Chapter

1

Lumbar Canal Stenosis: Introduction and Historical Background

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INTRODUCTION

Today lumbar canal stenosis (LCS) is a well-recognized, very common and disabling entity. It was present in the past but its recognition came much later. In a study in India by Chahal et al[4] in 1978 it was found that majority of the adults in the 4th decade of life suffered from back pain due to spondylotic degenerative changes of the facet joints and thickening and incurling of ligamentum flavum.

It is a degenerative disorder generally regarded as part of the aging process. Progressive and disabling symptomatology leads to significant compromise of activities of daily living.[8] Even after surgery, it is unfortunate, that over a period of time the functional quality of life is affected because of progression of disease. It is surprising to know in recent articles[1,2,6,7,9,11–13,15,17,22,32] that neither the current surgical options nor decompression with fusion[10,28] are able to adequately address the full pathophysiology of lumbar canal stenosis.[24,25] Recently Spine Patient Outcome Research Trial (SPORT) has reported re-operation rates of 13% at 4 years follow up.[3]

In the past the diagnosis depended on indirect findings of bulbous spinous processes, diminished interlaminar space on plain X-rays and hour glass constriction on coronal view of iophenydol (myodil myelogram). Basically the pathology involves cauda equina compression and some investigators in the past tried to establish the severity by measuring the antero-posterior diameter of less than 15 mm to confirm the diagnosis as well as severity. It is common in lumbar canal stenosis to get complete obstruction of thecal sac in both AP and LAT views of a myelogram (myelographic block) (Fig. 1.1). In fact the block can be reversed by assuming flexion posture suggesting the clinical diagnosis of LCS. Advent of MRI has provided better understanding and more objective evaluation of the pathology.

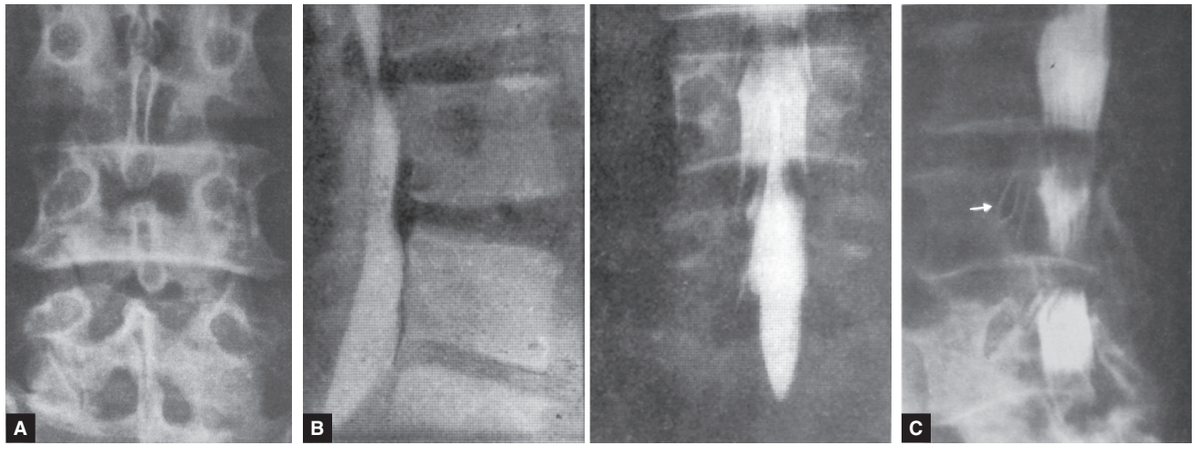

Fig. 1.1A to C: (A) Plain X-ray showing bulbous spinous processes, obliteration of interlaminar space and coronal orientation of facets in spinal stenosis; (B) Myelogram showing hour glass defect due to facet hypertrophy without any evidence of disc prolapse; (C) Myelogram showing multiple spinal stenosis

Chapter

2

Historical Landmarks in the Development of Degenerative Lumbar Canal Stenosis

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Lumbar canal stenosis was first observed in 1880 in Egyptian mummies.[6] The aging spine is but natural phenomenon as age advances. The degenerative process produces bending of the spine and kyphotic deformity resulting in aged people walking bending forwards.

Patient found relief on bending forwards (bony canal becomes long and the nerve). In 1893 English surgeon Lane did surgery in lumbar canal stenosis to decompress cauda equina and the patient obtained relief.[5]

In 1900 Sachs and Frenkel described a successfully operated patient and observed that before surgery.

The patient could walk with some comfort by bending forwards but after surgery he could walk comfortably in erect position. The canal is stretched along with nerve roots in flexion but in extension they are shorted and crowded superseded by in curling of ligamentum flavum and prolapsed disc.[8]

During this period the motion segment of spine was described by Junghans and he felt that any degeneration in one part will produce instability.[3]

The concept was followed further by R. Roy Camille when he divided the spine into three columns as follows.[7]

Anterior column: Anterior and posterior long. Ligaments, the vertebra and intervertebral disc.

Middle column: The two facet joints and the laminae.

Posterior column: Spinous processes and intra and supraspinous ligaments.

He too opined like Junghans that degeneration in one column will produce degenerative changes in all columns of the biomechanical construct.

A Danish radiologist CI Baastrup in 1933 described Baastrup disease. But he observed only the posterior column and went on to describe the grades in the development starting with breakdown in the interspinous ligament with abutment, enlargement, sclerosis and formation of bursa resulting in deformity which he called "Kissing Spine".

He described four types:

- Type I: End to end touching of spinous processes.
- Type II: Hypertrophy of spinous processes.
- Type III: Ball and socket formation.
- Type IV: Oblique lie.

He opined that loss of lordosis happened due to disc degeneration and hypertrophy in the spinous processes.[1]

The degenerated facet joints were further classified into four types by Grogan but Fujiwara graded them depending on formation of osteophytes.

The concept of biomechanical columns and degeneration was scientifically first described by Kirkaldy and Willis in 1978.[4]

He described three joint complex being intervertebral disc and the two facet joints, i.e. anterior and middle column. He ignored the posterior column.

In 2014, Bam Bang Darwono from Jakarta in Indonesia has proposed a new classification called Jakarta 2014 classification including all the three columns and has suggested appropriate treatment including stabilization depending on the grade and extent of degeneration.[2]

Conclusion

It is concluded that degeneration in any column has a tremendous bearance on other 2 columns and therefore the surgical strategy should be such that decompression and stabilization will address to keep all 3 columns healthy.

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Lumbar Canal Stenosis and Sex

Some researchers made an attempt to correlate sex to the incidence of lumbar canal stenosis but their analysis did not prove their assumption.[11,12] Among 11,283 cases studied, no statistically significant differences were found.[4]

Types of Lumbar Canal Stenosis

Putting together all types of stenosis (congenital or acquired), the prevalence of relative LCS was 23.6% and of absolute LCS 8.4%.

Splitting the group, the Framingham Study[9] found that congenital relative lumbar canal stenosis was 4.7% and absolute lumbar canal stenosis was 2.6% and acquired relative lumbar canal stenosis was 22.5% and absolute LCS was 7.3% in a population study aged 60–69 years.

Consistent with previous reports and consensus opinion, the results demonstrate that the prevalence of acquired LCS increases with age.

Lumbar Canal Stenosis and Different Levels

It occurs most frequently at L4–L5 level, followed by L5–S1 and L3–L4 levels. However, one study concluded that LCS is more prevalent at L5 than L4 and that individuals with AP canal diameters ≤12 mm, particularly at L5 have a statistically significant association between LCS and occurrence of LBP, odds ratio (OR = 0.3) (95% CI: 0.4–0.7).

In yet one more, the authors reported that the L5 root was involved in 91%, S1 in 63%, L1 to L4 in 28%, and S2 to S5 in 5%. Only 35% of their patients had single root involvement, the others having multiple roots involvement.[1]

Lumbar Canal Stenosis and Clinical Presentation

Lumbar canal stenosis can be asymptomatic or symptomatic.

Prevalence in asymptomatic individuals over 55 was distributed as follows: 21–30% for moderate radiographic stenosis and 6–7% for severe. Haig and colleagues[3] using a cut-point of 11.5 mm found 23% prevalence of LSS in 31 asymptomatic individuals. In an MRI study of 67 individuals who had never had LBP, sciatica, or neurogenic claudication. Boden and colleagues[15] found LCS in one percent of individuals younger than 60, and 21% in individuals over 60 years old. Wiesel and colleagues[5] reported that among 52 asymptomatic individuals over 40 years of age, 50% of CT scans were abnormal with the most common diagnosis being lumbar canal stenosis and facet degeneration.

The general incidence of symptomatic stenosis ranging from 1.7 to 8%.[1] The clinical hallmark finding of LCS is neurogenic intermittent claudication, presenting as intermittent pain or paresthesia in the legs brought on by walking and standing, and classically relieved with flexion. However, other symptoms are also frequent. It has been reported[1] that 95% of patients with LCS presented with back pain, 91% claudication, 71% leg pain, 33% weakness, and 12% voiding disturbances.

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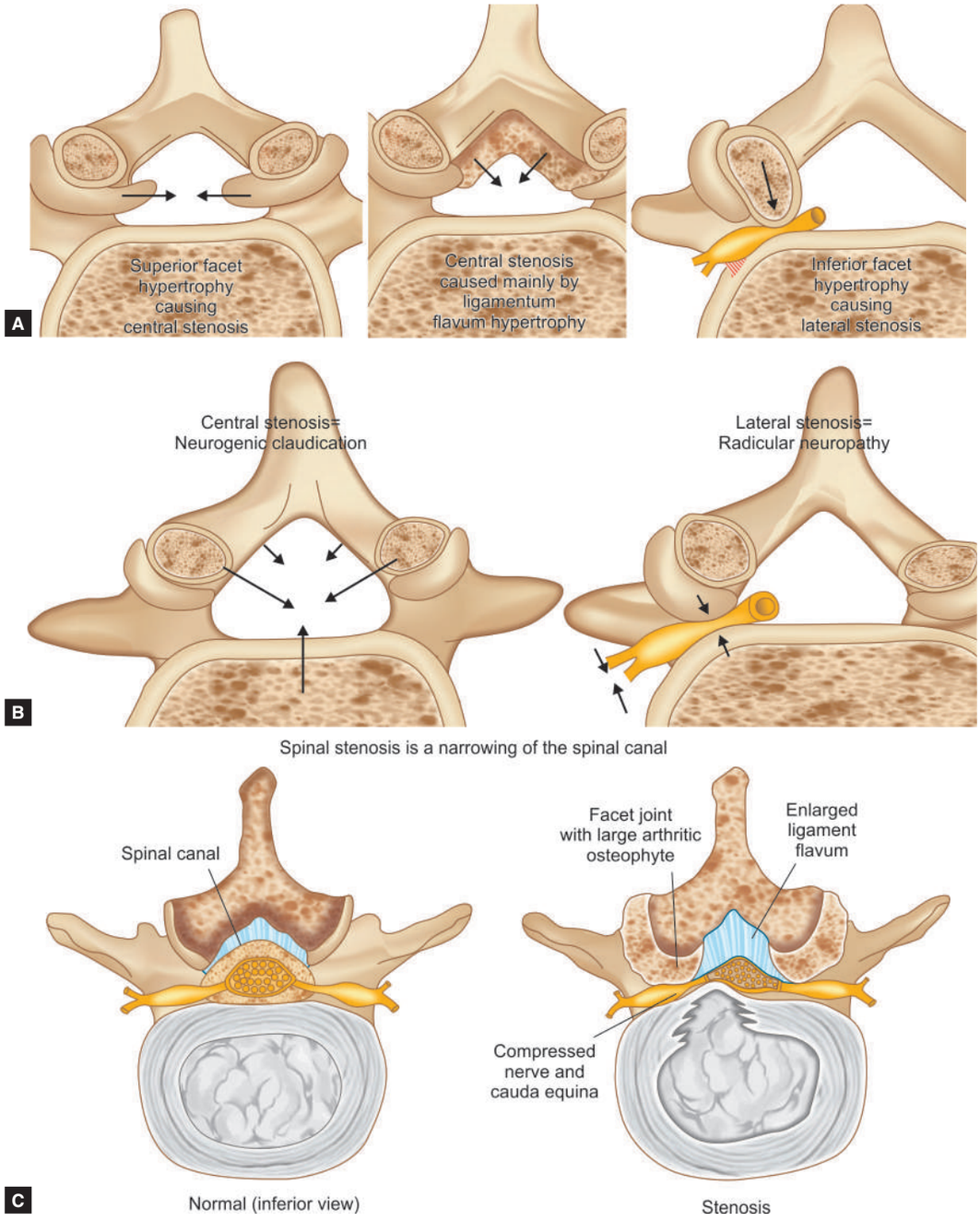

Fig. 4.6A to C: Stenosis in the central part of the canal as well as the lateral recesses

iliac joint to the acetabulum and also resists forward displacement of L5 on S1.

It is interesting to note that shortness of both pars interarticularis and pedicles at L5–S1 level reduces the moment arm (lever) of the forces applied to these supporting surfaces. However, the failure of pars interarticularis of L5 in spondylosis occurs because of its relative orientation being at right angles to the direction of forces applied against the inferior facet.

Average angles of facet joints relative to the spinous process as an aid to the insertion of transfacetal screws Table 4.7).

Table 4.7: Angle of facet joint

Level	L1–L2	L2–L3	L3–L4	L4–L5	L5–S1
Angle	25 (15–47)	28 (17–51)	3 (15–57)	48 (13–70)	53 (36–70)