

OPERATIVE MANAGEMENT OF LUMBAR DISC HERNIATION

Momir Jovanović PhD¹; Mojsije Radović MSc²; Vesna Simić, PhD²;

1-The Academy of Applied Preschool Teaching and Health Studies, Department of Ćuprija, Rep. Serbia,
mj_mj63@gmail.com

2-General Hospital Ćuprija, Rep. Serbia

Abstract Lumbar disc herniation (LDH) consists of displacement of the content of the pulposus nucleus through its external membrane fibrous ring, generally in its posterolateral region. This study presents an evaluation of surgical treatment of LDH in GH Ćuprija and discusses possible predictive factors for the outcome of surgical intervention. This study sample consists of 200 patients which underwent surgical treatment of LDH between 2018 and 2020. yr. Test and parameters which were included were: neurological examination, CT and NMR scan evaluation and electromyoneurography (EMNG). All patients were verticalized the day after surgical treatment and all patients underwent early physical rehabilitation followed with medical treatment. This result with 138 (69%) neurological deficit regression in first seven days after surgery treatment (avg. hospitalization time was seven days). After 1 month from surgery treatment, 179 patients had fully recovered. This study reveals importance of early and timely set diagnosis correlated with early physical rehabilitation and adequate medical therapy.

Key words: Lumbar disc herniation, Open discectomy, Neurological deficit, Recovery

Introduction

Lumbar disc degeneration is defined as the wear and tear of lumbar disc that act as a cushion for the spine. Lumbar disc degeneration can occur at any level, but mainly, it occurs on L4-L5 and L4-S1 vertebrae [1, 2]. It begins with small tears in the annulus of the disc to a decrease in the water content of the nucleus pulposus of the discs. The degenerative disc leads to disc bulging, osteophytes, disc space loss, and compression and irritation of the adjacent nerves [3]. With advanced degeneration, it loses water content and disc height, and it leads to segmental instability and causes degenerative spondylosis and scoliosis. The advanced degenerative changes affect disc facet joints and surrounding soft tissue and can result in canal narrowing also known as degenerative stenosis [3]. Because each lumbar disc is in direct contact with two or three pairs of dorsal roots, disc degeneration may compress the adjacent nerve root [4, 5]. This can cause the pain syndrome but, more characteristically, causes neuropathic pain and neurological symptoms and, in severe cases, dysfunction of the nerve. Risk factors causing lumbar disc degeneration disease and associated lumbosacral nerve compression includes advancing age, socioeconomic status [6], torsional stress [7], smoking, obesity [8–10], heavy lifting, vibration [9], trauma, immobilization [10], psychosocial factors, gender, height, hereditary, genetic factors [8, 9], and occupations like machine drivers,

carpenters, and office workers [11–12]. Genetic inheritance plays a significant role in the rate of degradation. Approximately 50–70% disc degeneration is caused by an individual’s genetic inheritance [12, 13]. Disc degeneration becomes prevalent and common in the individual’s 40s and usually in the lower lumbar spine. Some individuals, however, can become afflicted by this disease much earlier than the norm, depending on both the severity of their genetic deficiencies and lifestyles.

Patients and methods

This study covers 200 patients, which underwent surgical treatment in General Hospital of Ćuprija, department of neurosurgery, in period between 2018.-2020. y. These were patients with chronic and acute back pain. This study contains patients of age between 17-73 years. In total there were 132 male and 68 female patients (Figure 1.).

	Male	Female
Number of patients	132	68
Average age	40,3	46,6

Figure 1.

Diagnostic procedures which were used in diagnosis setting were: good and extensive anamnesis, detailed neurological exam, auxiliary, MSCT (multi slice computer tomography), NMR (nuclear magnetic resonance), EMNG (electromyoneurography) etc.

There were totally 200 patients with different symptoms and neurological damage and deficit. in figure 2. we can see difference and correlation between neurological deficit, pain, sensor deficit, and side of damaged leg.

	Left side	Right side	Total
Only pain	8	11	18
Peroneal musculature deficit	40	43	83
Tibial musculature deficit	47	52	99

Figure 2.

Medical therapy which follow surgery procedure contained multivitamin therapy (200 patients) combined with proteolytic enzymes (195 patients (5 made allergic reaction)) and glucocorticoide (preoperative management 43 patients).

Operative procedure which were used were open discectomy followed by interhemilaminectomy and foraminotomy. In total there were 179 one level surgery procedure, and 21 several level procedures (20 procedures with two level procedure and 1 with tree level).

Postoperative management contain early rehabilitation procedure. Early rehabilitation procedure implies learning safety position during getting up and different exercise for strengthening leg, pelvic and paravertebral musculature. It contain continual exercise which implies everyday work between 45 and 60 minutes. The day after surgical management patient walking with support. Next day patient walk without support and every next day introduce longer strengthening exercise. Physical rehabilitation start at 7th day and include laser and electrotherapy. Postoperative rehabilitation were followed with proteolytic and multivitamin therapy.

Recovery success correlated with postoperative time shown in figure 3.

	10 days .a.op.	21days a.op.	3 month ž .a.op.	6 month a.op.
Pain - expiring	152	168	181	192
Tibial deficit - expiring	68	75	78	80
Peroneal deficit - expiring	55	62	67	71

Figure 3.

Recovery success most commonly depend on time of setting diagnosis. Late diagnosis, which include more then 2 or 3 weeks of persisting neurological deficit, reduce recovery success dramatically, which is shown in Figure 4.:

	1 week	2 weeks	3 weeks	4 weeks
Tibial deficit – persisting	0	2	7	13
Peronealdefict - persisting	1	3	9	15

Figure 4.

Postoperative complication were very rare. It contain 2 surface infections complication, where both were successfully recovered after antibiotic therapy. There were no complications like nerve injury, dural puncture, epi/peridural abscess, paralysis, spondylodiscitis etc.

During data collection we notced an association between lumbal disc herniation and cardiovascular diseases, primarily hypertension, dominantly in younger patients.

The cause is most likely in connective tissue weakness.

Figure 5. show correlation between a. hypertension and lumbal disc herniation:

Age	Patients with hypertension	Patients with lumbar disc herniation
<20	2	6
20-30	11	24
30-40	37	63
40-50	42	67
50>	33	40

Figure 5.

Conclusion

Lumbar disc herniation is a common etiology in low back pain. In the last years, significant advances have been made in our understanding in the etiology of LDH including microstructural changes, molecular pathways, and microbial load. Additionally, over this same time period, minimally invasive approaches to LDH resection have demonstrated increasingly positive outcomes. As such, this approach remains a key area of investigation in the coming years in addition to better defining the absolute indications for maximal clinical benefit in surgical treatment of LDH. Correlation between arterial hypertension and LDH is obvious but not enough examined, which deserves more attention research.

References

1. Andersson GB. Epidemiological features of chronic low-back pain. *Lancet*. 1999;354(9178):581–585. doi: 10.1016/S0140-6736(99)01312-4.
2. Martin BI, Deyo RA, Mirza SK, et al. Expenditures and health status among adults with back and neck problems. *JAMA*. 2008;299(6):656. doi: 10.1001/jama.299.6.656.
3. Kadow T, Sowa G, Vo N, Kang JD. Molecular basis of intervertebral disc degeneration and herniations: what are the important translational questions? *ClinOrthopRelat Res*. 2015;473(6):1903–1912. doi: 10.1007/s11999-014-3774-8.
4. Kepler CK, Ponnappan RK, Tannoury CA, Risbud MV, Anderson DG. The molecular basis of intervertebral disc degeneration. *Spine J*. 2013;13(3):318–330. doi: 10.1016/j.spinee.2012.12.003.
5. Kalb S, Martirosyan NL, Kalani MYS, Broc GG, Theodore N. Genetics of the degenerated intervertebral disc. *World Neurosurg*. 2012;77(3–4):491–501. doi: 10.1016/j.wneu.2011.07.014.
6. Urban JPG, Roberts S. Degeneration of the intervertebral disc. *Arthritis Res Ther*. 2003;5(3) 10.1186/ar629.
7. Casiano VE, Dydyk AM, Varacallo M. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Jul 12, 2021. Back Pain.
8. De Cicco FL, Camino Willhuber GO. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Jul 18, 2021. Nucleus Pulposus Herniation.
9. Dydyk AM, Khan MZ, M Das J. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Jul 12, 2021. Radicular Back Pain.
10. Camino Willhuber GO, Piuze NS. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Jul 18, 2021. Straight Leg Raise Test
11. Petersen T, Laslett M, Juhl C. Clinical classification in low back pain: best-evidence diagnostic rules based on systematic reviews. *BMC MusculoskeletDisord*. 2017 May 12;18(1):188.
12. Notohamiprodjo S, Stahl R, Braunagel M, Kazmierczak PM, Thierfelder KM, Treitl KM, Wirth S, Notohamiprodjo M. Diagnostic accuracy of contemporary multidetector computed tomography (MDCT) for the detection of lumbar disc herniation. *EurRadiol*. 2017 Aug;27(8):3443-3451.
13. Yu LP, Qian WW, Yin GY, Ren YX, Hu ZY. MRI assessment of lumbar intervertebral disc degeneration with lumbar degenerative disease using the Pfirrmann grading systems. *PLoS One*. 2012;7(12):e48074.

