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Retrospective Evaluation

Is the History of a Surgical Discectomy Related to the Source of Chronic Low Back Pain?

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Background: Recurrent or persistent low back pain (LBP) after surgical discectomy (SD) for intervertebral disc herniation has been well documented. The source of low back pain in these patients has not been examined.

Objective: To compare the distribution of the source of chronic LBP between patients with and without a history of SD.

Study Design: Retrospective chart review.

Setting: Academic spine center.

Patients: Charts from 358 consecutive patients were reviewed. Charts noting the absence/presence of SD in patients who subsequently underwent diagnostic injections to determine the source of chronic LBP were included resulting in 158 unique cases for analysis.

Methods: Patients underwent either dual diagnostic facet joint blocks, intra-articular diagnostic sacroiliac joint injections, provocation lumbar discography, or anesthetic injection into putatively painful interspinous ligaments/opposing spinous processes/posterior fusion hardware. If the initial diagnostic procedure was negative, the next most likely structure in the diagnostic algorithm was interrogated. Subsequent diagnostic procedures were not performed after the source of chronic LBP was identified.

Outcome: The source of chronic LBP was diagnosed as discogenic pain (DP), facet joint pain (FJP), sacroiliac joint pain (SIJP), or other sources of chronic LBP.

Results: Based on a Fisher's exact test, there was marginal evidence the distribution of the source of chronic LBP differed for those with and without a history of SD ($P = 0.080$). Post-hoc comparisons suggested that patients with a history of SD have a higher probability of DP compared to those without a history of SD (82% versus 41%; $P = 0.011$). Differences in the probability of FJP, SIJP, or other sources between the SD history groups were not significant.

Limitations: Small sample size, restrospective design, and possible false-positive results.

Conclusions: This is the first published investigation of the tissue source of chronic LBP after SD. It appears that DP is the most common reason for chronic LBP after SD. If more rigorous study confirms our findings, future biologic treatments may hold value in repairing symptomatic annular fissures after SD.

Key words: surgical discectomy, chornic low back pain, discogenic pain, facet joint, sacroiliac joint, low back pain, diagnostic injections, medial branch block, lumbar provcation discography

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Surgical discectomy is regarded as an effective treatment for lumbosacral radicular pain caused by a corroborative disc herniation that is recalcitrant to less invasive treatment (1-3). Herniated nuclear material occurs when an intradiscal pressure gradient develops in the setting of an annular incompetence or fissure. Removal of the offending hernia and repair of the outer annulus are the surgical objectives for persistent radicular pain (1-4). Internal derangement represented by persistent annular defects may persist after the fibrotic healing process runs its course.

Recurrent or persistent low back pain (LBP) after surgical discectomy (SD) for intervertebral disc herniation has been well documented (5-8). Post-operatively, approximately 20% to 25% of SD patients experience moderate LBP; 9% to 13% experience severe LBP (6-8) as well as a reduced quality of life (9). More severe LBP may be associated with a younger age (< 35 years) at index SD (8). Despite SD patients afflicted with more severe LBP typically undergoing fusion surgery at the SD level (6,8), the source of the LBP in these patients has not been examined..

Post-operative, chronic LBP may be related to persistent discogenic LBP (DP) at the index level or at a different level versus facet joint pain (FJP) or sacroiliac joint pain (SIJP). Understanding specific LBP sources after SD better could help improve direct treatment strategies.

Previous studies have employed diagnostic spinal injections to locate the precise source of chronic LBP [10-23]. The primary purpose of the current study was to compare the distribution of the sources of chronic LBP (DP, FJP, SIJP, or other sources) between patients with and without a history of SD.

Methods

Participants

After Institutional Review Board approval, 378 charts from 358 consecutive chronic LBP patients presenting from November 2007 through December 2008 were reviewed. Enrolled cases were patients suffering from chronic LBP refractory to physical therapy, oral analgesics, and oral anti-inflammatory medications. The patients presented to a community-based, multi-disciplinary, academic spine center and were referred to the spine center from community and university physicians.

Eighteen patients presented with more than one case during the period the charts were reviewed; 16 patients with 2 cases and 2 patients with 3 cases (total of 38 cases). Seven of the 18 patients presented with multiple cases at the same time (6 with 2 cases, and one with 3 cases) and the remaining 11 presented at different times (10 with 2 cases and one with 3). The 7 patients with multiple cases at the same time were excluded since these sources could not be considered independent events. Of the remaining 363 cases from 351 patients, 157 cases underwent diagnostic injections and 206 did not due to clinical improvement in their symptoms. These patients were excluded from the analysis. Thus, the sample used for analysis consists of 157 cases from 153 patients who underwent diagnostic spinal injections to identify the source of their chronic low back pain (CLBP) in order to implement more definitive treatment.

Measures

To determine the source of CLBP, each patient underwent dual diagnostic facet joint blocks (FJB) with local comparative anesthetics, intra-articular diagnostic sacroiliac joint blocks (SIJB), provocation lumbar discography (PLD), or injection of anesthetic into putatively painful interspinous ligaments, opposing spinous processes, or posterior fusion hardware. Some patients underwent multiple diagnostic procedures until the source of their LBP was identified. If the initial diagnostic procedure was negative, the next most likely structure in the diagnostic algorithm was interrogated. However, once a source of the patient's LBP was identified, subsequent diagnostic procedures were not performed.

Patients reporting paravertebral LBP without midline LBP (14,24) whose clinical presentation supported FJP (25-27), typically underwent FJB first, followed by SIJB and then PLD if the preceding diagnostic procedure was negative. The side and joint level selected by pain referral pattern (28,29) were investigated first moving from most likely to less likely facet joint (FJ) level. Patients reporting paravertebral LBP without midline LBP (14,15,30,31) whose clinical presentation supported SIJP (26,27,32) underwent SIJB followed by FJBs and then PLD unless the initial diagnostic blocks were positive. Patients reporting paravertebral LBP with a previous history of posterior fusion with pedicle screws and hardware whose LBP was reproducible by single digit palpation over the hardware, underwent diagnostic blockade of the hardware in a triple blockade fashion using 2% lidocaine first, then 0.5% marcaine, followed by a placebo injection. Patients reporting midline LBP with or without paravertebral LBP, centralization dur-

ing McKenzie evaluation (27), and/or LBP during sustained hip flexion (SHF) (33) underwent PLD initially followed by FJB or SIJB if discography was negative. Patients reporting midline LBP without paravertebral LBP aggravated by standing and walking and not provoked by sitting or SHF with evidence of opposing lumbar spinous processes on imaging, first underwent diagnostic interspinous injection (16,33) of the segmental level supported by the cephalad to caudad location of the LBP. We have previously published our findings using this algorithmic approach (34).

Positive discography was defined as concordant/partial concordant LBP (> 6/10 on a VAS) at low pressure (< 50 psi over opening pressure) due to > Grade III annular tears (35-37). Diagnostic blockade of a facet joint (FJ), sacroiliac joint (SIJ), or other structures was deemed positive if the patient's index pain was relieved by ≥ 75% after injection of each anesthetic (38-40). In the case of fusion hardware blockade, minimal relief after the placebo injection was required to constitute a positive block.

If patients reported pain upon closed fist percussion and had advanced imaging evidence of an active insufficiency fracture, percutaneous augmentation was performed. Based on the results of diagnostic injections or LBP reduction after percutaneous augmentation, patients were classified as having DP, FJP, SIJP, or other sources of LBP (fusion hardware mediated soft tissue pain, Baastrup Disease, or vertebral or sacral insufficiency fractures). The source of chronic LBP was the primary outcome variable of interest in this study. The primary predictor variable was a history of SD (yes/no). History of SD was unavailable from the charts for only 2 patients. The sample of 155 cases that underwent diagnostic procedures and were not missing history of SD was analyzed in this study.

Statistical Analyses

The distribution of the source of chronic LBP was estimated and compared between the groups with and without a history of SD using a Fisher's exact test. If this overall test suggested that the distributions differed, then 4 separate Fisher's exact post-hoc tests were used to compare the probability of each specific source of chronic LBP between the groups. SAS v.9.2 (SAS Institute, Inc., Cary, NC) was used for all data analysis.

Results

Cases of chronic LBP were primarily female (66%), presented at an average age of 54 years (standard deviation [Std] = 16.2), and had a median duration of LBP of 12 months (interquartile range [IQR] = 6 to 36). Based on this sample, the prevalence of chronic LBP was estimated to be 43.9% DP, 31.6% FJP, 16.8% SIJP, and 7.7% other sources. Eleven (7.0%) of the 155 cases had a history of SD.

The distribution of the source of chronic LBP is summarized for cases with and without a history of SD in Table 1. Cases with a history of SD were classified as solely DP or FJP (Table 2). A Fisher's exact test indicated that the distributions of the sources of chronic LBP were marginally different between the 2 groups (P = 0.080). Since the test was at least marginally significant, and due to the exploratory nature of our data, post-hoc comparisons were conducted using 4 separate Fisher's exact tests for 2 x 2 tables (e.g. DP [yes/no] versus SD history [yes/no]). These tests indicated that those with a history of SD have a significantly higher probability of DP (versus any other source) compared to those without a history of SD (82% versus 41%; P value = 0.011). There were not significant differences in the probability of FJP, SIJP, or other sources between the SD history groups (FJP P value = 0.50, SIJP P value = 0.21, other P > 0.99).

Discussion

A history of SD appears to be related to the occurrence of chronic discogenic LBP. Approximately 82% of SD chronic LBP patients who undergo diagnostic procedures have painful annular fissures as the source of

Table 1. *Contingency table of source of low back pain by history of surgical discetomy.*

	Source of LBP				
History of SD	**DP**	**FJP**	**SIJP**	**Other**	**Total**
Yes	9 (81.8%)	2 (18.2%)	0 (0%)	0 (0%)	11
No	59 (41.0%)	47 (32.6%)	26 (18.1%)	12 (8.3%)	147
Total	68	49	26	12	155

Table 2. *Distribution of cases reviewed and excluded.*

Number of patients presenting with cases of chronic low back pain	58
Number of cases reviewed	378*
Total number of patients presenting with more than one case during the review period	18
Number of patients presenting with 2 cases	16
Number of patients presenting with 3 cases	2
Total number of patients presenting with multiple cases at the same point in time during the review period	7**
Number of patients presenting with 2 cases	6
Number of patients presenting with 3 cases	1
Number of patients excluded due to clinical improvement in their symptoms	206**
Number of patients who underwent diagnostic spinal injections	157
Number of patients for which history of SD was unavailable from chart review	2**

*Some participants presented with more than one case during the review period.
**Denotes participants excluded from the study.

their pain. When strict operational criteria and meticulous technique are applied, diagnostic procedures can reveal the source of chronic low back pain in 90% of patients and the source of chronic neck pain in 80% of patients (34,41,42). Internal derangement of the lumbar intervertebral disc has been identified as the most common source of chronic LBP in surgically-naïve spines (10,34). The presence of annular fissures reaching the outer third of the annulus is the strongest predictor of concordant pain during pressure-controlled discography (3543-46). Anesthetization of these fissures reliably reduces index LBP in 80% of cases (47). Therefore, painful annular fissures are probably responsible for chronic LBP symptoms in DP patients.

Once a disc herniates and offends a nearby nerve root, the disc itself can remain symptomatic after surgical removal of the herniated nuclear material. Our data supports this contention corroborating Heggeness' (48) findings suggesting that painful annular fissures still reside within the index disc's annulus. Our data does not allow us to differentiate between true internal derangement (defined as such by the presence of a normal disc external contour) and re-herniation. We did not exclude herniated discs. However, if a herniation was present, it was typically ≤ 4 mm. Nonetheless, even if a re-herniation was the culprit for persistent LBP, it presumably would have been related to the attenuated annular tissue in the previously herniated disc for which surgery was performed. We were not able to collect data on the previous location of the herniation for which SD was performed in order to correlate that with the location of the presenting annular fissure discovered by PLD. However, recurrent herniation after SD has been reported to not necessarily occur at the location of the initial herniation (49).

The incidence of FJP and SIJP were remarkably rare in our SD CLBP cohort. The number of FJP and SIJP SD cases was 2 and zero, respectively, increasing the likelihood that our observed estimates of FJP and SIJP in CLBP patients after SD is not accurate or representative. Notwithstanding this statistical shortcoming, it is interesting that posterior element and pelvic mediated chronic LBP appear much less common than intervertebral disc-related chronic LBP after SD. The mean age of FJP patients observed in our SD chronic LBP cohort (45 years) is categorically younger than the mean age of FJP or SIJP in CLBP patients in previous reports (11,34). The mean age of FJP cases in our SD cohort is below the lower bound of the 95% confidence interval (CI) for FJP in CLBP all-comers (11,34). A myriad of reasons could exist for the younger FJP presentation, ranging from a genetic predisposition (50) for degenerative lumbosacral spine disorders to an escalation in disc degeneration and increased load borne by the posterior elements with loss of disc height (51). However, addressing these queries cannot begin without inclusion of a larger number of FJP/SD subjects.

Significant false positive rates (40%) have been reported with discography in post-discectomy patients (4). Application of stringent operational criteria to these discography data effectively reduces the false positive rate to 15% (95% CI 0-32%) per patient and 9.1% (95% CI

0-19%) per disc (36). Discs having undergone previous surgical discectomy by definition contain outer annular fissures and therefore possess the morphologic substrate for potential DP (43). Over time these annular fissures within the surgically repaired disc may or may not become increasingly symptomatic resulting in worsening pain. If they do remain asymptomatic, stimulation of them by direct injection of contrast dye and distension of the annular fissure may result in LBP that does not meet stringent criteria for a positive discogram.

Conclusion

Despite a small sample size and retrospective nature introducing selection bias, this is the first published investigation of the tissue source of CLBP after SD. It appears that DP is the most common reason for CLBP after SD. If more rigorous study confirms our findings, future biologic treatments may hold value in repairing symptomatic annular fissures after SD.

References

1. Ebeling U, Reichenberg W, Reulen HJ. Results of microsurgical lumbar discectomy: review of 485 patients. *Acta Neurochir* 1986; 81:45-52
2. Wilson DH, Kenning J. Microsurgical lumbar discectomy: Preliminary report of 83 consecutive cases. *Neurosurgery* 1979; 4:137-140.
3. Abramovitz JN, Neff SR. Lumbar disc surgery: Results of the prospective lumbar discectomy study of the Joint Section on Disorders of the Spine and Peripheral Nerves of the American Association of Neurological Surgeons and the Congress of Neurological Surgeons. *Neurosurgery* 1999; 29:301-308.
4. Carragee EJ, Han MY, Suen PW, Kim D. Clinical outcomes after lumbar discectomy for sciatica: The effects of fragment type and anular competence. *J Bone Joint Surg Am* 2003; 85:102-108.
5. den Boer JJ, Oostendorp RA, Beems T, Munneke M, Evers AW. Continued disability and pain after lumbar disc surgery: The role of cognitive-behavioral factors. *Pain* 2006; 123:45-52. Epub 2006 Mar 24.
6. Gepstein R, Shabat S, Reichel M, Pikarsky I, Folman Y. Treatment of postdiscectomy low back pain by percutaneous posterior lumbar interbody fusion versus open posterior lumbar fusion with pedicle screws. *Spine J* 2008; 8:741-746. Epub 2007 Nov 5.
7. Hakkinen A, Ylinen J, Kautiainen H, Airaksinen O, Herno A, Kiviranta I. Does the outcome 2 months after lumbar disc surgery predict the outcome 12 months later? *Disabil Rehabil* 2003; 25:968-972.
8. Yorimitsu E, Chiba K, Toyama Y, Hirabayashi K. Long-term outcomes of standard discectomy for lumbar disc herniation: A follow-up study of more than 10 years. *Spine (Phila Pa 1976)* 2001; 26:652-657.
9. Boskovic K, Cigic T, Grajic M, Todorovic-Tomasevic S, Knezevic A. The quality of life of patients after a lumbar microdiscectomy: A four-year monitoring study. *Clin Neurol Neurosurg* 2010;112:557-562. Epub 2010 May 26.
10. Schwarzer AC, Aprill CN, Derby R, Fortin J, Kine G, Bogduk N. The prevalence and clinical features of internal disc disruption in patients with chronic low back pain. *Spine (Phila Pa 1976)* 1995; 20: 1878-1881.
11. Schwarzer A, Wang SC, Bogduk N, McNaught PF, Lauren R. Prevalence and clinical features of lumbar z joint pain: A study in an Australian population w/ chronic low back pain. Ann Rheum Dis 1995;54:100-6.
12. Manchikanti L, Manchikanti KN, Cash KA, Singh V, Giordano J. Age-related prevalence of facet-joint involvement in chronic neck and low back pain. *Pain Physician* 2008; 11:67-75.
13. Manchukonda R, Manchikanti KN, Cash KA, Pampati V, Manchikanti L. Facet joint pain in chronic spinal pain: An evaluation of prevalence and false-positive rate of diagnostic blocks. *J Spinal Disord Tech* 2007; 20:539-545.
14. Schwarzer AC, Aprill CN, Bogduk N. The sacroiliac joint in chronic low back pain. *Spine (Phila Pa 1976)* 1995; 20:31-37.
15. Maigne JY, Aivaliklis A, Fabrice P. Results of sacroiliac joint double block and value of sacroiliac pain provocation tests in 54 patients with low back pain. *Spine (Phila Pa 1976)* 1996; 21:1889-1892.
16. DePalma MJ, Slipman CW, Siegelman E, Bayruns TJ, Bhargava A, Frey ME, Chin KR. Interspinous bursitis in an athlete: A case report. J Bone Joint Surg Br 2004;86-B(7):1062-4.
17. Knievel S, Lamer T. Midline posterior element disorders. In: DePalma MJ (ed). *iSpine.* Demos Publishing, New York, 2011.
18. Jaffe MJ, Giovanello MT. Successful radiofrequency ablation for Baastrup's disease and intraspinous ligament pain: A case report. Poster presented at: 17th Annual International Spine Intervention Society Scientific Meeting; July 2009; Toronto, CN.
19. Maes R, Morrison WB, Parker L, Schweitzer ME, Carrino JA. Lumbar interspinous bursitis (Baastrup disease) in a symptomatic population: Prevalence on MRI. *Spine (Phila Pa 1976)* 2008; 33:E211-215.
20. Bywaters EG, Evans S. The lumbar interspinous bursae and Baastrup's syndrome: An autopsy study. *Rheumatol Int* 1982; 2:87-96.
21. Newman PH. Sprung back. *J Bone Joint Surg* 1952; 34:30-37.
22. Scapinelli R, Stecco C, Pozzuoli A, Porzionato A, Macchi V, De Caro R. The lumbar interspinous ligaments in humans: Anatomical study and review of the literature. *Cells Tissues Organs* 2006; 183:1-11.
23. Bhargava A. Fusion hardware mediated low back pain. In: DePalma MJ (ed). *iSpine.* Demos Publishing, New York, 2011.
24. Laslett M, McDonald B, Aprill CN, Tropp H, Oberg B. Clinical predictors of screening lumbar zygapophyseal joint blocks: development of clinical prediction rules. *Spine J* 2006; 6:370-379.
25. Revel M, Poiraudeau S, Auleley GR, Pay-

an C, Denke A, Nguyen M, Chevrot A, Fermanian J. Capacity of the clinical picture to characterize low back pain relieved by facet joint anesthesia: Proposed criteria to identify patients with painful facet joints. *Spine* 1998; 23:1972-1976.

26. Laslett M, Aprill CN, McDonald B, Young SB. Diagnosis of sacroiliac joint pain: Validity of individual provocation tests and composites of tests. *Manual Therapy* 2005; 10:207–218.
27. Laslett M, Young SB, Aprill CN, McDonald B. Diagnosing painful sacroiliac joints: A validity study of a McKenzie evaluation and sacroiliac provocation tests. *Australian Journal Physiother* 2003; 49:89-97.
28. Fukui S, Ohseto K, Shiotani M, Ohno K, Karasawa H, Naganuma Y. Distribution of referred pain from the lumbar zygapophyseal joints and dorsal rami. *Clin J Pain* 1997; 13:303-307.
29. Lipetz J. Lumbar Pain – An Algorithmic Methodology. In: Slipman CW, Derby R, Simeone FA, Mayer TG (eds). *Interventional Spine: An Algorithmic Approach*. Elsevier, London, UK, 2007, pp 975-990.
30. Fortin J, Aprill C, Ponthieux B, Pier J. Sacroiliac joint: Pain referral maps upon applying a new injection/arthrography technique. Part II: Clinical evaluation. *Spine* 1994; 19:1483-1489.
31. Fortin J, Dwyer A, West S, Pier J. Sacroiliac joint: Pain referral maps upon applying a new injection/arthrography technique. Part I: Asymptomatic volunteers. *Spine* 1994; 19:1475-1482.
32. van der Wurff P, Buijs EJ, Groen GJ.A multitest regimen of pain provocation tests as an aid to reduce unnecessary minimally invasive sacroiliac joint procedures. *Arch Phys Med Rehabil* 2006; 87:10-14.
33. Patel RK, Slipman CW. Lumbar degenerative disk disease. eMedicine Specialties Physical Medicine and Rehabilitation, January 18, 2007. http://www.emedicine.com/pmr/ topic67.htm.
34. DePalma MJ, Ketchum JM, Saullo TR. What is the source of chronic low back pain and does age play a role? *Pain Med* 2011; 12:224-233.
35. Derby R, Kim BJ, Chen Y, Seo, KS, Lee SH. The relation between annular disruption on computed tomography scan and pressure-controlled discography. *Arch Phys Med Rehabil* 2005; 86:1534-1538.
36. Wolfer LR, Derby R, Lee JE, Lee SH. Systematic review of lumbar provocation discography in asymptomatic subjects with a meta-analysis of false-positive rates. *Pain Physician* 2008; 11:513-538.
37. Lumbar disc stimulation (provocation discography). In: Bogduk N, ed. *Practice Guidelines. Spinal Diagnostic and Treatment Procedures.* International Spinal Intervention Society, San Francisco, CA, 2004, pp 20–46.
38. Bogduk N, Holmes S. Controlled zygapophysial joint blocks: The travesty of cost-effectiveness. *Pain Med* 2000; 1:25-34.
39. Manchikanti L, Pampati V, Fellows B, Bakhit CE. The diagnostic validity and therapeutic value of lumbar facet joint nerve blocks with or without adjuvant agents. *Curr Rev Pain* 2000; 4:337-344.
40. Slipman CW, Issac Z. The role of diagnostic selective nerve root blocks in the management of spinal pain. *Pain Physician* 2001; 4:214-226.
41. Yin W, Bogduk N. The nature of neck pain in a private pain clinic in the United States. *Pain Med* 2008; 9:196-203.
42. Lord SM, Barnsley L, Wallis BJ, Bogduk N. Chronic cervical zygapophysial joint pain after whiplash: A placebo-controlled prevalence study. *Spine (Phila Pa 1976)* 1996; 21:1737-1745.
43. Moneta GB, Videman T, Kaivanto K, Aprill C, Spivey M, Vanharanta H, Sachs BL. Guyer RD, Hochschuler SH, Raschbaum RF, Mooney V. Reported pain during lumbar discography as a function of anular ruptures and disc degeneration. A re-analysis of 833 discograms. *Spine (Phila Pa 1976)*1994; 19:1968–1974.
44. Vanharanta H, Sachs BL, Spivey MA, Guyer RD, Hochschuler SH, Raushbaum RF, Johnson RG, Ohnmeiss D, Mooney V. The relationship of pain provocation to lumbar disc deterioration as seen by CT/ discography. *Spine (Phila Pa 1976)*1987; 12:295-298.
45. Vanharanta H, Sachs BL, Spivey M, Guyer RD, Hochschuler SH, Raushbaum RF, Johnson RG, Ohnmeiss D, Mooney V. A comparison of CT/discography, pain response and radiographic disc height. *Spine (Phila Pa 1976)*1988; 13:321-324.
46. Sachs BL, Vanharanta H, Spivey MA, Guyer RD, Videman T, Raushbaum RF, Johnson RG, Hochschuler SH, Mooney V. Dallas discogram description. A new classification of CT/discography in low-back disorders. *Spine (Phila Pa 1976)*1987; 12:287-294.
47. DePalma MJ, Lee JE, Peterson L, Wolfer L, Ketcum J, Derby R. Are Outer Annular Fissures Stimulated During Diskography the Source of Diskogenic Low-Back Pain? An Analysis of Analgesic Diskography Data. *Pain Med* 2009; 10:488-494.
48. Heggeness MH, Watters, WC, Milton GP. Discography of Lumbar Discs After Surgical Treatment for Disc Herniation. *Spine (Phila Pa 1976)* 1997; 22:1606-1609.
49. Wera GD, Marcus RE, Ghanayem AJ, Bohlman HH. Failure within one year following subtotal lumbar discectomy. *J Bone Joint Surg Am* 2008; 90:10-15.
50. Battie MC, Videman T, Patent E. Lumbar disc degeneration: Epidemiology and genetic influences. *Spine (Phila Pa 1976)* 2004; 29:2679-2690.
51. Pollintine P, van Tunen MSLM, Luo J, Brown MD, Dolan P, Adams, MA. Time-dependent compressive deformation of the ageing spine: Relevance to spinal stenosis. *Spine (Phila Pa 1976)* 2010; 35:386-394.