

Lumbar Radiculopathy Due to Unilateral Facet Hypertrophy Following Lumbar Disc Hernia Operation: A Case Report

Lomber Disk Hernisi Operasyonu Sonrası Gelişen Ünilateral Faset Hipertrofisine Bağlı Lomber Radikülopati: Bir Olgu Sunumu

ABSTRACT

OBJECTIVE: To present a radiculopathy case due to unilateral facet hypertrophy developing three years after a lumbar disc hernia operation.

CASE REPORT: A fifty two-year-old female patient, who had been operated on for a left L5-S1 herniated lumbar disc three years ago, was hospitalized and re-operated with a diagnosis of unilateral facet hypertrophy. She had complaints of left leg pain and walking restrictions for the last six months. Left Straight Leg Raising test was positive at 40°, left ankle dorsiflexion muscle strength was 4/5, left Extensor Hallucis Longus muscle strength was 3/5, and left Achilles reflex was hypoactive. Lumbar spinal Magnetic Resonance Imaging revealed left L5-S1 facet hypertrophy.

RESULTS and CONCLUSIONS: Lumbar radiculopathy due to lumbar facet hypertrophy is a well-known neurological condition. Radicular pain develops during the late postoperative period following lumbar disc hernia operations that are often related to recurrent disc herniation or to formation of post-operative scar tissue. In addition, it can be speculated that unilateral facet hypertrophy, which may develop after a disc hernia operation, might also be one of the causes of radiculopathy.

KEY WORDS: Complication, Lumbar disc hernia, Radiculopathy, Surgery, Treatment, Unilateral facet hypertrophy

ÖZ

AMAÇ: Lomber disk hernisi ameliyatı sonrası gelişen ünilateral faset hipertrofisine bağlı bir radikülopati olgusunu sunmak ve bu durumun da ameliyat sonrası geç dönem radikülopati sebeplerinden biri olabileceğini vurgulamak.

OLGU SUNUMU: Elli iki yaşında, üç yıl önce sol L5-S1 disk herniasyonu nedeniyle ameliyat olan bir kadın hasta son altı ayda sol bacak ağrısı ve uzun süreli yürüyememe şikâyeti ile başvurdu. Nörolojik muayenesinde sol düz bacak germe testi 40° de pozitif, sol ayak dorsifleksiyonu 4/5 kas gücünde, sol extansor hallucis longus 3/5 kas gücünde ve sol Achille refleksi hipoaktif idi. Yapılan Lomber MRG'de sol L5-S1 faset eklem hipertrofisi ve lateral resesde obliterasyon saptanan hasta bu bulgularla yeniden ameliyata alındı.

SONUÇLAR ve YORUM: Lomber faset hipertrofisine bağlı lomber radikülopati bilinen bir nörolojik tablodur. Lomber disk hernisi ameliyatlarından sonra geç dönemde ortaya çıkan radiküler ağrılar sıklıkla nüks disk herniasyonu ya da post-operatif gelişen skatris dokusu oluşumuna bağlıdır. Bununla birlikte, disk hernisi ameliyatından sonra gelişebilecek tek taraflı faset hipertrofisinin de radikülopatiyeye yol açabileceği düşünülmelidir.

ANAHTAR SÖZCÜKLER: Cerrahi, Komplikasyon, Lomber disk hernisi, Radikülopati, tedavi, Ünilateral faset hipertrofisi

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INTRODUCTION

Lumbar facet hypertrophy is known to lead to radiculopathy via compressing the nerve root and has been investigated by many authors (1, 2, 5, 6). The hypertrophic changes in posterior spinal elements compress the neural structures, especially in the lumbar and cervical regions (1). However, unilateral facet hypertrophy and an associated root lesion are very rare conditions. In this report, we present a patient who had been operated for left L5-S1 herniated lumbar disc three years ago and was re-operated for radiculopathy due to unilateral facet hypertrophy and emphasize that unilateral facet hypertrophy should be kept in mind as a cause of late postoperative radiculopathy.

CASE REPORT

A 52-year-old female patient was admitted to the hospital with low back pain, left leg pain and walking restrictions since the last 6 months. She had been operated for the left L5-S1 disc hernia three years ago (Figure 1A,B). At this previous operation a simple discectomy was carried out without any extra bone removal. The patient affirmed that the operation improved her symptoms. Three years after the operation, her low back pain restarted, intensified in the hip and extended to the lateral side of her thigh. The leg pain increased with walking, thus restricting her walking, and had become more intense in the last 3-4 months. Her walking distance had decreased to 200 meters without pain.

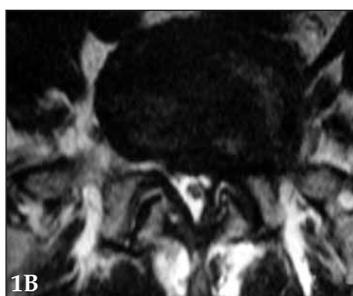


Figure 1A,B: Lumbar spinal MRI revealed L5-S1 grade I listhesis and disc hernia on the sagittal T1-W sequence of MRI. Broad-based herniation and mild left facet joint hypertrophy at the same level were observed on the axial slice of MRI when she underwent the first operation.

The neurological examination results were as follow: left Laseque test was positive at 40°, left dorsal flexion muscle strength was 4/5, Extensor Hallucis Longus (EHL) muscle strength was 3/5, and left Achilles reflex was hypoactive. No sensory deficit was detected. Left L5-S1 facet hypertrophy and obliteration of lateral recess were seen as well as compression of the S1 nerve root on sagittal and axial MRI images (Figure 2A,B). The patient underwent surgery with these findings.

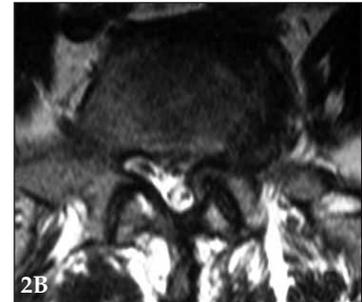


Figure 2A,B: Lumbar spinal MRI reveals left L5-S1 facet hypertrophy compressing the left S1 root before the second operation for radiculopathy.

During the operation, the L5 inferior facet was observed to be extremely hypertrophic and calcified. It was compressing the S1 root, which was decompressed through a partial facetectomy and foraminotomy. The medial side of the facet joint was removed. The medial facet bone was extremely calcified and hard to remove. A wide chisel was used for removal. Bone removal was extended to the upper root (L5) exit zone. Complete decompression of the S1 root was achieved. A careful and wide-ranging foraminotomy of the S1 root was then carried out. Calcification of the foramen was not as intense as the facet.

Histopathological investigation of the removed tissues revealed normal bone and cartilage tissue with no pathological structure. There was no histopathological calcification of the bone.

The patient's symptoms ceased in the early postoperative period; and the neurological examination results were within normal limits at the third postoperative month.

DISCUSSION

The etiology of unilateral facet hypertrophy has not yet been fully understood. In 1967, Farfan and Sullivan pointed to the relation of abnormality in the disc and posterior articular changes. They reported asymmetry in the facet joint at the lesion level in patients with acute disc protrusion. Furthermore, they suggested that such asymmetric structure caused abnormal stress on the disc, which resulted in weakening. Concerning development of unilateral facet joint hypertrophy, Epstein et al. underlined the abnormal load-bearing factor regarding these joints (6).

Reynolds et al. (5) have reported 3 years of experience with 22 patients (14 males, 8 females; 19-75 years old) who underwent surgery for lumbar monoradiculopathy due to unilateral facet hypertrophy. They suggested that trauma might have a role in the onset of symptoms. They also reported that the nerve root could be compressed by a hypertrophic superior or inferior facet.

Wilde et al. (6) presented four cases of unilateral facet hypertrophy relating lower back pain as well as radicular symptoms. They stated that degenerative changes of intervertebral disc can cause an abnormal burden in the moving segment accompanied by unilateral facet joint osteoarthritis.

Kirby and Maimaris (2) repeated that the pathogenesis for unilateral facet hypertrophy had not been fully understood, but a former trauma could be an initiating factor. They reported a patient who had a whiplash injury in the cervical vertebra due to an intravehicular traffic accident and gradually developed unilateral facet hypertrophy (left C4-5) with radiculopathy at the same level. They suggest that unilateral facet joint hypertrophy in the spine could be due to post-traumatic myositis ossificans. They claimed that whiplash injuries of the cervical spine causing musculo-ligamentous sprains of facet joints with periosteal tearing are becoming increasingly common when drivers or passengers are wearing a seat belt and either decelerate suddenly or are shunted from the rear.

Kornberg (3) presented a case with a unilateral defect of the pars interarticularis and contralateral facet joint degeneration and hypertrophy with first degree spondylolisthesis of the L5-S1 level. The facet degeneration noted might represent chronic increase

of interfacet forces that predispose towards premature degenerative changes, as described with facet joint tropism.

Deogaonkar et al. (1) reported two cases with thoracic cord compression due to unilateral lamina and facet hypertrophy. They emphasized that symptomatic compression due to hypertrophy of posterior spinal elements was more usual in lumbar and cervical sites than the thoracic area. The authors also stated that the etiopathogenesis of unilateral hypertrophy of vertebral posterior elements could not be detected.

Our patient, who presented with low back pain, sciatalgia on the left leg and neurogenic claudication, developing after a lumbar disc hernia operation, was diagnosed as unilateral facet hypertrophy; and complete recovery was obtained after a decompression operation.

The reason for the unilateral facet hypertrophy occurring on the same side and at the same level (left L5-S1) almost 3 years after the surgical operation is probably the same factor that caused abnormal loading of the facet joint on the same side during the postoperative period. Disc removal may change the load-bearing pattern at the same level and side that already experienced abnormal loading. It should be taken into account that ossification and/or hematoma may develop in the facet due to a potential ligamentous and periosteal injury during the operation, which may then be calcified, and become an effective factor in the etiology of sciatalgia. Nishida et al. (4) presented a spinal epidural hematoma case with right L4-5 facet joint in a 58-year old female patient. The authors stated that hemorrhage and spontaneous epidural hematoma was mostly from the posterior internal vertebral venous plexus (Batson plexus). It is also reported that coagulation disorders or minor trauma might be another reason for such hemorrhages. Wiltse et al. suggested that disc herniation alone could result in hemorrhage from Batson plexus. In the radiculopathy case that followed a facet joint apoplexy without a preceding synovial cyst formation, as reported by Nishida et al, the etiology for hemorrhage in the lumbar facet joint was unknown. However, it was suggested that the number and volume of veins increased in degenerative synovium might cause bleeding following a minor trauma (4).

CONCLUSION

Radiculopathies in the late postoperative period can generally be explained by recurrent herniation of the nucleus pulposus and scar tissue that cause nerve root compression. Unilateral facet hypertrophy developing after hemilaminectomy might be considered another reason for this well-known pathology even though it is seen rarely.

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