 BCE) was the first to use a term for sciatic pain and low back pain; Galen was the first person to describe the concept of LBP in some better detail. Through out the end of first millennium physicians did not attempt any specific treatment and simply recommended wait and watch policy for results to happen. Through the Medieval period, folk medicine practitioners provided non-specific treatments for back pain based on the belief that it was caused by spirits.[5]

By the beginning of 20th century the factors such as inflammation and compression of nerves were considered to be the leading cause for low back pain. At the same time, an American neurosurgeon Harvey Williams Cushing initiated the mode of surgical treatments for low back pain.[6] In the 1920s and 1930s, new theories of the cause arose, with physicians proposing a combination of neurological and psychological disorders such as nerve weakness (neurasthenia) and female hysteria. Muscular rheumatism (now called fibromyalgia) was also cited with increasing frequency.[6,7]

From then onwards there has been no stopping and researchers have been able to define number of modifiable and non-modifiable causative factors for LBP. This has lead, along with advancements in science and technology, to specific treatment modalities as per an individual's age, gender and work status. The surgical interventions have become the mainstay for treatment of degenerative disease of the spine, this may include spinal manipulation, spinal implants, ozone therapy, prolotherapy etc.[6,7] For a better success in these operative/surgical procedures a thorough understanding of anatomy and kinematics of lumbar spine is essential.

3. Anatomy and pathophysiology of lumbar spine

The human lumbar spine transmits the weight of the body to the lower limbs through pelvic girdle. It consists of five vertebrae and intervening intervertebral discs. The lumbar vertebrae are heavily built and progressively increase in size from L1–L5. The intervertebral discs (IVDs) are fibrocartilaginous tissues which act as shock absorbers during transmission of forces and provide elasticity for the various vertebral column movements.

3.1. *Lumbar spinal canal*

It is bounded anteriorly by posterior margin of the vertebral body and IVD; and posteriorly by vertebral arches and ligamentum flavum. The shape of the lumbar canal varies from near oval to trifoliate form[8] (Fig. 1).

The trefoil configuration is commonly seen at L5 vertebral level, making the intervertebral foramen between L4 and L5 the smallest in diameter. Of the various anthropometric measures taken for lumbar canal the values of transverse diameters are considered critical for assessing the size of the canal.[9–11] According to a study by Rakhawy et al[12] on 100 lumbar vertebrae from 20 complete skeletons it was concluded that the shape of the lumbar canal was variable from LI to L5. As per this study there have been proposed six types of lumbar canal (Fig. 2); Pattern I (Fig. 2a) where the width of the canal increased from L1 to L2 then narrowed at L3 and rewidened gain from L3 to L5; Pattern II (Fig. 2b) where the width of the canal increased gradually from L1 to L5; Pattern III (Fig. 2c) where width of the canal remained constant from L1 to L2 then narrowed at L3 to widen at L4–L5; Pattern IV (Fig. 2d) where canal narrowed from LI to L2 then remained constant from L2-3 to widen further till L5; Pattern V (Fig. 2e) demonstrated that the canal width remained constant till L4 and finally widened at L5; Pattern VI (Fig. 2f) where canal narrowed consistently till L3 and widened then onwards till L5.

Similar studies by various other research workers have defined different dimensions at varying levels of lumbar vertebrae.[13–18] Thus assessment of the size of the vertebral canal is an important diagnostic procedure for low back pain of unknown etiology.

3.2. *Facet joints*

Other than the spinal canal size, lumbar facet joints play an important and critical role in maintaining stability of lumbar spine. These are typical diarthrodial joints which may be C or J shaped having a joint space with a potential capacity of 1–2 ml .The presence of menisci have also been studied and are present to support the incongruence between the subjacent articular surfaces.[19] The facet joints thus play an important role in load transmission; they stabilize the motion segment in flexion and extension and also limit the axial rotation.[20] A study by Kirkaldy-Willis[21] emphasized the

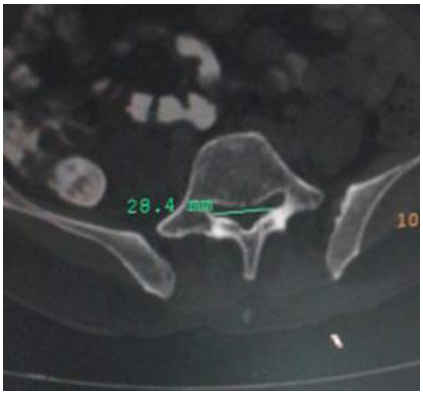


Fig. 1 – Trifoliate lumbar canal.

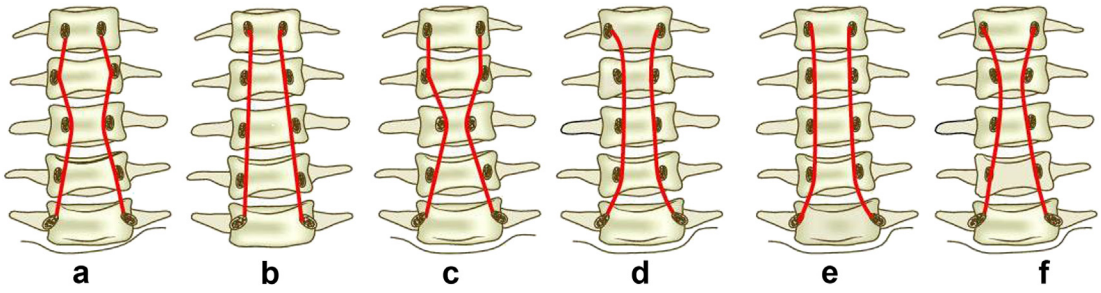


Fig. 2 – Patterns of lumbar canal.

interdependence of the disc and facet joints for normal spinal function and described how derangement or injury to either of these articulations, leads to abnormal forces and impairment of the other, the so called "tripod" effect. They further described the morphologic features of degeneration of facets and postulated how these might be associated with various clinical syndromes.

3.3. *Intervertebral discs*

Intervertebral discs lie between adjacent vertebrae in the spine. Each disc forms a fibrocartilaginous joint between the vertebrae to allow slight movement of the vertebrae, and also acts as a ligament to hold the vertebrae together. Discs consist of an outer fibrous ring, the annulus fibrosus, which surrounds an inner gel-like material in the center, the nucleus pulposus. The annulus fibrosus consists of several layers of fibrocartilage. The strong annular fibers enclose the nucleus pulposus and distribute pressure evenly across the disc. The nucleus pulposus contains loose fibers suspended in a mucoprotein gel. The intervertebral discs and spinal ligaments connects the adjacent vertebrae and provide support for the transfer and constraints of loads applied to the spinal column.[22]

The nucleus of the disc acts as a shock absorber, absorbing the impact of the body's activities and keeping the two vertebrae separated. Nearly 20–40% of LBP is discogenic in etiology.[23] Before 40 years of age approximately 25% of people show evidence of disc degeneration but beyond age of 40 years, more than 60% of people show evidence of disc degeneration at one or more vertebral levels.[24] Lumbar disc disorders most commonly include degenerative disc disease and disc displacement. The most common areas of disc herniation are between L4 and L5 and between L5 and S1. Since the spinal cord ends at level of L1/L1–L2 vertebrae, only herniations of L1 to L2 discs can cause spinal cord compression or myelopathy. Disc disorders with myelopathy occurring between lumbar vertebrae L3 to L5 are rare. Herniations of L2 to L3 through L5-S1 can cause radiculopathy (compression of one spinal nerve root) or cauda-equina syndrome (compression of leash of spinal nerve roots L2-Cx1).[25]

4. Etiology of LBP

The causative factors for LBA are multifactorial which may arise in the spine per se or may be extrinsic in origin. Of the various factors included are: 1. injuries-musculoligamentous or bony, 2. degenerative changes of the vertebral column, intervertebral discs (IVD) or the facet joints, 3. disc prolapse-herniation of nucleus pulposus of the IVD with nerve root compression, 4. spinal stenosis – central or lateral, 5. structural anomalies of spine-scoliosis, spondylolisthesis, 6. metabolic diseases – Paget's disease, metastatic cancer and last but not the least 7. diseases of pelvic visceras.

Inspite of number of causes defined for LBP still around 85% of symptomatic individuals cannot be ascribed a definite etiology for the LBP[26,27] because of weak correlation between the symptoms, pathological changes and imaging results. More so evidences of anatomic herniated discs have been found in 20–30% of individuals with no evidence of LBP.[25,28]

The degenerative changes of spine are one of the major causes of low backache and are believed to be associated with segmental instability of the spine.[29,30] The three major components involved in degeneration include: IVD (causing degenerative disc disorders), facet joints (causing facet joint arthrosis) and lumbar canal (leading to lumbar spinal stenosis). These changes may/may not be associated with degeneration of adjacent soft tissues.

In 1982, Kirkaldy-Willis and Farfan[21] proposed three clinical and biomechanical stages of spinal degeneration: dysfunction, instability, and de-stabilization. In their widely-quoted work, White and Panjabi[31] defined spinal stability as the ability of the spine under physiological loads to limit patterns of displacement so as not to damage or irritate the spinal cord and nerve roots and, in addition, to prevent incapacitating deformity or pain due to structural changes. Conversely, instability refers to excessive displacement of the spine that would result in neurological deficit, deformity, or pain. Panjabi was the first to suggest that instability of the spine likely results from any dysfunction of either spinal structures or trunk muscles or from reduced neural control over the latter, and, is an important aspect of LBP. Instability of the spine could lead to excessive tissue strain and consequent pain.

5. Kinesiology

The types of movements occurring at the lumbar spine include: rotational, translational and coupling movements. Rotational movements are movements of the vertebra around an axis. All rotations produce a change in the orientation of the facet articular surface.

Translational movements are gliding movement of the vertebra and there is no change in the orientation of the facet

joints. Coupling movements are grouped movements that occur simultaneously viz; flexion, extension, translation, axial rotation, and lateral bending are physiologically coupled. In the lumbar spine, movements of flexion and extension increase in range from the top to the bottom with exception of the lumbosacral joint (L5-S1).[32]

The effect of line of gravity on the kinesiology and transmission of body weight has been discussed extensively by Pal et al[33] in their work on change in direction of articular surfaces of facet joints. In the lumbar region the line of gravity passes posterior to the vertebral bodies hence there is tendency for greater weight transmission on the facet joints. The orientation of lumbar facet joints changes from coronal plane at birth to sagittal plane with advancing age. This process of sagittalization begins at 6th postnatal month and is completed by 18 months of age. These sagittally oriented lumbar facet joints hence can bear the maximum impact of torsional stress. To maintain the line of gravity and to provide the stability, a three-column concept of the spine, as proposed by Denis,[34] is considered. As per this concept the spine is composed of 3 columns: Anterior, Middle and Posterior (Fig. 3).

The anterior column is composed of: Anterior longitudinal ligament (ALL), Anterior half of vertebral body and intervertebral disc (AAF).

The middle column is composed of: Posterior longitudinal ligament (PLL), Posterior half of vertebral body and intervertebral disc (PAF).

The posterior column is composed of: Transverse process, Spinous process, Pedicle, Lamina, Facet joints (FC), Interspinous ligament (ISL), Supraspinous ligament (SSL) and ligamentum flavum. The interaction of the anterior and posterior lumbar spinal columns is critical for normal physiologic function, load transmission, and kinematics. There can be tensile, compressive, shear and torsional loads applied on the spine.

Further for the spinal stability to be maintained during kinematic phases, two more important factors have been defined: first, the Neutral Zone and second, the Instantaneous Axis of sagittal Rotation. The neutral zone (NZ) is the range over which a spinal motion segment (SMS) moves with minimal resistance. The NZ offers a more direct measure of spinal instability and more recently techniques have been developed to estimate this parameter in vivo.[35,36] The Instantaneous Axis of sagittal Rotation (IAR) is located in posterior third of disc and by maintaining the correct location of the axis of rotation, the facet joints would slide efficiently across each other and loads would be distributed appropriately at the level replaced and at the adjacent levels.

The intervertebral discs and facet joints are the major units that work together to maintain the spinal kinematics. The Facet joints contribute 30% axial rotation control and annulus fibrosus of the disc contribute about 50% torsional stability. Abnormal biomechanics can be classified as (a) hypomobile or hypermobile movements between vertebrae or (b) instability of vertebral column. Muscle weakness, ligament injury, broken bones or damage to the intervertebral disc can all lead to abnormal biomechanics, and in the development of low back pain.

6. Lumbar spinal stenosis and facet joint arthrosis per se

As discussed above, the common degenerative conditions arising in the lumbar region are; the lumbar spinal stenosis, the lumbar facet arthrosis and degenerative disc disorders (DDD). The first two clinical entities are reviewed in detail in the present article.

6.1. *Lumbar spinal stenosis*

The spinal stenosis is defined as the narrowing of the bony spinal canal. According to the North American Spinal Society (NASS), around 20% of the adult population suffers from this pathology (5% central stenosis and 15% lateral stenosis). In elderly patients of over 60 years age, it is well tolerated and remains asymptomatic, being diagnosed only radiologically. On the other hand, 98% patients under 60 years are usually symptomatic.[37,38]

The spinal stenosis occurs by a combination of both bone and soft tissues disorders, which cause mechanical compression of spinal nerve roots. The two main types of stenosis: developmental and degenerative, can be easily differentiated with the help of recent advances in radiological and imaging techniques, the CT scan and MRI. In developmental type of stenosis, there is typical narrowing in several

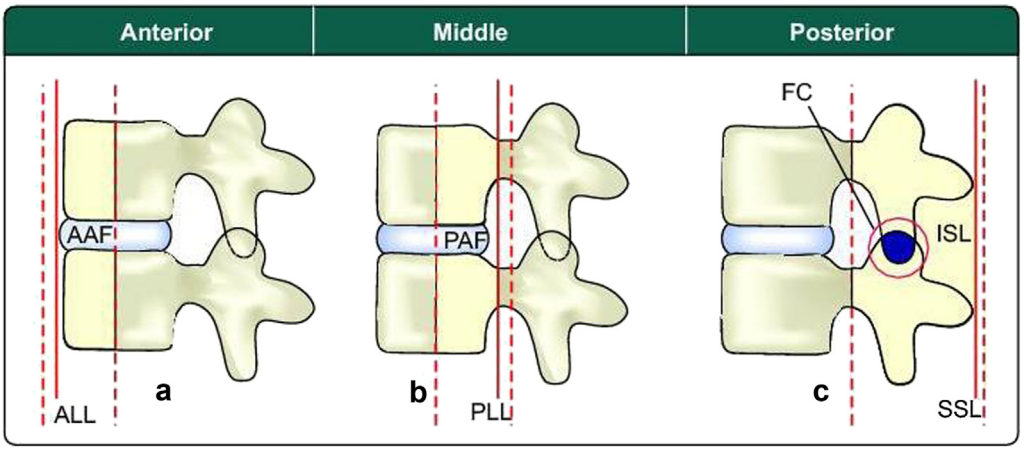


Fig. 3 – Three-column concept of spine.

or all spinal segments. On the other hand, the degenerative stenosis is typically segmental in nature and characteristically occurs at the level of disc spaces and articular processes.[38]

The first medical report on Spinal Stenosis appeared in the 1800s. In 1803, Portal of France had postulated that back and leg pain could be caused impingement on the nerves by the bone. In 1893, Lane carried a decompressive laminectomy to relieve a women of low back pain caused by lumbar spinal stenosis. In 1911, Bailey and Casamajor suggested a new hypothesis that spinal nerve symptoms were caused by facet joint exostosis, which in turn is responsible for spinal stenosis. To relieve the patient of existing symptoms he also proposed laminectomy as a surgical procedure.[39,40]

After this initial recognition of symptoms a gradual understanding of anatomy, pathology, biomechanics, causative and associated risk factors were evolved over the next 150 years. This revealed the pathoanatomic changes occurring in the spinal canal and surrounding bone and soft tissues leading to spinal stenosis and facet joint arthrosis.

By the end of the 19th century a large number of causative factors were defined for spinal stenosis. But it was Arnoldi[41] in the year 1978 who first classified Lumbar spinal stenosis as per the causative factors into Congenital/Developmental and Acquired types. He suggested that congenital stenosis can be further divided into dwarfism, and idiopathic types while acquired may have number of causes including degenerative, post-traumatic, metabolic and iatrogenic types.

This classification of lumbar stenosis leads to defining and analyzing the morphometry of bony canal by measuring its transverse and anteroposterior diameter by various researchers.[13–18] The further studies of Verbiest[42] proved that in developmental stenosis the transverse (interpedicular) diameters are normal but anteroposterior diameters are reduced due to short pedicles or thickening of lamina and articular processes. Hence the transverse diameters of the lumbar vertebral canal are considered to be a reliable index for the assessment of the size of the canal in degenerative stenosis.[10–12]

Earlier studies performed by various researchers have reported variable values of ratio of interpedicular distance and vertebral body width for different race at different age groups of male and female sex. The value of these diameters helps in defining the prevalence and number of individuals at risk to develop stenosis.

Tables 1 and 2 depict mean interpedicular distance of lumbar spinal canal obtained from plain radiography in male and female populations of different regions of the world reported by earlier researchers.

These studies define that the interpedicular diameter shows regional and racial variations. Although the authors could not notice normal standard values for the Delhi-NCR region population.

Apart of this classification based on measurement of interpedicular distance, another type of classification has been studied with respect to the site of nerve root compression-the central stenosis and lateral stenosis.[43]

6.2. *Central stenosis*

It occurs at the level of IVD and occurs due to ligamentum flavum buckling or hypertrophy, disc protrusion, hypertrophy

Table 1 – Reported mean transverse diameter (in mm) in males of different populations of the world.

Authors/population studied	No. of cases	Vertebral level				
		L1	L2	L3	L4	L5
Hinck et al, 1966, White Americans[13]	59	25.9	26.5	26.8	27.6	30.7
Amonoo Kuofi et al, 1982 Nigerian[14]	150	22.6	22.7	24.5	26.0	28.7
Piera et al 1990, Spanish[15]	110	27.79	28.39	29.44	30.89	34.31
Amonoo Kuofi et al 1990, Saudis[16]	160	25.1	25.3	26.3	27.2	30.9
Chhabra 1991, North Indians[17]	124	26.0	27.7	29.7	32.5	37.4
Nirvan et al 2005, Gujaratis[18]	101	24.0	25.4	26.4	27.9	30.9

of zygapophyseal joints or degenerative spondylolisthesis. Both morphologic and immunohistochemical studies have suggested the hypertrophy of ligamentum flavum due to fibrotic and chondrometaplastic changes which occur due to aging.[44] Degenerative process causes proliferation of collage type II fibrocartilage of ligamentum flavum along with deposition of calcium crystals (Botwin). Another recent study by Park et al[45] have proposed the higher expression of TGF-β1 responsible for hypertrophy of ligamentum flavum. The normal thickness is 4 mm but it can hypertrophy to size 7–8 mm thickness. The midsagittal diameters of less than 10 mm are suggestive of absolute stenosis and diameters less than 13 mm are suggestive of relative stenosis.[42] Schonstorm et al measured the cross sectional area of the dural sac in stenotic individuals and found it to be 89.6 $mm^2 \pm 35.1$ mm^2 while in normal individuals it is 178 $mm^2 \pm 50$ mm^2. This causes compression of the dural canal hence producing symptoms of LBP and neurogenic claudication.[10]

6.3. *Lateral stenosis*

The lateral lumbar column includes the nerve root canal (lateral recess) and the intervertebral foramen (neural canal). It was an extensive work by Lee et al to divide the lateral lumbar canal into three segments: the entrance zone, the mid

Table 2 – Reported mean transverse diameter in females of different populations of the world.

Authors/population studied	No. of cases	Vertebral level				
		L1	L2	L3	L4	L5
Hinck et al, 1966, White Americans[13]	59	24.3	24.9	25.4	26.9	29.0
Amonoo Kuofi et al 1982, Nigerians[14]	140	21.3	22.5	23.7	25.4	28.4
Piera et al 1990, Spanish[15]	105	25.66	26.25	27.53	29.53	33.39
Amonoo Kuofi et al 1990, Saudis[16]	180	23.5	24.0	25.2	26.9	29.0
Chhabra 1991 North Indians[17]	91	24.1	25.7	27.3	30.1	34.4
Nirvan et al 2005, Gujaratis[18]	101	23.3	24.3	25.8	27.0	29.8

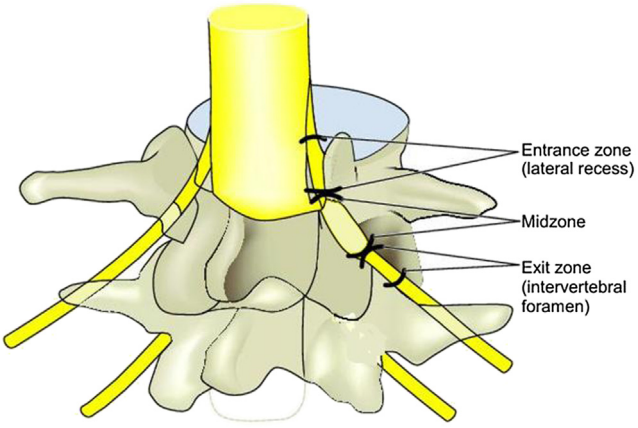
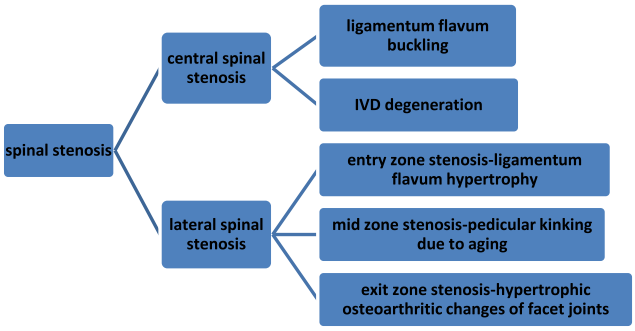


Fig. 4 – Three sites of Lateral Stenosis.

zone and the exit zone (Fig. 4) The entrance zone (lateral recess) is anatomically located medial to the pedicle and underneath the superior articular process of the facet joint.[46] The lateral recess is defined as the most common cause of "failed back surgery syndrome". The mid zone is located under the pars interarticularis and the pedicle. The exit zone is the intervertebral foramen. The algorithm below clearly defines the types of stenosis with their most common causative factors.



Diagnostic imaging techniques continues to play a pivotal role in the diagnosis, clinical and surgical management of lumbar spinal stenosis.

Though plain radiographs are inferior to myelography, CT scans and MRI for detecting spinal stenosis but since it is the technique easily available, hence is commonly used. The measurements for lumbar spinal stenosis are made by measuring the transverse (Fig. 5) and anteroposterior diameters of bony canal. The methods of measuring these diameters have been suggested by Amonoo Kuofi,[16] and Weber and deKlerk[47] in their earlier studies.

The "clothes pin" sign in the AP image and short pedicles in lateral views are the diagnostic signs for central stenosis. The "Kissing spine" occurs when the spinous processes are seen impinging on the lateral radiographs.

The CT scans alone or associated CT myelography can easily diagnose central or lateral stenosis. The accuracy of CT scans to diagnose spinal stenosis is defined as 100% by Heithoff in 1990.[48] The severity of facet joints and osteophytes presence can be visually appreciated. MRI is regarded an excellent tool to define the lateral stenosis and supplements the findings of CT scans. The surrounding soft tissue can be imaged to assess the effect on spinal diameters.

Thus, recognition and management of clinical problems inherent to lumbar spinal stenosis require understanding of diverse anatomical changes detected by imaging modalities (vide supra), and assessing the correlation with a wide spectrum of clinical manifestations.

6.4. *Lumbar facet arthrosis*

Lumbar facet arthrosis is defined as an inflammation and enlargement of the facet joints in the lumbar region. The etiology of facet joint arthrosis is degenerative, affecting all the tissues related to the specific joint; bone, cartilaginous and soft tissues, but primarily affecting the cartilage and subchondral bone causing osteoarthritis. Kettler et al[49] and Grogan et al[50] have even identified grading schemes to describe lumbar facet arthrosis, using both CT scan and MRI studies. The size of osteophytes at the facet joints have also been classified: Grade1 indicates no osteophytes; Grade2, a mild or possible osteophytes; Grade3, a moderate osteophytes; and Grade 4, a large osteophytes. Degeneration of the facet joint, in progression of osteoarthritis, leads to damage of cartilage, exposing the subchondral bone under it to also undergo such changes. This leads to limitation of mobility and pain as the bones rub over each other.

The first indication that the facet joint can be a source of back pain was described by Joel Goldthwait in 1911.[51] The use of the term Facet Syndrome was first coined by Ghormley in 1933.[52] It has been estimated that facet joint pathology is a contributory factor in 15%–52% of patients with chronic low back pain.[23,49,50] However, it has also been reported that the prevalence of isolated facet joint pain may be as low as 4%.[53]

The orientation of the lumbar facets is independent of gender and ethnic group but the presence of facet joint arthrosis in different age groups is not clear yet. In an effort to elucidate the extent and morphology of degenerative changes

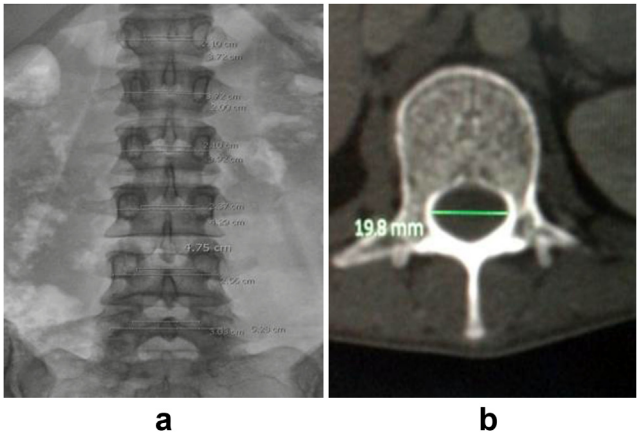


Fig. 5 – Methods of measuring transverse diameter of lumbar canal. a: on radiographs, b. on CT scans.

within the facet joint, Tischer et al carried out a detailed gross morphological study in an elderly population.[54] In his study on 32 cadaveric lumbar facet joints he analyzed various stages of cartilage degeneration. Eubanks et al examined the prevalence rate of facet arthrosis on 647 cadaveric lumbar spines.[55] The sample size studied included 57% cases between 20 and 29 years of age and 93% of the samples between 40 and 49 years of age with evidence of facet arthrosis. By the age of 60, the 100% of the cadavers had prominent facet arthrosis. The highest prevalence and the greatest severity of arthrosis were found at L4–L5 vertebral levels. All of these studies agreed on some common factors: (a) degeneration is seen even in younger age groups (b) lumbar facet degeneration increases with age, (c) the severity of degeneration gradually increases caudally.

These osteoarthritic changes affect the kinematics of the facet joints and subsequent are the cause for spinal instability and LBP.

Oblique views in radiography are needed to define degenerative features of facet joints. These include classic signs of arthritis of any small joint in the body, viz: narrowing of joint space, subchondral sclerosis and presence or formation of osteophytes. On CT images hypertrophy of facet joints, with decreased disc space is clearly evident. MRI is more accurate to define the soft tissue changes than the bony components of the joints. It is indicated in the lumbar spine where radicular pain is present. It is the investigation of choice where the secondary degenerative conditions manifest such as focal edema, venous stasis or nerve root edema or where pathological changes inside or outside the foramen are suspected.

Various modalities of Facet joint interventions have been used to manage the chronic back pain arising due to the arthrosis. Various evidences are present supporting the clinical effectiveness of these procedures (facet joint injections, medial branch blocks and facet joint neurotomy), and defining their potential complications.

For better prognosis of these interventional procedures a thorough morphometric knowledge of facet joints is essential. The studies related to facet joint arthrosis specially in Indian population are meager. Moreso, the studies done have dealt with mainly pathological changes in the cartilage of the joint but did not grade the facet joint arthrosis as per their symptomatology or in association with LSS. The significant data are not available to pronounce that there is correlation between the two degenerative conditions as regard to their concomitant presence, gender variations and vertebral levels of occurrence.

7. Conclusion

The two most common osteoarthrological causes of lumbago are: spinal stenosis and facet joint arthrosis. In both these causes the pain, called Lumbago, originates from the direct/indirect compression of spinal nerve roots which comes out from the spinal canal through the intervertebral foramen. The pain in spinal stenosis occurs only if dural sac ensheathing the nerve roots is compressed and that is the reason that in many of the cases inspite of stenosed canal the individuals remain asymptomatic. On the other hand, the pain in facet joint arthrosis always is due to direct compression of nerve root emerging underneath it. After thorough review of literature on Lumbago, we conclude that Spinal canal stenosis may or may not be always associated with Lumbago whereas Lumbar facet joint arthrosis is always associated with Lumbago. Hence it can be conceptualized that in symptomatic stenosed individuals it is necessary to look for arthritic changes in the facet joints.

Conflicts of interest

All authors have none to declare.

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