

Prevalence of “congenital lumbar spinal stenosis” in patients with chronic low back pain in Mombasa

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Abstract

Background Context

Chronic low back pain is one of the commonest maladies of man. There are multiple causes of chronic low back pain that will include degenerative, inflammatory and mechanical causes.

Developmental lumbar spinal stenosis (DLSS) is known to cause symptoms of axial back pain with or without leg pain in the young adult. These symptoms become severer when patients with DLSS develop degenerative changes in the spine as the severity of theca sac and foramina compression increases. We hypothesized that developmental lumbar spinal stenosis is a major predisposing factor for chronic low back pain (CLBP) in adult population.

Purpose

The purpose of this study was to evaluate the prevalence of DLSS in a group of individuals suffering CLBP. We compared this prevalence with another group of asymptomatic individuals (without low back pain). Both computed tomography (CT) and magnetic resonance imaging (MRI) images were used for this analysis.

Study design/Setting

This is a prospective, case-control, radiographic study.

Sample

Radiological material from 118 individuals undergoing MRI scans for CLBP with or without lower limb pain were analyzed to obtain the AP diameters of the lumbar vertebral body and vertebral canals at L4 and L5 levels. 96 were enrolled in this study. Radiological materials (abdominal CT scans) of patients without back pain were obtained from hospital records for control analysis.

Methods

Using simple statistical methods we examined the association between DLSS and LBP.

Results

At LV5 level, 7% of the patients in the study group had severe D LSS compared to none in the control group; 59% had moderate stenosis (30% in control) while only 15% had a normal canal (33% in control group). The differences were found to be statistically significant (95% CI: 12.3–13.7 and $P=0.0071$). At L4 level, 2% of the patients in the study group had severe D LSS compared to 0% in the control group; 35% had moderate stenosis (13% in control) while only 31% had a normal canal (43% in control group). The differences were found to have a weak statistical significant (95% CI:

14.2-15.6 and $P = 0.4514$). It is apparent that in the study group 66% of the patients had a small canal (severe or moderate stenosis). It can therefore, be assumed the presence of congenital stenosis is prevalent in CLBP patients. There was weak correlation between size of the body and size of the canal (Pearson's $r = 0.4$).

Conclusions

The prevalence of congenital spinal stenosis in a cohort of patients with chronic back pain is compared to that of individuals from the same community without back pain. There is significant association between preexisting congenital stenosis and back pain in adults.

Introduction

There is a multitude of causes for low back pain (pain generators). In a study evaluating the pathophysiology of back pain presenting to a primary care physician, 4% of patients had a compression fracture, 3% had spondylolisthesis, 0.7% had a tumor, 0.3% had ankylosing spondylitis, and 0.01% had an infection. The overwhelming cause of back pain remained nonspecific [19, 20]. It is in this nonspecific group that we believe we find congenital lumbar spinal stenosis as a major participant.

Spinal stenosis whether congenital or acquired is defined as a narrowing of the spinal canal (vertebral canal) by either the bony cage or a combination of bone and soft tissues, which causes mechanical compression of the theca sac and or spinal nerve roots. However, congenital (or developmental) stenosis and the acquired (or degenerative) type are distinct from one another and although this distinctness is generally acknowledged [1], degenerative changes will make a hitherto quiescent congenital type symptomatic. The compression of the nerve roots may remain asymptomatic in childhood, but eventually become symptomatic in adulthood. These symptoms include: muscle weakness, reflex alterations, gait disturbances, bowel or bladder dysfunction, motor and sensory changes, radicular pain or atypical leg pain, and neurogenic claudication.

Very little is known about the epidemiology of congenital stenosis in the general population despite the fact that lumbar spinal stenosis is one of the most commonly diagnosed and treated conditions affecting the spine. The pathoetiology, predisposition and the clinical syndrome seen in adults with narrow spinal canals as opposed to those with acquired or degenerative spines is unknown. There is no universally accepted diagnostic criterion for spinal stenosis [2].

This is a major difficulty in performing an epidemiologic analysis. Computerized imaging (particularly MRI and CT scanning) are most frequently utilized modalities for diagnosis in clinical practice. Recognizing these limitations, we developed a criterion for classifying congenital stenosis

according to the canal diameters measured in scaled CT or MRI scans. Apart from direct measurement of canal diameter, these imaging modalities also reveal abnormal developmental changes in the vertebrae and of particular interest are spina bifidas and trefoilness. These three anomalies are clinically important when affected spines are compromised further by other pathologies. It is not surprising therefore, that symptomatic disc protrusion is more common in patients with trefoil shaped vertebral canals, where space is at a premium, than in the general population, and it is less common in patients with spina bifida occulta and with isthmic spondylolisthesis where the canal is more spacious. Acquired changes such as disc degeneration, hypertrophied ligamentum flavum, listhesis and spondylolysis, subarticular and or foramina narrowing in an already stenotic canal can only worsen the symptoms.

Methodology

Study design: A prospective, case-control, radiographic study.

Patient Sample: All study participants were voluntary patients of the author between July 2010 and July 2013. All underwent a thorough clinical assessment including filling in the validated and widely used modified Nordic Low Back Questionnaire [3]. The questionnaire defines significant LBP as “low back pain on most days of at least one month in the last 12 months”. Recorded neurological symptoms included saddle anaesthesia, bowel or bladder disturbance, pain in the buttocks or thighs or below the knee, numbness or tingling in the leg or foot, or weakness in the leg or foot.

Axial and sagittal magnetic resonance imaging (MRI) and lumbosacral plain radiographs were then done on all the patients with a clinical diagnosis of LBP. Out of 222 patients 96 cases were picked to match (for age, sex and ethnicity).

Soft copies of Abdominal CT scans done at various times since the installations of a Siemens sixteen-slice multi-detector CT scanner in 2012 were retrieved. One hundred eighteen (154) such copies were retrieved together with the hospital records. These patients or their relatives were contacted on phone to fill in the modified Nordic Low Back Questionnaire. Out of 154 records, we were able to get 96 participants who had no history of recurrent back pain. Their scans have been used to control the study.

Outcome Measures:

MRIs and CT scans were assessed by the author. MRI scans were scaled at source to allow direct manual measurement of the desired parameters. CT scans were evaluated in a blinded fashion with respect to clinical and demographic data. Bone windows were used for measurements both measurements. The antero-posterior diameter of the spinal canal was measured at the mid-vertebral body level. This level considered more precise than the mid-sagittal view due to avoidance of inaccurate measurements resulting from scoliosis or improper patient positioning [4]. Similarly, measurements from the axial MRIs scans were restricted to the midline anterior-posterior (AP) vertebral body diameter and midline AP canal diameter on L4 and L5 vertebral body only. On the CT scans of the control group similar measurements were done using appropriate computer software (Painter Image editor).

Analysis

The results of measurement on the images of the 192 individuals were tabulated in worksheets. The results were then grouped and graded utilizing a four-tier grading scales as shown in table1 and table 2 below:

SPINAL CANAL SIZE (mm)	DESCRIPTION
>15	Normal
13.1- 15	Mild Stenosis
10.1 - 13	Moderate Stenosis
<10	Severe Stenosis

Table 1: showing the four- tier grading for the spinal canal size.

VB SIZE (mm)	DESCRIPTION
<25	Underdeveloped
25 - 30	Small
31- 35	Average
>36	Normal

Table 2: showing the four- tier grading for the vertebral body size

Statistical analysis

All statistical analyses were performed using IBM SPSS Statistics 19 and other computer based programs.

RESULTS:

The study sample included 96 study participants, 37 (39%) males and 59 (61%) females. The mean age was 39.3 ± 8.2 (age range: 18–50). In the control group there were 41(43%) males and 55 (57%) females; the mean age in the group was 48.7 ± 17.2 . The comparison tests (F.TEST) showed no age difference between the study and the control groups ($p=2.7999$), or in prevalence of males. In our study sample all participants reported experiencing LBP on most days of at least one month in the last 12 months. Most patients presented with multiple symptoms that are characteristic of low back pain syndrome. The distribution of symptoms is as shown in table 3 below.

SYMPTOM	n	%
Back pain and Stiffness	73	76
Pain in lower leg (below knee)	56	58
Numbness in the leg or foot,	43	45
Pain in a buttocks or thighs	13	14
Bowel and bladder disturbance	10	10
Weakness in the leg or foot.	9	9
Claudication	7	7

Table 3: showing the frequency of symptoms

The AP diameter of the canal was significantly smaller in the low back pain patients at both lumbar levels measured than in the control group (L4: 14.9 mm (0.7) vs. 15.3 mm (0.7), $p = 0.4514$; and at L5: 13.0mm (0.7) vs. 14.7 mm (0.8), $p = 0.0071$)-Table 4.

CANAL SIZE LV4

CANAL SIZE (mm)	MEAN	CONFIDENCE I	T TEST
STUDY	14.9	0.7	

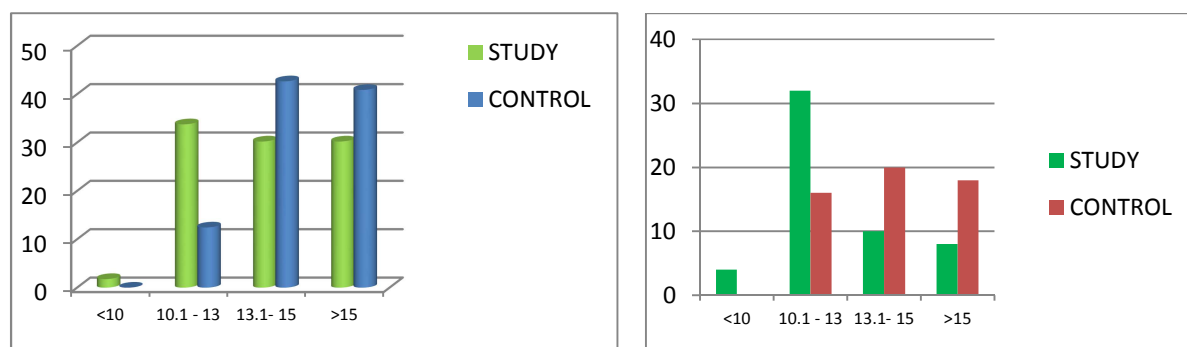
CONTROL	15.3	0.7	0.4514
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CANAL SIZE LV5

CANAL SIZE (mm)	MEAN	CONFIDENCE I	T TEST
STUDY	13.0	0.7	
CONTROL	14.7	0.8	0.0071

Table 4: Spinal canal diameter (a) comparing the means at LV4 (b) in LV5

The differences at L5 level were statically significant. The distribution of various grades of stenosis in the study and control samples was done separately for LV4 and LV5. The results are shown in charts 1(a) and 1(b) below.



Charts 1(a) grades of canal stenosis at LV4

(b) grades of canal stenosis at LV5

In the study group, absolute stenosis was observed in 2 (2%) at LV4 level and 7 (7%) at LV5 level; whereas there was no absolute stenosis in the control group. When 13mm AP diameter is used as a cut-off between normal and stenotic canal, then in the study group 60(62%) of the participants had a normal canal at LV4 compared to 32 (34%) at LV5. This is sharply contrasted by the control group where 88 (87%) of the canals at L4 and 68 (70%) at LV5 were normal respectively. Therefore, majority (66%) of the LBP had a canal of less than 13mm at LV5 in comparison to the control group where only 30% of the control group had a canal less than 13mm (Charts 2(a) and (b)).

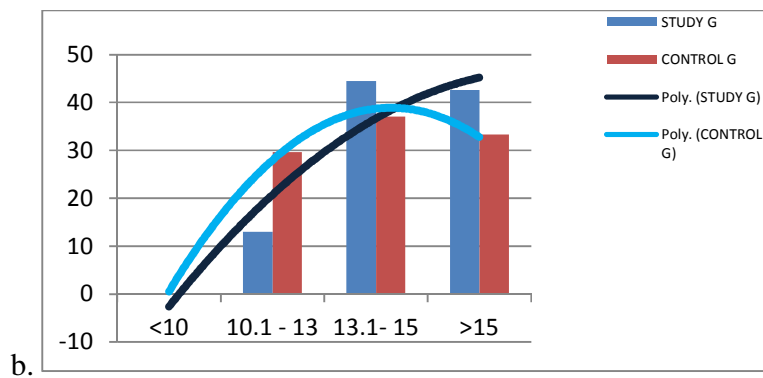
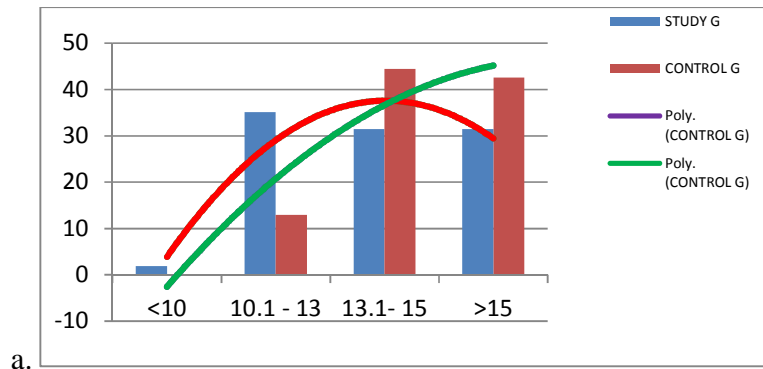


Chart 2 grades of canal stenosis (a) frequency of distribution LV4 which is almost binomial, while (b) distribution at LV5 shows a shift to the left.

Our study shows no difference in prevalence of radiographic CLSS between men and women (Male 13.8 mm (3.2), Female 14.1(2.2) P = 0.66369).

canal size	>13 mm	DLSS (<13 mm)	TOTAL
STUDY GROUP	60	36	96
CONTROL GROUP	84	12	96
TOTAL	144	48	192
CONFIDENCE INTERVAL	0.4	0.7	
P VALUE (at LV5)		0.0024033	
ODDS RATIO (OR)		3	

Table 5: The odds ratio for CLSS between the two groups

Similarly the vertebral body length was markedly shorter in the LBP group at both lumbar levels, although the differences were not statically significant (LV4: 30.3 mm (0.9) vs. 31.2 mm (1.3), p = 0.2713); and at LV5: 31.2 mm (1.0) vs. 32.1 mm (2.2), p = 0.2551).

VERTEBRAL BODY SIZE LV4

VB SIZE (mm)	MEAN	CONFIDENCE I	T TEST
STUDY	30.3	0.9	
CONTROL	31.2	1.3	0.2713

VERTEBRAL BODY SIZE LV5

VB SIZE (mm)	MEAN	CONFIDENCE I	T TEST
STUDY	31.2	1.0	
CONTROL	32.1	2.2	0.2551

Table 5: Vertebral body diameter (a) comparing the means at LV4 (b) in LV5

There was a weak correlation between AP diameter of the vertebral body and of the spinal canal (Pearson product moment correlation coefficient: $r = 0.4$).

Discussion

Low back pain is one of the most common disorders of mankind. Although back pain is ubiquitous, more than 70% of people in developed countries experience low back pain at some time in their lives [18]. Every year, one third to one half of adults suffers low back pain and 5% of people present to their medical provider with a new episode. Low back pain is most common in patients between the ages of 35 and 55 years [18]. Low back pain is also a major cause of pain and disability and a common indication for spine surgery. In the developing world, where spine surgery is not readily available, degenerative spine disorders are a major cause of deformity such as stooping, humping, general shortening and premature aging.

The contribution of congenital spinal stenosis to low back pain has not been quantified. The disease process usually begins with degeneration of the intervertebral disks and facet joints, resulting in narrowing of the spinal canal and neural foramina. Associated factors may include a developmentally narrow spinal canal and degenerative spinal instability. Hilibrand AS and N Rand [16] reported in a short-term follow-up data of surgically decompressed patients showed superiority of operative management over non-operative treatment, perhaps, suggesting that morbidity is more related to narrowness than inflammation or instability. They reported surgical success rates as high as 85%.

We conducted a study intended to relate the prevalence of congenital lumbar spine stenosis among low back pain patients. We used MRI scans to measure the AP diameter of the lumbar canal at LV4 and LV5 levels in patients with chronic low back pain. Bone windows were used for both measurements. The antero-posterior diameter (AP) of the spinal canal was measured at the mid-vertebral body level. This level is considered more precise than the mid-sagittal view due to avoidance of inaccurate measurements resulting from scoliosis or improper patient positioning [4]. Similarly, measurements of the AP vertebral body diameter from the axial MRIs scans were done on L4 and L5 vertebral body only. For the control group, similar measurements were done on CT scans using appropriate computer software (Painter Image editor). We developed a criteria where <10mm was considered severe stenosis, 10-13mm moderate stenosis, 13-15 mm mild stenosis and >15 mm was considered normal. Our cut-point for purposes of discussion was set at 13mm; so that any canal below 13mm was considered stenotic and any canal above 13mm was considered normal. Using this criterion we have shown that 66% of patients with LBP and 30% of asymptomatic individuals have stenotic canals at LV5 level, whereas, the prevalence was 37% (LBP) and 13% (asymptomatic) at LV4. In this study, we conclude that DLSS is more prevalent at LV5 than LV4 and that individuals with AP canal diameters ≤ 13 mm, particularly at LV5 have a statistically significant association between DLSS and occurrence of LBP (odds ratio (OR)=3.0 (95% CI: 0.4–0.7)). Kern Singh et al [17] used a cross-sectional area of the canal in 20 symptomatic congenitally stenotic individuals and age- and sex-matched with 20 asymptomatic, nonstenotic individuals. They showed that the cross-sectional area of the canal was significantly smaller in the congenitally stenotic patients at all lumbar levels measured (LV2-LV5). However, little is known regarding the epidemiology of congenital spinal stenosis in the general population. Verbiest [10] measured the mid-sagittal diameter of the lumbar canal at operation and proposed two major types of stenosis: *absolute stenosis*, with diameter 10 mm or less; and *relative stenosis* with diameters ranging from 10 to 12 mm. In a CT study, the same author suggested that mid-sagittal lumbar canal diameters less than 10 mm represent absolute stenosis and diameters less than 13 mm represent relative stenosis [5]. Ulrich and colleagues [4] suggested that the antero-posterior diameter of the spinal canal (measured on axial plain CT) of less than 11.5 mm is small. In another CT study, Lee and colleagues [6] reported that the sagittal diameter of the lumbar spinal canal is never smaller than 10 mm in a normal spine. Haig et al. [16] demonstrated that antero-posterior measurements of the spinal canal (using 11.95 mm as a threshold) can distinguish between patients with clinical spinal stenosis and asymptomatic individuals. The association between CLSS and LBP has been studied in the past but not fully established. De Villiers and Booyesen [8] in a report of 850 myelograms found a 6% prevalence of lumbar spinal stenosis but did not separate the congenital from the acquired forms of this condition. Fanuele et al.

[9] reported a prevalence of 13.1% among 17,744 patients. This was a large study utilizing a multicenter clinical database without providing criteria for diagnosis of lumbar canal stenosis. In a multicentre study confounders (such as genetic and environmental factors) are not eliminated. Our study selected patients from one community who were living under similar environmental circumstances. Using a cut-point of 13mm between narrow and stenotic canals, we found 30% of our control patients asymptomatic DLSS. We should have analysed this group further, particularly for age but the numbers were few. However, there have been multiple studies reporting the occurrence of congenital spinal stenosis in asymptomatic individuals. **Haig and colleagues [10]** using a cut-point of 11.5 mm found 23% prevalence of DLSS in 31 asymptomatic individuals. Other studies show different results, perhaps, due to different cut-points. Most do not differentiate DLSS with the degenerative type lumping them together as DLSS. For example, **Boden and colleagues [11]** found DLSS in 1% of individuals younger than 60, and 21% in individuals over 60 years old in an MRI study of 67 asymptomatic individuals. **Wiesel and colleagues [12]** reported 50% of CT scans were abnormal among 52 asymptomatic individuals over 40 years of age. Leonid Kalichman [13] found a prevalence of absolute DLSS (cut-point 10 mm) to be 6.0% of asymptomatic individuals and 18.9% in individuals with LBP. **Jarvik and colleagues [7]** also found that severe DLSS is less common in individuals without LBP and is likely to be diagnostically and clinically relevant.

Our study shows no difference in prevalence of radiographic DLSS between men and women. This is consistent with findings in other studies (13, 14). **Jansson and colleagues [14]** in a study among 11,283 cases also found no statistically significant differences between sexes. We can, therefore, conclude that there is no significant sex difference in the prevalence of DLSS.

In this study, 57% of the CLBP group had the vertebral body AP diameter smaller than 30mm at LV5 level compared to 48% of the control group. At LV4 44% had smaller bodies compared to 28% in the control group. We therefore, conclude that DLSS is caused by failure of development of distal lumbar vertebrae in general while there appear to be a lag in growth of LV5 with failure to catch-up.

One drawback of this study was that all the measurements were done by one person, the author.

There are several limitations of the present study. First, is the use of two different scanning modalities; CT images for the control group and MRI images for the study group; assumedly introducing observer error? However, both modalities are considered reasonable alternative methods of evaluation of lumbar stenosis. Secondly, is the use of the antero-posterior diameter of the spinal canal alone, which may lead to underestimation of the prevalence of spinal stenosis, for example, in

patients with trefoil shape of the spinal canal [15]. Use of both the AP and lateral (transpedicular) diameters has been shown to be more accurate (13).

Conclusion

This study shows that the majority of the patients with chronic back pain have a narrow spinal canal. The prevalence of DLSS in this study is 7% at a cut-point of 10 mm and 67% at a cut-point of 13 mm (0% and 30% in the control group respectively). There is a large schism in these findings with those of Leonid Kalichman [13] where they found prevalence rates of 4.71% and 2.62% for relative and absolute stenosis, respectively. The very high prevalence of DLSS in this population may explain the high prevalence of neurological symptoms associated with chronic low back pain. It also poses a possibility of an aetiopathological process in this population that result in small lumbar spinal canals. This calls for a study in possible causes of lumbar vertebra and lumbar spinal canals.

REFERENCES

1. Arnoldi CC, Brodsky AE, Cauchoix J, Crock HV, Dommissie GF, Edgar MA, et al. Lumbar spinal stenosis and nerve root entrapment syndromes. Definition and classification. *Clin Orthop Relat Res.* 1976;4–5. [[PubMed](#)]
2. Gunzburg R, Keller TS, Szpalski M, Vandeputte K, Spratt KF. A prospective study on CT scan outcomes after conservative decompression surgery for lumbar spinal stenosis. *J Spinal Disord Tech.* 2003; 16:261–267. [[PubMed](#)]
3. Kuorinka I, Jonsson B, Kilbom A, Vinterberg H, Biering-Sorensen F, Andersson G, et al. Standardised Nordic questionnaires for the analysis of musculoskeletal symptoms. *Appl Ergon.* 1987; 18:233–237.
4. Ullrich CG, Binet EF, Sanecki MG, Kieffer SA. Quantitative assessment of the lumbar spinal canal by computed tomography. *Radiology.* 1980; 134:137–143. [[PubMed](#)]
5. Verbiest H. The significance and principles of computerized axial tomography in idiopathic developmental stenosis of the bony lumbar vertebral canal. *Spine.* 1979;4:369–378. [[PubMed](#)]
6. Lee BC, Kazam E, Newman AD. Computed tomography of the spine and spinal cord. *Radiology.* 1978; 128:95–102. [[PubMed](#)]
7. Jarvik JJ, Hollingworth W, Heagerty P, Haynor DR, Deyo RA. The Longitudinal Assessment of Imaging and Disability of the Back (LAIDBack) Study: baseline data. *Spine.* 2001; 26:1158–1166. [[PubMed](#)]

8. De Villiers PD, Booyesen EL. Fibrous spinal stenosis. A report on 850 myelograms with a water-soluble contrast medium. *Clin Orthop Relat Res.* 1976:140–144. [[PubMed](#)]
9. Fanuele JC, Birkmeyer NJ, Abdu WA, Tosteson TD, Weinstein JN. The impact of spinal problems on the health status of patients: have we underestimated the effect? *Spine.* 2000; 25:1509–1514. [[PubMed](#)]
10. Haig AJ, Geisser ME, Tong HC, Yamakawa KS, Quint DJ, Hoff JT, et al. Electromyographic and magnetic resonance imaging to predict lumbar stenosis, low-back pain, and no back symptoms. *J Bone Joint Surg Am.* 2007; 89:358–366. [[PubMed](#)]
11. Boden SD, Davis DO, Dina TS, Patronas NJ, Wiesel SW. Abnormal magnetic-resonance scans of the lumbar spine in asymptomatic subjects. A prospective investigation. *J Bone Joint Surg Am.* 1990; 72:403–408. [[PubMed](#)]
12. Wiesel SW, Tsourmas N, Feffer HL, Citrin CM, Patronas N. A study of computer-assisted tomography. I. The incidence of positive CAT scans in an asymptomatic group of patients. *Spine.* 1984; 9:549–551. [[PubMed](#)]
13. Leonid Kalichman, Robert Cole, David H. Kim, Ling Li, Pradeep Suri, Ali Guermazi, and David J. Hunter. Spinal stenosis prevalence and association with symptoms: The Framingham Study. *Spine J.* Jul 2009; 9(7): 545–550.
14. Jansson KA, Blomqvist P, Granath F, Nemeth G. Spinal stenosis surgery in Sweden 1987–1999. *Eur Spine J.* 2003; 12:535–541. [[PMC free article](#)] [[PubMed](#)] Schönström Spine. 1985; 10:806. [[PubMed](#)]
15. Eisenstein S. Lumbar vertebral canal morphometry for computerised tomography in spinal stenosis. *Spine.* 1983; 8:187–191. [[PubMed](#)]
16. Hilibrand AS. and N.Rand. Degenerative lumbar stenosis: diagnosis and management. *Am Acad Orthop. Surg* July 1999 vol. 7 no. 4 239-249
17. Kern Singh, Dino Samartzis, Alexander R. Vaccaro, Ahmad Nassr, Gunnar B. Andersson, S. Tim Yoon, Frank M. Phillips, Edward J. Goldberg, Congenital lumbar spinal stenosis: a prospective, control-matched, cohort radiographic analysis. *The Spine Journal* Volume 5, Issue 6, Pages 615–622, November–December, 2005.
18. William Lavelle, Allen Carl, Elizabeth Demers Lavelle. Invasive and Minimally Invasive Surgical Techniques for Back Pain Conditions. *Anesthesiology Clin.* 25 (2007) 899–911
19. Koes BW, van Tulder MW, Thomas S. Diagnosis and treatment of low back pain. *BMJ* 2006; 332 (7555):1430–4.
20. Deyo RA, Rainville J, Kent DL. What can the history and physical examination tell us about low back pain? *JAMA* 1992;268 (6):760–5.