Radiculopathy Contralateral to the Side of Disc Herniation —Microendoscopic Observation—

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Abstract:

Introduction: There are patients with lumbar disc herniation (LDH) having contralateral sciatic symptoms although the mechanisms of this clinical feature are still not well understood. The purpose of this study was to investigate these mechanisms by microendoscopic findings.

Methods: Patients were performed microendoscopic surgery using over-the-top approach (ME-OTT), with laminoplasty, extirpation of herniation, and observation of the contralateral nerve root. The over-the-top approach was applied through the same incision from the herniation side. Clinical results were assessed according to the clinical scoring system established by the Japanese Orthopedic Association (JOA) score.

Results: This study consisted of five patients, with the average age of 55.6 years old. The mean preoperative JOA score was 13 points. Three cases were Grade II and two were Grade III degrees of disc herniation. Levels of herniation were one at L3-4 and four at L4-5. Remission of sciatic symptoms was obtained in all cases after surgery. The average and percent improvements (%IP) of JOA scores at 2 months after surgery were 27.8 points and 92%, respectively. By the approach from the herniation side using ME-OTT, image around the contralateral nerve root was obtained without radical intervention. By ME-OTT, redness of the nerve root and fibrosis around the symptomatic nerve root were identified, whereas inflammatory changes were not apparent on the ipsilateral nerve root.

Conclusions: Operative treatment of LDH with contralateral symptoms by ME-OTT was a useful procedure for decompression and observation of the affected nerve root. Asymptomatic disc herniation, "silent disc herniation," was considered at the herniation side since there were less inflammatory changes around the ipsilateral nerve root. In contrast, compression of dura toward the opposite side by disc herniation could have led to mechanical stress against the contralateral nerve root and triggered inflammation at lateral recess, resulting in radicular pain.

Keywords:

lumbar disc herniation, contralateral symptom, microendoscopic surgery, friction radiculitis

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Introduction

The pathology of contralateral symptoms due to lumbar disc herniation (LDH) has been referred to in previous reports (Fig. 1)[1,2]. One of the possible mechanisms suggested was "friction radiculitis" caused by traction of the nerve root at the pedicle. Reports of inflammatory cytokines associated with radicular pain support the pathology of cytokine-mediated inflammatory pain at the lateral recess. In this study, we try to explain the mechanism of contralateral sciatic pain with relevance to previous reports and microendoscopic findings.

Materials and Methods

This was a retrospective chart review of patients who presented to our hospital from 2010 through 2014. A total of five patients with LDH were included in this study. Diagnoses were made through medical interview, clinical, and radiological examinations. Disc herniation was classified into four grades[3], according to the location of disc fragment: grade I-subligamentous, grade II-extrusion without migration, grade III-extrusion with cephalad or caudad migration, grade IV-sequestered. The lateral recess was assessed for any stenosis responsible for radiculopathy. The stenosis of

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the lateral recess was defined as <4 mm in the axial plane at the level of the pedicle[4]. Diagnoses were made by excluding other possibilities that may cause radiculopathy, such as foraminal stenosis, lateral lumbar herniation, and scoliosis. Operative treatment was considered for patients for whom it was difficult to obtain remission by conservative treatment.

The operation was performed by microendoscopic surgery using over-the-top approach (ME-OTT), with laminectomy of the herniation side, extirpation of herniation, and observation of the opposite-side nerve root (Fig. 2)[5].

Clinical results were assessed according to the JOA score: a clinical scoring system established by the JOA. The JOA score included assessments of lumbar pain, numbness or pain of lower limbs, walking ability, SLR test, esthesia, muscle strength, ADL, and bladder function. Results are shown as mean±2SD (standard deviation).

Results

Four men and one woman from 29 to 66 years of age (mean age: 55.6±27.1 years) were evaluated.

Four patients had L5 radiculopathy with contralateral disc herniation at the L4-5 intervertebral disc, and one patient had L4 radiculopathy with contralateral disc herniation at the L3-4 intervertebral disc.

The SLR test was positive on the limb of the symptomatic side, and selective root block performed on one of the five patients on the symptomatic side was effective to obtain pain reduction. On radiographic examinations, the stenosis of lateral recess was not seen on the symptomatic side (Fig. 3).

The JOA scores of the LDH ranged from 8 to 19 points with a mean score of 13±8.0 points. Three cases were Grade II, and two were Grade III degrees of disc herniation. Cases classified as Grade III had caudad disc migration[3]. The sciatic symptoms disappeared after surgery in all cases. The percent improvement (%IP) of the JOA scores at 2 months after surgery was from 90% to 95% (mean %IP: 92%±0.04; Table 1). Epidural fat tissue around the nerve roots of the herniated side remained in all cases. However, on the opposite side, epidural fat tissue had disappeared, and there were congested fibrous changes on the surface. Redness of the dura and affected nerve root around the lateral recess were observed (Fig. 4).

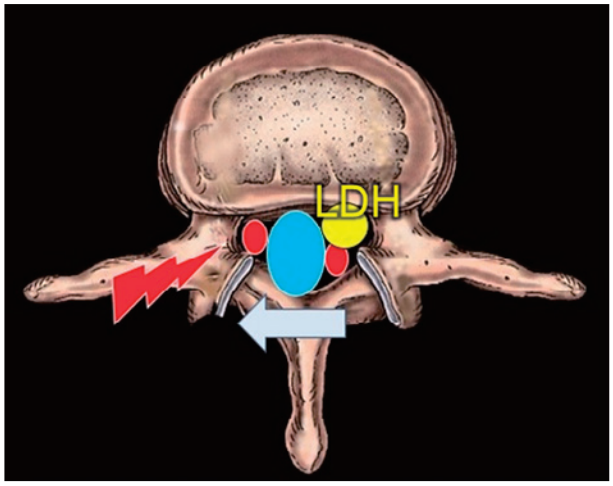

Figure 1. Lumbar disc herniation with contralateral symptoms.

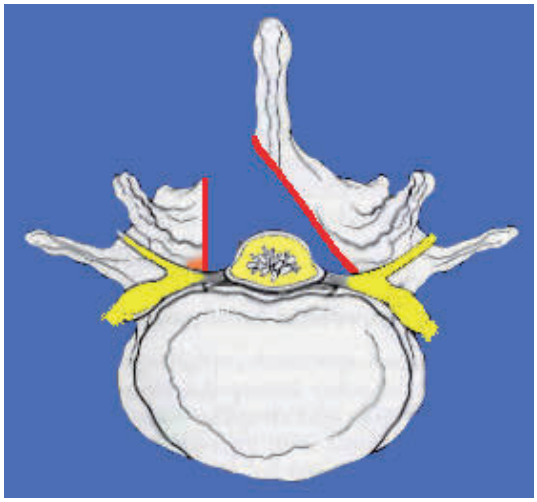

Figure 2. Over-the-top approach via the same incision[5].

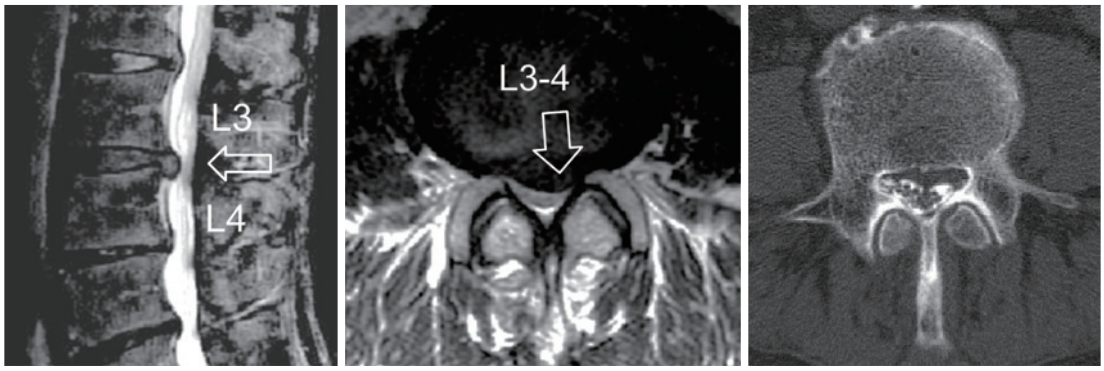

Figure 3. Preoperative MRI showing L3-4 lumbar disc herniation (indicated by arrows in A and B). Preoperative CTM showing L4 lateral recess and disc herniation (C).

Table 1. Classification of Patients, Herniation, and Results of Microendoscopic Surgery.

#	Age (years)	Gender	LDH			Symptoms Site	Affected NR Redness and DEF*	JOA score		
			Grade	Level	Site			Preop	Postop	%IP
1	61	M	III	L3-4	Lt	Rt	+	8	28	95
2	64	M	II	L4-5	Rt	Lt	+	19	28	90
3	66	F	III	L4-5	Rt	Lt	+	14	28	93
4	29	M	II	L4-5	Rt	Lt	+	15	28	92
5	58	M	II	L4-5	Lt	Rt	+	9	27	90
(Mean)								13	27.8	92

DEF*: disappearance of epidural fat tissue

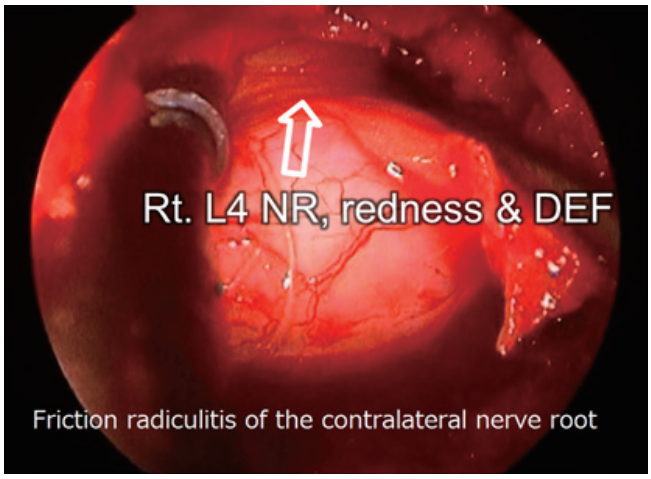

Figure 4. Microendoscopic findings: redness of dura and contralateral nerve root (the nerve root is indicated by arrow). DEF: disappearance of epidural fat tissue.

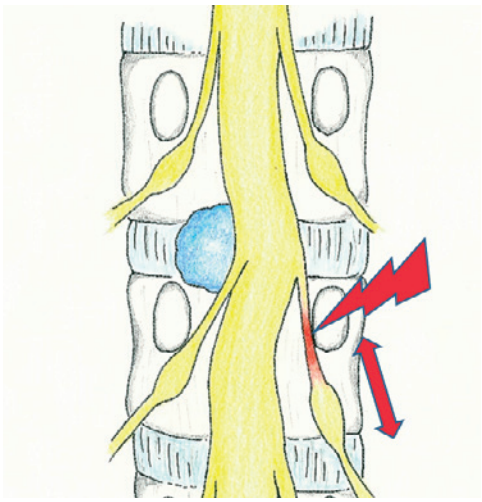

Figure 5. Displacement of dura by herniation toward the opposite side and traction of contralateral nerve root along the pedicle at lateral recess.

Discussion

Albert et al. have reported a case of LDH with contralateral sciatica treated by laminectomy of both sides. Their observations were that there was displacement of dura toward the contralateral side by disc herniation, and the symptomatic nerve root was stretched along the pedicle[6].

Smith et al. explained the effect of SLR on nerve root motion[7]. SLR induced both linear motion (0.5-5 mm) and strain (2%-4%) in spinal nerves L4, L5, and S1. The lumbar nerve root shifted toward the pedicle when the lower limb was raised by SLR.

With these studies taken into consideration, mechanical stress to the nerve root at the specific anatomical structure of lateral recess, where decompression is required for some cases with lumbar radiculopathy, seemed to be responsible for the generation of contralateral sciatica. The herniation could have compressed the dura to the contralateral side, and "friction radiculitis" of the nerve root was induced at lateral recess by motion of lower limbs (Fig. 5).

In general, when tissue cells are damaged by some cause, damage-associated molecular patterns (DAMPs) are released from the disrupted cells. Neutrophils detect these DAMPs released from damaged tissues and promote broad inflammatory reactions such as pro-inflammatory cytokine release. Inflammatory changes such as redness, swelling, and pain are mediated by neutrophils, and this reaction can occur without the intrusion of pathogens, resulting in sterile inflammation[8].

Allegri et al. described the main cause of radicular pain as inflammation of the affected nerve root rather than its compression. Although radiculopathy and radicular pain often accompany one another, radiculopathy has been observed in the absence of pain[9,10].

When considering what has been the very triggering factor of contralateral radiculopathy, the pathology of "friction radiculitis"; the impingement of the nerve root along the pedicle due to the mechanical traction force describes our cases as previously reported[11]. This pathology can possibly happen since there remains a motion capacity in the neve root as seen in the SLR[7].

Cooper et al. have reported disc-associated periradicular fibrosis and vascular abnormalities around the affected nerve root[12]. In their study, inflammatory cytokine, IL-1, and TypeI collagen mRNA were elevated around the peridiscal tissue. Other reports show that TNFα is highly associated

with the symptoms of lumbar radicular pain. The amount of pro-inflammatory cytokine TNFα correlates with the level of lumbar radicular pain[13,14]. In a clinical trial by Korhonen et al., intravenous infusion of infliximab, a monoclonal antibody of TNFα, was not recommended to use in treating radicular pain, although TNFα was mentioned as being involved in the pathophysiology of sciatica[15]. Where these reports are concerned, the contralateral sciatica could have been cytokine-mediated inflammation, and as a result, friction radiculitis were observed as inflammatory changes and sciatic symptom.

In our series, some patients did not show any symptoms on the ipsilateral side suggested the pathology of "silent disc herniation," and there were less inflammatory changes around the ipsilateral nerve root. The existence of lumbar canal stenosis or disc herniation could progress to produce sciatic symptoms over a long period, but nonetheless, it does not always accompany sciatic pain[16]. The nerve root could have been compressed by disc herniation but did not cause radicular pain of the ipsilateral side.

The indication for ME-OTT is structural spinal canal stenosis with predominant leg symptoms. In our cases, ME-OTT was indicated to decompress the shifted nerve root by removal of herniation with avoidance of radical intervention to the opposite side. Our clinical results may show that extirpation of herniation from the ipsilateral side is sufficient for pain reduction.

Recently, the advancement of radiographic diagnosing has been progressing dramatically. The diffusion-weighted image (DWI) of MRI is able to capture edematous changes in brain and peripheral nerves[17]. The apparent diffusion coefficient could be an indicator when detecting damage of the nerve root, the amount of which increases at the lesion of angiogenic edema[18]. Fractional anisotropy (FA) of DWI allows us to visualize and track bundles of nerve fibers. The low amount of FA reflects the decrease in density or diameter of nerve fibers due to mechanical compression[19,20].

Our study shows that the contralateral sciatic pain due to LDH could have been induced by displacement of dura toward the contralateral side of herniation, which led to traction stress against the contralateral nerve root. As a result, radicular pain was induced by inflammatory reaction around the contralateral nerve root. Inflammatory changes of redness and fibrosis were able to observe microendosopically by the procedure of ME-OTT, and the treatment resulted in a good outcome of pain relief.

Limitations of this study were that assessment of histological changes around the nerve root and quantitative analysis comparing preoperative and postoperative levels of inflammatory cytokine were not conducted. The pathophysiology of LDH having contralateral symptom remains to be a consideration for future study.

Conflicts of Interest: The authors declare that there are no relevant conflicts of interest.

Author Contributions: Norito Hayashi wrote and prepared the manuscript, and all of the authors participated in the study design. All authors have read, reviewed, and approved the article.

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