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Pattern and Outcome of Surgery for Degenerative Disease of the Lumbar Spine using the Visual Analogue Score (VAS) and Oswestry Disability Index (ODI) at a Tertiary Hospital

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Abstract

Introduction: With the sustained evolution of complex spine surgery in our service, there is an increasing concern about safety, clinical outcome and overall patient confidence. There emerges a need to determine the outcome of surgery using standard clinical parameters (VAS and ODI).

Methodology: A retrospective review of cases of lumbar spine surgeries performed at the National Hospital Abuja from Jan 2015-Dec 2017 was conducted. Main study parameters were VAS and ODI before and one year after surgery. Data was assembled in Excel spreadsheet and analyzed with SPSS version 21. Results were presented in tables and charts.

Results: 41 cases met selection criteria. Mean age 50.56 ± 10.99 years. 22 (53.7%) females, 19 (46.35%) males. M:F= (1:1.15). 22 (53.65%) had chronic radicular low back pains. 12 (29.2%) had neurogenic claudication, while 7(17%) presented with paraparesis. Mean time to presentation was 15.33 ± 12.09 months. 25 (60%) presented within a year of onset of symptoms but only 4 (10%) presented after 2 years. Mean pre-op VAS was 8.07 ± 0.712. 32 (78%) recorded VAS >7. Mean pre-op ODI score was 81.95 ± 12.11. 28 (68.2%) recorded ODI of 81 to 100 range. Commonest pathology was lumbar canal stenosis. Commonest level involved was L4/5 in 21/40 (52.5%). The commonest operative intervention was Posterior Lumbar Interbody Fusion (PLIF) in 31/41(75.6%) followed by Primary Posterior Lumbar Decompression (PPLD) 10/41(24.3%). The most important post op complication was deep wound/implant infection in 1/41 (2.4%). Mean post op VAS was 1.76 ± 1.51. (P= <0.05). 34 (87.8%) recorded VAS, ≤ 2 but 5 (12.2%) had VAS ≥ 3. Mean post op ODI was 10.5 ± 14.24. 27 (65.8%) recorded significant improvements in the ODI but 1/41 (2.4%) maintained a similar ODI score post op.

Conclusion: There is a significant improvement in VAS and ODI following surgical intervention with minimal complication. Therefore, there should be no hesitation in recommending this treatment when the need arises.

Keywords: Degenerative Lumbar Spine Disease; Lumbar Decompression and Fusion; Visual Analogue Score (VAS); Oswestry Disability Index (ODI)

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Introduction

It is true that degenerative diseases of the spine may affect any of the component parts and manifest as osteophytes on the vertebral bodies, disc degeneration and herniation, hypertrophy of ligamentum flavum, and sometimes facet joint hypertrophy. But they often do not happen in isolation but in combination. They cause back pain from disc and facet joint related pathology, with or without radiculopathy due to entrapment/compromise of the natural foramina and canal where neural structures pass. Therefore, one common approach in surgical management is decompression of neural elements with or without instrumented fusion depending on the extent of the pathology. It is the patient's perception to pain and quality of live following surgical intervention that forms the major focus of this study. The expected objective of treatment is to improve quality of life by reducing pain. Surgical treatment is usually deployed only when conservative measures fail. Definition of a good surgical outcome depends on how success is assessed [1]. In the past, outcomes were assessed based on surgeons' perspective, as "excellent", "good", "moderate" and "bad". Despite technical successes and satisfactory post-op imaging, surgeons' perspectives do not always correlate with patient satisfaction [2]. In the recent past, more patients afflicted with degenerative lumbar spine

disease have undergone different complex lumbar spine operations with variable success rates. There is therefore an increasing concern about the safety, clinical outcome and overall patient confidence with these procedures. Therefore, there emerges a need to apply patient related outcome of these operations using standard clinical parameters such as VAS and ODI as a reliable tool to substantiate or otherwise refute the usefulness of these procedures. Visual Analogue Score (VAS) - is a standardized scale for assessing the pain intensity due to the subjectivity of the symptom [3]. Three varieties of VAS scales have been in clinical application. These includes:

(1) VAS relating patient pain perception to the level of liquid filling inside a glass cup [4].

(2) VAS relating patient pain perception to the facial expression [5].

(3) VAS relating patient pain perception to metric analogue scales.

ODI scale assesses pain related disability in lumbar spine pathologies [6]. It consists of 10 questions with six possible answers. Parameters include pain, personal care (dressing and bathing), lifting, walking, sitting, standing, sleeping, sexuality, socializing and the ability to travel. Values range from 0 to 5. Total score is calculated and presented on a scale from 0 to 100 as follows: 0 to 20 indicates minimal disability; 2,140 indicates moderate disability; 41 to 60 indicates severe disability; 61 to 80 indicates crippling back pain; and 81 to 100 indicates that the patient is either bed-bound or exaggerating their symptoms [7]. Clinically relevant minimum difference is 10 to 12.8 points in the total score [8]. There are other instruments that have been used to assess disability due to lower back disorders such as Roland-Morris Disability Questionnaire (RMDQ) [9] and the Quebec Back Pain Disability Scale (QBPDS) [10], but the ODI has remained more popular in clinical practice.

Surgical treatment for lumbar spinal stenosis is considered by many to be superior to non-surgical treatment [11], but the most recent Cochrane review of the efficacy of surgical treatment versus non-surgical treatment, in which only trials with neurogenic claudication as main inclusion criterion were included, did not support this conclusion [12].

Lumbar spine stenosis as a result of degenerative disease is a constellation of different pathologies including disc herniation, collapse of disc space, hypertrophy of ligamentum flavum, facet joint hypertrophy etc. All these contribute in various degrees to entrapment of the cauda equina as a whole with resultant classical neurogenic claudication or entrapment of the exiting nerve root at the intervertebral foramina with resultant radiculopathy. The resultant clinical effect is back and leg pains which impair the quality of lives of the afflicted, the degree of which can be assessed with VAS and ODI. The basic principle in surgical management is to provide decompression of the affected nerve roots and cauda equina, and to provide instrumented fusion when spine stability is threatened. It is hoped that the scientific application of these patient-related outcome assessment parameters will provide a reproducible insight into the relationship between surgical intervention and pain outcome.

Methodology

Ethical approval was sought for and obtained from the Institutional Review Board of the National Hospital Abuja in Dec 2014 to conduct a retrospective review of cases of Lumbar spine surgeries performed at the National Hospital Abuja from Jan 2015 to Dec 2017 for disc/degenerative disease (Abuja NHA/EC/051/2014). Permission was therefore granted to apply for and retrieve the relevant case notes from the medical records department of the hospital. Being a retrospective analysis, all relevant case notes were retrieved from NHA medical records (cases done from Jan 2015 to Dec 2017) and relevant research data quickly extracted there from within two weeks and the case notes returned to the Records to avoid any inconveniences to the patients during follow up visits. The sample size was therefore solely dependent on the volume of cases done within the stipulated time of this study. As a matter of unit policy, degenerative spine surgery protocol stipulates that for each of the patients, the back and leg pains perception as well as the associated disability and limitations of activities of daily living were assessed during the pre-operative work up using a questionnaire for VAS and ODI. For purposes of this study metric analogue scale was preferred option for VAS assessment due to its simplicity and wide application in spine surgery [13]. ODI was also the preferred choice in this study. Results and figures obtained were documented in the case notes. Also documented were other neurological findings including bowel/bladder dysfunction, limb weakness and sensory loss, duration before presentation, radiological findings and exact diagnosis for surgery, operation method, peri-op and post-op complications. The nature of the surgery is either a stand-alone decompressive surgery or with additional instrumented fusion when spine stability is threatened. Following discharge, they were followed up periodically in the clinics where VAS and ODI were assessed and documented up to one-year post surgery. It was considered more appropriate to apply both assessment tools because ODI is required to evaluate the effect of severity of back and leg pains as assessed by VAS on mobility and overall quality of live. Only patients who passed through this clinical process were selected for this study. The relevant data were extracted from the case notes retrospectively, entered on excel spread sheet and subsequently analyzed statistically using SPSS version 21. Results obtained there from were presented in tables and figures and discussed.

Results

41 cases met selection criteria. Mean age 50.56 ± 10.99 years (Table 1). 22 (53.7%) females, 19 (46.35%) males. M:F= (1:1.15). 22 (53.65%) had chronic radicular low back pains. 12 (29.2%) had neurogenic claudication, while 7 (17%) presented with paraparesis (Figure 1). Mean time to presentation was 15.33 ± 12.09 months. 25 (60%) presented within a year of onset of symptoms but only 4 (10%) presented after 2 years. The mean pre-op pain score as assessed by the VAS was 8.07 ± 0.712 (Table 2). A greater majority of patients, 32 (78%) recorded pain score as assessed by VAS, >7. On the other hand, only about a fifth of respondents, 9 (21%) had pain scores ≤ 7 as assessed by VAS (p= <0. 05). The mean pre- op ODI score was 81.95 ± 12.11 (Table 2). Greater than a third of patients, 28 (68.2%) recorded values within the 81 to 100 range. Commonest pathology

Table 1: Age distributions.

Age (years)	Frequency (n=41)	Percentage (%)
31-40	11	26.8
41-50	7	17
51-60	15	36.5
61-70	8	19.5
Mean age ± SD		**50.56 ± 10.99**

Table 2: Pre-op VAS and ODI.

VAS Score	Frequency (n=41)	Percentage (%)
6	2	4.8
7	3	7.1
8	27	64.3
9	10	23.8
Mean Score ± SD		**8.07 ± 0.712**

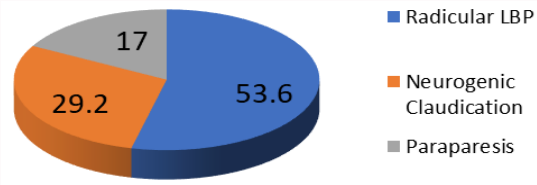

Figure 1: Major symptoms.

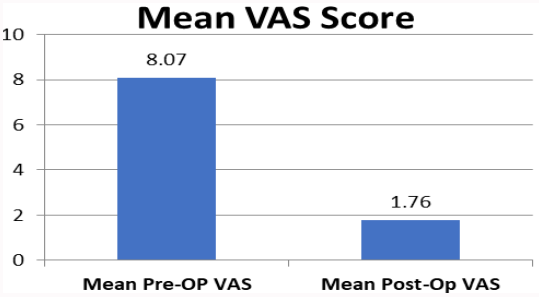

Figure 2: Pre and Post Op Visual Analogue Score (VAS).

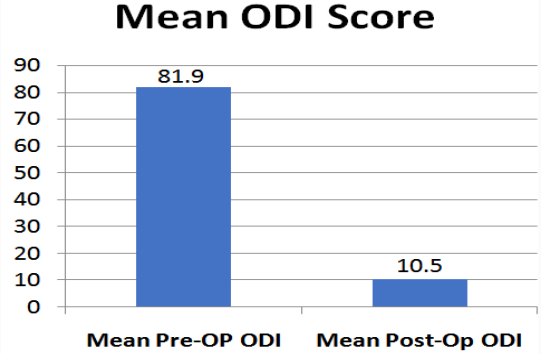

Figure 3: Pre and Post Op Oswestry Disability Index (ODI).

was lumbar canal stenosis. Commonest level involved was L4/5 in 21/40 (52.5%). The commonest operative intervention was Posterior Lumbar Interbody Fusion (PLIF) in 31/41(75.6%) followed by Primary Posterior Lumbar Interbody Fusion (PPLD) 10/41(24.3%). The most important post op complication was deep wound/implant infection in 1/41 (2.4%) but there was also unintended durotomy in 2/41 (4.8%). Mean post op VAS (Figure 2) was 1.76 ± 1.51 ($P \leq 0.05$). 34(87.8%) recorded VAS, ≤ 2 but 5 (12.2%) had VAS ≥ 3. Mean post op ODI (Figure 3) was 10.5 ± 14.24. 27 (65.8%) recorded significant improvements in the ODI but quite a number (Table 3) still had ODI score below their expectation.

Discussion

In the three years under review, 41 patients met the selection criteria having been followed up for at least one year. This period of

Figure 4: MRI of Degenerative Disease of the Lumbar Spine.

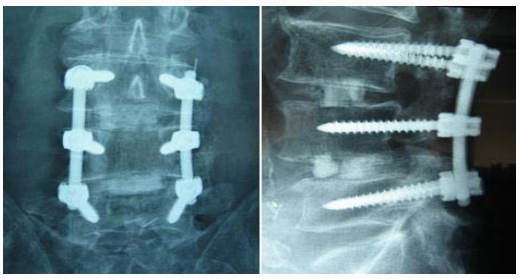

Figure 5: Post op check X-ray of the L/S spine following instrumented fusion with pedicle screws and cage.

Table 3: Post op VAS And ODI.

VAS Score	Frequency(n=41)	Percentage (%)
≤ 2	36	87.8
>2	5	12.2
Mean Score ± SD		**1.76 ± 1.51**
0-20	27	65.8
21-40	11	26.8
61-80	2	4.8
81-100	1	2.4
Mean ODI score		**10.5 ± 14.24**

time was considered reasonable to obtain a fair representation of the outcome of the surgical intervention based on patient perception and realistic expectations. At this time, most of the inflammations associated with post-surgical changes and healing process would have subsided significantly to allow for a fair assessment of both VAS and ODI in comparison to pre-op scores. In a similar by Yang Cong et al. [14] both parameters assessed at one year was equally considered representative. This study revealed that females were more affected than male (F:M=1.15:1). It also showed that the mean age of those affected was 50 years with a Standard Deviation (SD) of 10.9 years. This demographic features is similar to a related study by Andrew et al. [15], where females demonstrated a significantly increased risk of lumbar Disc Degenerative Disease (DDD) when compared to men 1.21 (95% CI 1.18 to 1.23) and those aged >40 were found to be at greatest risk, with an adjusted IRR of 32.53 (95% CI 29.72 to 35.60). Few studies have focused on the role of gender in the development of lumbar degenerative disease and they come up with conflicting reports. Takatalo et al. [16] reported that men were at greater risk for developing lumbar degeneration than women, while Evans W [17] found women to be at greater risk than men. The study published by Kanayama et al. [18] found no particular sex predilection. Others postulated that the lifetime risk of developing the condition is the same between sexes, but that women probably have onset 10 years later than men [19]. Individuals aged 40 and over were found to have

the greatest risk, with an adjusted incidence of 32.5. A number of prior studies have identified patient age as an important factor in the development of degenerative disease [20]. Videman [21] reported that magnetic resonance imaging findings of lumbar degenerative disease, including decreased signal intensity, loss of disc height, and osteophyte formation, were associated with increasing patient age. Similarly, Boos et al. [22] histologically correlated degenerative changes within the lumbar disc with increased patient age in a postmortem study. Pathophysiologically, it is postulated that with aging, the collagen content of the nucleus pulposus increases and transforms from type II to type I. This renders the nucleus more fibrous. Non-collagenous proteins in the nucleus increase subsequently coalesce with the annulus. The increased collagen-proteoglycan binding leaves fewer polar groups of the proteoglycans available to bind water and this makes the disc material non-compliant, progressively more solid, dry and granular [23], the collagen lamellae of the annulus increase in thickness and become increasingly fibrillated. Cracks and cavities may develop within it and present weak points for rupture and extrusion of the nucleus pulposus. This could potentially narrow the intervertebral foramen or the actual spinal canal and compress/irritate the traversing neural elements including cauda equina and nerve roots. The appearance of progressive ligamentum flavum hypertrophy posterior-laterally further compromises spinal canal and foramina to worsen the nerve entrapment symptoms. Facet joint hypertrophy also may contribute occasionally. The commonest symptom was radicular low back pains as recorded in 22 (53.65%). It is due to pressure induced ischemic changes in a nerve root with symptoms transmitted to the dermatomal or myotomal distribution of same nerve root. These include numbness, lancinating pains, parasthesias and motor weakness. Neurogenic claudication featured prominently in 12 (29.2%). Neurogenic claudication arises from ischemia of lumbosacral nerve roots and sustained increase in metabolic demands due to persisting pressure from surrounding structures and is highly specific for spinal stenosis [24]. Features of neurogenic claudication include pain, paresthesia, weakness, and muscle cramps especially in the calves all precipitated by walking but relieved by resting and ad opting a forward bend position. It is quite distinct from vascular claudication. In 7 (17%) the disease had progressed to motor involvement paraparesis, bowel and bladder dysfunction. These happen in the context of cauda equina compression below the conus medullaris. It may be a spectrum of urinary retention, through urinary overflow incontinence with or without fecal incontinence [25]. Mean time to presentation was 15.33 ± 12.09 months. 25 (60%) presented within a year of onset of symptoms but only 4 (10%) presented after two years. Late presentation when symptoms have become disabling is usually due to several prior consultations with GPs, physiotherapists, self-medications, prayer houses and faith healing centers. Mean pre-op VAS was 8.07 ± 0.712. 32 (78%) recorded VAS>7. Mean pre-op ODI score was 81.95 ± 12.11. 28 (68.2%) recorded ODI of 81 to 100 range. This high scoring on both parameters (VAS and ODI) confirm that the degenerative disease had attained advanced stages before presentation to the definitive care provider. Commonest pathology was lumbar canal stenosis. It involves the narrowing of Anteroposterior (AP) dimension of the spinal canal below a critical value (Figure 4). It is most commonly acquired and due to facet hypertrophy, ligamentum flavum hypertrophy, osteophytosis, disc herniation, collapse of disc space and spondylolisthesis. It may sometimes be superimposed on congenital narrowing of the spinal canal. This stenosis may sometimes extend laterally to compromise the intervertebral foramen with a resultant entrapment of exiting nerve root to cause profound radiculopathy; otherwise central stenosis will usually cause neurogenic claudication with or without urinary bladder or bowel dysfunction. It is usually a stable spine disease but can potentially become unstable when associated with spondylolisthesis. Commonest level involved was L4/5 in 21/40 (52.5%). This is in agreement with findings in a similar study where symptomatic lumbar stenosis was found to be most common at L4/526. It is important to stress that Magnetic Resonance Imaging (MRI) is the gold standard in establishing diagnosis of lumbar canal stenosis due to high soft tissue resolution, and sensitivity and specificity similar to CT Myelography [27]. Lumbar spinal stenosis usually responds to decompressive surgery which may require instrumented fusion sometimes. The goal of surgery was to achieve pain relief, slow down or stop progression of symptoms and possibly improve quality of live. The commonest operative intervention was Posterior Lumbar Interbody Fusion (PLIF) in 31/41 (75.6%) followed by Primary Posterior Lumbar Decompression (PPLD) 10/41 (24.3%). PLIF basically involves instrumented fusion of the lumbar spine following decompression using pedicle screws, lumbar cages and bone grafts (Figure 5). Posterior decompression basically involves widening the lumbar canal and intervertebral spaces by the removal of all elements compressing the neural structures through a midline posterior approach and laminectomy. These include hypertrophied ligamentum flavum, medial third facet joints, herniated disc, osteophytes etc. Discectomy provides a space in between the vertebral bodies which can should be filled with a cage impregnated with cancellous bone to stimulate natural fusion. Instrumentation with pedicle screws is necessary when spine instability becomes a potential threat. Dynamic X-Rays with lateral view of lumbo-sacral spine in full flexion and full extension positions predicts risk of spondylolisthesis following decompression and therefore need for providing additional support with instrumented fusion. It is an established fact that spondylolisthesis may occur without decompression but is more common following surgery [28]. Primary Posterior Lumbar Decompression without fusion (PPLD) was conducted in 10/41 (24.3%). It involves straightforward decompression without instrumented fusion as dictated by the presenting pathology and clinical judgment such as laminectomy, foraminotomy, microdiscectomy etc. There is usually no threat of spine instability pre-operatively or potential risk of destabilizing the spine by the decompressive surgery.

Postoperative spine infection can be a devastating complication after spine surgery in both the short term and long-term basis. The most important post op complication in this study was deep wound/implant infection in 1/41 (2.4%). This compares with a similar study which recorded infection rate of 0.9% to 5%, ranging from superficial to severe dehiscent wound infection and deeper infection including discitis, osteomyelitis, and epidural abscess [29]. Deep infection following instrumented spine surgery will almost invariably require removal of all implants and prolonged IV antibiotics. This was the unfortunate situation with the lone case recorded in this study. As previously reported, deep infection with hardware in place is treated with 4 to 6 weeks of IV antibiotics based on culture and sensitivity and guided by inflammatory markers [30]. Removal of implants may occasionally become necessary. Unintended durotomy was recorded in two cases (4.8%). The incidence of incidental durotomies vary amongst authors (1% to 17%) but generally depends on the type and complexity of the spinal procedures [31]. Cerebrospinal Fluid (CSF) leaks following unintended durotomy are frequently detected during the initial surgical procedure. In these cases, they are immediately

repaired with suture, fibrin glue, autologous muscle or fascial graft, heterologous dural graft [32]. Occasionally they present during the post-operative period and pose potentially serious complications such as CSF fistula formation, pseudomeningocele, meningitis, arachnoiditis and epidural abscess [33]. For the two cases recorded in this study, the dural tear was detected and repaired immediately with 6-0 prolene under microdissection. No post op CSF leak through the wound was observed and there was no pseudomeningocele or any further complications of concern. Although it appears as a setback during the procedure, calm, confidence and patience must be exercised to deal with the repair whenever possible primarily. Outcome of this surgical intervention was the main focus of this study. From m the mean post op VAS of 1.76 ± 1.51 compared to pre-op VAS of 8.07 ± 0.712, it is clear that a statistically significant improvement in pain score was achieved (P= <0.05). Similarly, mean post op ODI 10.5 ± 14.24 in comparison to mean pre-op ODI score of 81.95 ± 12.11 also confirmed a statistically significant improvement in quality of life following surgical interventions ($P \leq 0.05$). 27 (65.8%) recorded significant improvements in the ODI ($P \leq 0.05$) but quite a number (Table 3) still had ODI score below their expectation. Whereas 11/41 (26.8%) functioned at ODI of 21% to 40% at one year, 2/41 (4.8%) functioned at ODI of 61% to 80% and 1/41 maintained pre-op levels of ODI at 81% to 100%. It was interesting to note that in spite of residual back pains and restricted quality of live most except the latter returned to work and activities of daily living with analgesics, physiotherapy and postural exercises. One patient maintained ODI of 81 to 100 ranges in spite of all post op pain management and was unable to return to work at one year and therefore fulfilled all criteria for Failed Back Surgery Syndrome. It has been documented that the failure for lumbar decompressive surgery to provide satisfactory long-term pain relief was 8% to 25% depending on the nature and complexity of the primary indication and pre-op morbidity [34]. For this category of patients, further operative intervention is not advisable due to high complications and a more conservative approach such as pain management is thus preferable.

Conclusion

From the foregoing findings and discussion the mean post op VAS of 1.76 ± 1.51 compared to pre-op VAS of 8.07 ± 0.712 and a mean post op ODI 10.5 ± 14.24 in comparison to mean pre-op ODI score of 81.95 & plus mn; 12.11 both confirmed a statistically significant improvement in pain perception and quality of life following surgical interventions ($P \leq 0.05$). Although a limitation here is the retrospective nature, on balance of evidence, PLIF and PPLD are standardized procedures for patients who suffer advanced degenerative disease of the lumbar spine and should be deployed when the need arises. They are safe procedures with reasonably good outcomes. However, it is important to remember during consent discussions that there remains a small but ethically important fraction of patients who recorded ODI above 61 to 80 with residual pains and marked impairment in quality of live in spite of these surgical interventions.

References

1. Chapman R, Norvell DC, Hermsmeyer JT, Bransford RJ, DeVine J, McGirt MJ. Evaluating common outcomes for measuring treatment success for chronic low back pain: Spine (Phila Pa 1976). 2011:1;36(21 Suppl):S54-S68.
2. Haefeli M, Elfering A, Aebi M, Freeman BJ, Fritzell P, Guimaraes Consciencia J, et al. What Comprises a Good Outcome in Spinal Surgery? A Preliminary survey among spine surgeons of the SSE and European Spine Patients. Eur Spine J. 2008;17(1):104-16.
3. Longo UG, Loppini M, Denaro L, Maffulli N, Denaro V. Rating Scales for Low Back Pain. Br Med Bull. 2010;94:81-144.
4. Djurasovic M, Carreon LY, Crawford CH 3rd, Zook JD, Bratcher KR, Glassman SD. The Influence of Preoperative MRI Findings on Lumbar Fusion Clinical Outcomes. Eur Spine J. 2012;21(8)1616-23.
5. Djurasovic M, Glassman SD, Dimar JR 2nd, Howard JM, Bratcher KR, Carreon LY. Does fusion status correlate with patient outcomes in lumbar spinal fusion? Spine (Phila Pa 1976), 2011;36(5):404-9.
6. Fairbank JC. Why are there different versions of the oswestry disability index? J Neurosurg Spine. 2014;20(1):83-6.
7. Fairbank JC, Pynsent PB. The oswestry disability index. Spine (Phila Pa 1976). 2000:25(22):2940-52.
8. Hagg O, Fritzell P, Nordwall A; Swedish lumbar spine study group. The clinical importance of changes in outcome scores after treatment for chronic low back pain. Eur Spine J. 2003;12(1):12-20.
9. Roland M, Morris R. A study of the natural history of back pain. Part I: Development of a reliable and sensitive measure of disability in low-back pain. Spine (Phila Pa 1976). 1983;8(2):141-4.
10. Rodrigues MF, Michel-Crosato E, Cardoso JR, Traebert J. Psychometric properties and cross-cultural adaptation of the brazilian quebec back pain disability scale questionnaire. Spine (Phila Pa 1976). 2009;34(13):E459-64.
11. Weinstein JN, Tosteson TD, Lurie JD, Tosteson A, Blood E, Herkowitz H, et al. Surgical *versus* nonoperative treatment for lumbar spinal stenosis four-year results of the spine patient outcomes research trial. Spine (Phila Pa 1976). 2010;35(14):1329-38.
12. Zaina F, Tomkins-Lane C, Carragee E, Negrini S. Surgical *versus* nonsurgical treatment for lumbar spinal stenosis. Spine. 2016;41(14):E857-68.
13. Scheer JK, Smith JS, Clark AJ, Lafage V, Kim HJ, Rolston JD. Comprehensive study of back and leg pain improvements after adult spinal deformity surgery: Analysis of 421 patients with 2-year follow-up and of the impact of the surgery on treatment satisfaction. J Neurosurg Spine. 2015;22(5):540-53.
14. Cong Y, Lian H, Hu L, Zhang B, Lu J. Efficacy of lumbar fusion in treating lumbar degenerative disease. Int J Clin Exp Med. 2017;10(12):16395-401.
15. Andrew J Schoenfeld MC, James H. Nelson robert burks, LTC Philip J Belmont Jr. Incidence and risk factors for lumbar degenerative disc disease in the united states military 1999–2008. Mil Med. 2011;176(11):1320-4.
16. Takatalo J, Karppinen J, Niinimäki J, Simo T, Simo N, Majro-Riitta J, et al. Prevalence of degenerative imaging findings in lumbar magnetic resonance imaging among young adults. Spine. 2009;34(16):1716-21.
17. Evans W, Jobe W, Seibert C. A Cross-sectional prevalence study of lumbar disc degeneration in a working population. Spine. 1989;14(1):60-4.
18. Kanayama M, Togawa D, Takahashi C, Terai T, Hashimoto T. Cross-sectional magnetic resonance imaging study of lumbar disc degeneration in 200 healthy individuals. J Neurosurg Spine. 2009;11(4):501-7.
19. Battié MC, Videman T. Lumbar disc degeneration: Epidemiology and genetics. J Bone Joint Surg Am. 2006;88 (Suppl 2):3-9.
20. Vernon-Roberts B, Moore RJ, Fraser RD. The natural history of age-related disc degeneration: The pathology and sequelae of tears. Spine. 2007;32(25):2797-804.
21. Videman T, Battié MC, Gill K, Manninen H, Gibbons LE, Fisher LD. Magnetic resonance imaging findings and their relationships in the thoracic and lumbar spine. Insights into the etiopathogenesis of spinal degeneration. Spine. 1995;20(8):928-35.

22. Boos N, Weissbach S, Rohrbach H, Weiler C, Spratt KF, Nerlich AG. Classification of age-related changes in lumbar intervertebral discs: 2002 volvo award in basic science. Spine. 2002;27(23):2631-44.

23. Vernon-Roberts B, Pirie CJ. Degenerative changes in the intervertebral discs of the lumbar spine and their sequelae. Rheumatol rehab. 1977;16(1):13-21.

24. Turner JA, Ersek M, Herron I, Deyo R. Surgery for lumbar spinal stenosis: Attempted meta-analysis of the literature. Spine. 1992;17(1):1-8.

25. Kostuik JP, Harrington I, Alexander D, Rand W. Cauda equina syndrome and lumbar disc herniation. J Bone and Joint Surg Am. 1986;68(3):386-91.

26. Epstein NE, Maldonado VC, Cusick JF. Symptomatic lumbar spina stenosis. Surg Neurol. 1998;50(1):3-10.

27. Jackson RP, Cain JE, Jacobs RR, Cooper BR, McManus GE. The neuroradiologic diagnosis of lumbar herniated nucleus pulposus: A comparison of Computed Tomography (CT), Myelography, CT-myelography and magnetic resonance imaging. Spine.1989;14(12):1362-7.

28. Silvers HR, Lewis PJ. Decompressive lumbar laminectomy for spinal stenosis. J Neurosurg. 1993;78(5):695-701.

29. Shektman A, Granick MS, Solomon MP, Black P, Nair S. Management of infected laminectomy wounds. Neurosurgery.1994;35(2):307-9.

30. Dowdell J, Brochin R, Kim J, Overley S, Oren J, Freedman B, et al. Postoperative spine infection: Diagnosis and management. Global Spine J. 2018;8(4 Suppl): 37S-43S.

31. Wang JC, Bohlman HH, Riew KD. Dural tears secondary to operations on the lumbar spine. Management and results after a two-year-minimum follow-up of eighty-eight patients. J Bone Joint Surg Am. 1998;80(12):1728-32.

32. Bosacco SJ, Gardner MJ, Guille JT. Evaluation and treatment of dural tears in lumbar spine surgery: A Review. Clin Orthop Relat Res. 2001;(389):238-47.

33. Cammisa FP Jr, Girardi FP, Sangani PK. Incidental durotomy in spine surgery. Spine (Phila Pa 1976). 2000;25(20):2663-7.

34. Law Jd, Lehman Raw, Kisch WM. Reoperation after lumbar intervertebral disc surgery. J Neurosurg. 1978;48(2):259-63.