Early Postoperative Iatrogenic Neuropraxia After Lumbar Disc Herniation Surgery: Analysis of 87 Cases

Ziya Asan

BACKGROUND: **Postoperative early neuropraxia after lumbar disc herniation surgery is common. The emergence of new paresthesia findings with increased sensory and motor deficits in the postoperative period suggests iatrogenic neuropraxia. This study aimed to discuss the causes and prognosis of iatrogenic neuropraxia detected in the early postoperative period in patients who have been operated on for lumbar disc herniation.**

METHODS: **Cases with postoperative iatrogenic neuropraxia were determined retrospectively. Deficits were evaluated at intervals of 0–2 hours, 2–12 hours, 12–24 hours, and 24–48 hours. The cases were evaluated in 2 groups as those who underwent aggressive discectomy and simple discectomy. In addition, the treatment results were compared between the 2 groups as the cases that were treated and not treated with methylprednisolone.**

RESULTS: **The iatrogenic neuropraxia rate was significantly higher in patients who underwent aggressive discectomy. Although it was observed that paresthesia findings improved more rapidly in cases treated with methylprednisolone, no difference was found between the 2 groups in terms of its effects on the motor deficit.**

CONCLUSIONS: **Iatrogenic neuropraxia is a finding whose cause cannot be determined by quantitative criteria. It is common in patients who underwent aggressive discectomy. Methylprednisolone treatment is effective in recovering the paresthesia finding faster and may show that the radicular injury is in the neuropraxia stage in the early period.**

INTRODUCTION

Postoperative early neuropraxia is a common manifestation after lumbar disc herniation (LDH) surgery.[1-3] Early neuropraxia may go unnoticed due to the rapid recovery of deficits making the diagnosis difficult. Although sensory deficits are the most common sign of neuropraxia, motor deficits are also common. Detection of a motor deficit on the surgical side immediately after the surgery or during the recovery period from anesthesia is the most common and rapidly resolving motor neuropraxia. It shows total improvement within the first minutes or hours.

Sensory neuropraxia lasts longer. Sensory deficits associated with disc herniation are associated with radicular compression or traction and are mainly in the form of sensory loss.[1,3,4] The neuropraxia encountered in the postoperative period is mainly in the form of increased sensory loss and burning or stinging paresthesia. Newly occurring paresthesia findings suggest iatrogenic neuropraxia.

This study aimed to discuss the neuropraxia detected in the early postoperative period in patients after LDH surgery and to discuss the early and late response to the treatments applied.

MATERIAL AND METHODS

The data of the cases operated for LDH between 2013 and 2022 were determined retrospectively by examining the service records. Cases with neuropraxia in the early postoperative period were recorded. The demographic data of the cases were recorded by creating a database in an Excel file. The lumbar magnetic resonance imaging examinations of the cases were examined, and the

Key words
- Iatrogenic neuropraxia
- Lumbar disc herniation
- Lumbar disc surgery
- Methylprednisolone
- Surgical complication

Abbreviations and Acronyms
EMG: Electromyography
LDH: Lumbar disc herniation
MRI: Magnetic resonance imaging

Department of Neurosurgery, Kirsehir Ahi Evran University Faculty of Medicine, Kirsehir, Turkey

To whom correspondence should be addressed: Ziya Asan, M.D. [E-mail: ziyaasan@gmail.com]

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operating surgical levels were recorded. Operation notes were also examined, and perioperative complications were recorded.

Cases were evaluated in 2 different groups according to the surgical technique applied. The cases in Group 1 were recorded as those who underwent aggressive discectomy in which the intervertebral disc was excised together with the herniated disc material. The cases in Group 2 were recorded as those with sequestrectomy, in which only the excision of herniated-sequestered disc material was applied.

The duration of motor and sensory deficits and additional sensory changes were recorded. An increase in hypoesthesia has been noted as new-onset burning or stinging paresthesia. The deficits were recorded at intervals of 0–2 hours, 2–12 hours, 12–24 hours, and 24–48 hours.

The cases were evaluated in 2 groups: those treated with methylprednisolone (MP) in the early postoperative period and those who were not. MP treatment was given intravenously at a dose of 1 mg/kg, lower than the application dose for spinal cord injury. The treatment's effectiveness and the recovery process's differences were discussed by comparing the differences between the groups.

The cases evaluated in the study were those who were operated on under general anesthesia for a single-level lumbar disc herniation. All of the cases were operated with the standard microdiscectomy method, and the cases that were operated with different methods were excluded from the study. Cases that underwent additional surgical procedures other than microdiscectomy were not evaluated in the study.

Statistical Analyses

The cases were evaluated in 2 groups: those who underwent simple microdiscectomy and those who had an aggressive microdiscectomy. SPSS (IBM, Armonk, New York, USA) software was used for statistical analysis. An independent sample t test was used for statistical comparison between groups. P-value < 0.05 was accepted as significant.

Ethics Committee Approval

Ethics Committee Approval was obtained from Kırşehir Ahi Evran University Clinical Research Ethics Committee. Ethics committee number of the study: 2022-14/126.

RESULTS

Five hundred thirty-eight cases were identified who underwent surgery at L4-5 and L5-S1 levels. The number of early postoperative neuropraxia cases was 87 (14, 87%). Fifty of the cases were male, and 37 were female, and the mean age was calculated as 54.03 + 12.81. Fifty-one cases L4-5; 36 cases were operated on to diagnose L5-S1 disc herniation. The demographic data of the cases, the level at which they were operated on, the side, and the type of surgery performed are shown in Table 1.

The rate of iatrogenic neuropraxia in patients who underwent aggressive discectomy was higher than in patients who underwent only sequestrectomy (P: 0.009). Iatrogenic neuropraxia was diagnosed most frequently after L5-S1 microdiscectomy. The additional motor deficit was detected in 26 cases in the early postoperative period. Accompanying sensory neuropraxia was observed in all cases with the motor deficit. MP treatment at a loading dose of 250 mg 1 × 1 was applied to 57 cases.

Table 1. Demographic Distribution of the Cases According to the Type of Surgery Performed

	Agressive Discectomy (n = 362)	Simple Discectomy (n = 176)	Total (n = 538)
L4-5	37	14	51
L5-S1	24	12	36
Side	Left:34 Right: 27	Left:15 Right: 11	Left: 49 Right: 38
Ratio	16.85%	14.77%	

The motor deficit improved within the first 24 hours in all cases with or without MP therapy. There was no significant difference between the improvement of motor deficit in the groups given and not given MP ($P = 0.070$). It was observed that the sensory deficits improved more rapidly in the group with sensory deficit combined with a motor deficit and MP given. The degree of motor deficit was not detected as plegia in any case when it was first evaluated.

The finding of paresthesia in the form of burning was significantly more common in the group that underwent aggressive discectomy (P: 0.011). Tingling and stinging paresthesia was common in the simple discectomy group (P: 0.020).

Twenty-seven cases have electromyography (EMG) examination in the late period after the operation. EMG examinations were determined by the chronic period of radicular injury involvement

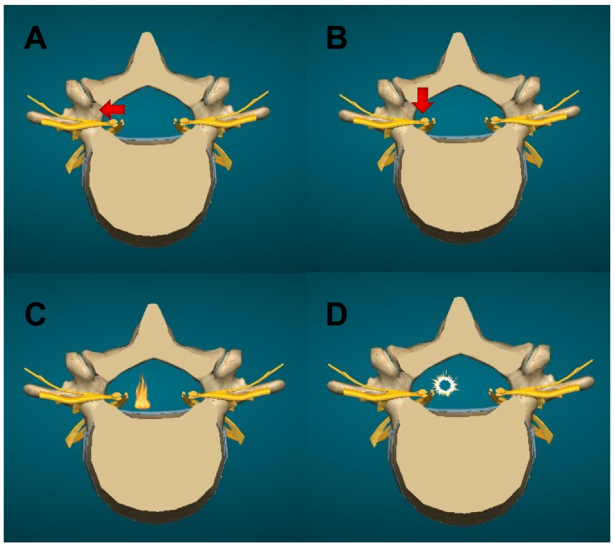

Figure 1. The radix's traction direction (*red arrow*) (**A**), compression on the radix (*red arrow*) (**B**), exposure to heat (**C**), and exposure to electrical damage due to bipolar coagulation (**D**) are schematized.

Table 2. Number of Neuropraxia Findings Encountered by Surgical Method

	Agressive Discectomy (n = 362)	Simple Discectomy (n = 176)	Total (n = 538)
Motor deficit	23 (27.38%)	3 (10.34%)	26 (23.01%)
Paresthesia			
Burning	32 (38.09%)	4 (13.79%)	36 (31.86%)
Tingle	24 (28.57%)	14 (48.28%)	38 (33.63%)
Stinging	5 (5.95%)	8 (27.59%)	13 (11.50%)
Total	84	29	113

at the operation level. These data have been accepted as far from supporting the existence of intraoperative iatrogenic neuropraxia.

DISCUSSION

An iatrogenic neuropraxia is a common occurrence after LDH surgery.[4-6] However, the rapid improvement of the clinical finding may cause it not to be considered as a diagnosis. Especially the faster recovery of the motor deficit makes the diagnosis difficult or causes it to be ignored.

Cases diagnosed with LDH are cases in which clinical findings occur due to disc compression or the traction effect of radicular involvement.[7-9] While radicular pain and sensory or motor deficits are familiar with these effects, paresthesia findings such as burning and stinging are not typical. The addition of these findings in the postoperative period is described as iatrogenic neuropraxia.

Many factors can increase radicular injury or impact during surgery.[1,2,10-12] The degree of radicular compression due to herniated disc, the presence of foraminal stenosis, the localization and size of the disc, excessive bleeding at the surgical site, the use of bipolar, the experience of the surgeon, the duration of the surgery are the reasons that may be associated with surgical complications and radicular injury.[3,6,11,13] The majority of these reasons are criteria that cannot be evaluated quantitatively. Manipulations on the radix often increase the radix's tension (Figure 1A) or compression (Figure 1B). However, a radicular injury may also occur due to the heat (Figure 1C) and electrical activity (Figure 1D) produced by bipolar coagulation.[14,15]

The cases were evaluated in 2 groups. The cases with disc herniation and intervertebral disc excision were evaluated in Group 1 as the cases that underwent aggressive discectomy. The cases in which only herniated disc was excised were evaluated in Group 2. In Group 1 cases, longer surgical time, more root compression and traction due to retraction, and more prolonged and frequent use of bipolar are expected. The rate of cases with iatrogenic neuropraxia was significantly higher in Group 1. In addition, it was observed that it increased motor and sensory deficits in Group 1 cases, and additional paresthesia findings in burning and stinging were observed more frequently. The number and rates of neuropraxia findings encountered by the surgical method are shown in Table 2.

Radicular injury during surgery may not occur only with root manipulations.[1,2,4,6,16] Bipolar coagulation for bleeding control can also cause neuropraxia with electrical and thermal effects.[14,15,17,18] While herniated disc material may cause root compression or traction in cases with LDH, the burning and stinging paresthesias encountered in the postoperative period are not due to radicular manipulations during surgery. A radicular injury may occur due to electrical activity and heat during bipolar coagulation.[14,15,17,18]

Although bipolar coagulation can be held responsible in iatrogenic neuropraxia cases in which burning, stinging paresthesia occurs; data such as how close the bipolar is to the radix during coagulation, at what degree and for how long are criteria that cannot be evaluated quantitatively. Bipolar coagulation may cause radicular injury with electrical activity and thermal effects, which may cause paresthesias in the form of burning and stinging.[14,17,18]

The total recovery time of motor deficit in cases with paresthesia and motor deficit was significantly longer than in cases with only motor deficit and motor deficit with hypoesthesia (Tables 3 and 4). It has been observed that neuropraxia, which is thought to occur due to bipolar, has a more prolonged course than neuropraxia due to traction and compression. In these cases, burning-stinging paresthesia continues after completely resolving the motor deficit.

Table 3. Duration of Neuropraxia Findings According to the Surgical Method (h = hours)

	Agressive Discectomy				Simple Discectomy			
	First 2 h.	First 12 h.	First 24 h.	First 48 h.	First 2 h.	First 12 h.	First 24 h.	First 48 h.
Motor deficit	10	10	3	0	3	0	0	0
Paresthesia								
Burning	2	3	17	12	0	0	3	1
Tingle	0	2	12	11	0	0	8	6
Stinging	0	3	2	0	6	2	0	0

Table 4. Distribution of Neuropraxia Times in the Group That Received and Did Not Receive Methylprednisolone Therapy (h = hours)

	Motor Deficit		Burning Paresthesia		Tingling Paresthesia		Stinging Paresthesia	
	First 24 h. Recovered	First 48 h. Recovered	First 24 h. Recovered	First 48 h. Recovered	First 24 h. Recovered	First 48 h. Recovered	First 24 h. Recovered	First 48 h. Recovered
MP treatment(applied) (n:57)	18 (100%)	0	15 (71.43%)	6 (28.57%)	17 (56.67%)	13 (43.33%)	6 (100%)	0
MP treatment (not applied) (n:30)	8 (100%)	0	8 (53.33%)	7 (46.67%)	4 (50.00%)	4 (50.00%)	7 (100%)	0

In these cases, EMG findings of the late postoperative period cannot confirm postoperative iatrogenic neuropraxia. Because there are also chronic radicular neuropraxia findings related to LDH in the operated area. Whether these data are due to iatrogenic neuropraxia cannot be definitively determined.

Limitations of the Study

It is possible to evaluate the degree of the motor deficit by scoring over 5 points. However, there is practically no quantitative criterion for the sensory deficit, and cases can qualitatively indicate the degree of hypoesthesia. Therefore, the increase in the sensory deficit was stated qualitatively according to the descriptions of the cases.

Neuropraxia is defined as a reversible injury to peripheral nerves. EMG examination is not valid in the early period of peripheral nerve injuries. For this reason, the sensitivity of EMG examination is low in evaluating postoperative neuropraxia in cases of LDH. In addition, chronic radicular injury findings may be present due to disc herniation in LDH cases. Postoperative iatrogenic neuropraxia cannot be justified by the coexistence of chronic radicular injury findings. For this reason, iatrogenic neuropraxia can be accepted as a diagnosis that can be confirmed in clinical findings, not laboratory tests. The neuropraxia defined in the study is a terminological term defined according to clinical findings, not laboratory findings.

CONCLUSIONS

Early radicular neuropraxia is a common condition after LDH surgery. Methylprednisolone treatment may help sensory paresthesias to disappear more quickly. Factors that can cause neuropraxia are often caused that cannot be measured quantitatively. Early neuropraxia is frequently encountered in patients who underwent aggressive discectomy with prolonged surgical time and excessive surgical manipulation.

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