Clinical Study

Differential diagnosis of intraspinal and extraspinal non-discogenic sciatica

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Abstract

The aim of this study is to present a series of 11 patients with non-discogenic sciatica (NDS), and to review the diagnostic techniques of careful clinical and radiological examination. The cases include lumbar radicular herpes zoster, lumbar nerve root schwannoma, lumbar instability, facet hypertrophy, ankylosing spondylitis, sacroiliitis, sciatic neuritis, piriformis syndrome, intrapelvic mass and coxarthrosis. The pain pattern and accompanying symptoms were the major factors suggesting a non-discogenic etiology. Pelvic MRI and CT scans, and sciatic nerve magnetic resonance neurography were the main diagnostic tools for diagnosis of NDS. The treatment of choice depended on the primary diagnosis. Detailed physical examinations with special attention paid to the extraspinal causes of sciatica and to pain characteristics are the major components of differential diagnosis of NDS.

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1. Introduction

Sciatica is common, and is frequently caused by lumbar disk herniation.1 However, some intraspinal or extraspinal pathologic processes along the sciatic nerve may also cause sciatica. Whereas lumbar spine imaging reveals the causes of intraspinal non-discogenic sciatica (NDS), extraspinal sciatica is often misdiagnosed because routine diagnostic tests focus on the lumbar spine.2 Extrapelvic causes affect the nerve as it progresses distally from the sciatic notch. A careful patient history and clinical examination are important in identifying extraspinal sciatica. Further diagnostic imaging may clarify the diagnosis.

In general, studies of NDS tend to focus on only one disorder.1–7 This study reports a series of 11 patients with NDS, and overviews the different causes of intraspinal and extraspinal sciatica.

2. Patients

The patients, 6 female and 5 male, were aged between 25 and 65 years old. There were 4 patients with extraspinal NDS, 4 with intraspinal NDS, and 3 with sciatica secondary to both spinal and extraspinal processes.

2.1. Intraspinal non-discogenic sciatica

2.1.1. Lumbar radicular herpes zoster (Patient 1)

A 64-year-old woman was admitted with a 1-week history of left leg pain that was not responding to analgesics. A neurological examination revealed no abnormal findings. A lumbar MRI showed degenerative changes (Fig. 1a). A physical examination revealed typical skin lesions along the L3 and L4 dermatomes (Fig. 1b). The patient was referred to the dermatology department, where the lesions
were determined to be caused by herpes zoster. After receiving medication for herpes zoster, the patient’s symptoms resolved.

2.1.2. Schwannomatosis (Patient 2)

A 27-year-old female patient was admitted with a 2-month history of low back pain (LBP) and left leg pain. Physical examination revealed a positive straight leg raising (SLR) test at 45°, and hypoesthesia at the left S1 dermatome. Lumbar MRI showed multiple schwannomatosis of the bilateral L5 and left S1 nerve roots (Fig. 2). The symptoms improved after medical therapy. Because of the lack of neurological deficit and the small size of the schwannomas, surgical treatment was not indicated.

2.1.3. Facet syndrome and lumbar disk herniation (Patient 3)

A 63-year-old woman was admitted with a history of LBP, left leg pain and numbness. The SLR test was negative. The patient had pain with extension of the trunk. There was no neurological deficit. A lumbosacral anteroposterior (AP) radiograph showed sclerotic and degenerative changes in the L5–S1 facet joint on the left side. A lumbar MRI showed a L4–L5 and L5–S1 central disc protrusion. After physical therapy and flexion exercises, the symptoms resolved partially.

2.1.4. Lumbar instability (Patient 4)

A 31-year-old male was admitted with a 12-year history of LBP and a 1-month history of left leg pain. Physical examination showed a positive SLR test at 60°. The patient was mistakenly diagnosed as having a lumbar disc hernia (LDH) at another center and was treated with physiotherapy, which was not beneficial. Sagittal lumbar MRI showed no disc herniation, and axial MRI showed pars defects of L5. Oblique radiographs showed bilateral pars interarticularis defects. The LBP was attributed to the lumbar instability related to the isthmic defects.
2.2. Extraspinal cases

2.2.1. Sciatic neuritis (Patient 5)
A 64-year-old woman was admitted to hospital with left buttock pain. The patient had undergone surgery for lumbar spondylolisthesis 10 years previously. The results of a physical examination were unremarkable. A lumbar MRI showed left sciatic neuritis between the piriformis muscle and the proximal femur (Fig. 3).

2.2.2. Bilateral sacroiliitis (Patient 6)
A 25-year-old female was admitted with a 2-month history of LBP, left-side buttock and posterior thigh pain. The patient had no lower limb weakness or paresthesias and had no history of morning stiffness or night pain. On physical examination the SLR test was limited to 60° on the left side. There was no other neurologic deficit in the lower limbs. Her range of motion of the trunk was not limited and was not painful. Deep palpation of the left buttock over the sciatic nerve course was painful. Gaenslen’s test was positive on the left side. The sacroiliac compression test was positive on the left side. MRI of the lumbar spine and sacroiliac joint showed L4–L5 disc protrusion with no nerve root compression and bilateral sacroiliitis. The sciatica was attributed to the sacroiliitis. The patient received indomethacin at 150 mg/day for 15 days. The patient’s symptoms resolved. The visual analogue scale (VAS) pain score decreased from 7 to 2. Further analyses were performed to identify the etiology of the sacroiliitis and the patient was diagnosed with seronegative spondyloarthritis.

2.2.3. Sacroiliitis (Patient 7)
A 53-year-old woman was admitted with left leg pain. Physical and neurological examinations yielded no abnormal findings. Lumbar MRI showed no abnormality. Sacroiliac MRI showed sacroiliitis and edema in the joint compressing the sciatic nerve (Fig. 4). The erythrocyte sedimentation rate (ESR) was 88 mm/h. After further examination, the patient was diagnosed with seronegative spondyloarthritis. The patient started to receive indomethacin at 300 mg/day, salazosulphapyridine at 400 mg/day and prednisolone acetate at 7.5 mg/day. Pain decreased and the ESR was 68 mm/h after 2 weeks. Symptoms completely resolved after completion of the medical treatment.

2.2.4. Soft tissue tumor (Patient 8)
A 55-year-old male was admitted with a 1-month history of right buttock pain. The patient had undergone L4–L5 discectomies five and three years previously. Neurological examination revealed no deficit. The buttock was painful upon palpation. Lumbar spine MRI showed modic changes at the L4–L5 level. A pelvic MRI showed a 6 cm soft tissue mass anterior and right to the sacrum. Examination of a pelvic CT scan revealed destruction of the anterior surface of the sacrum (Fig. 5). A CT-guided needle biopsy revealed an angiosarcoma.

2.2.5. Piriformis syndrome and hamstring tendinopathy (Patient 9)
A 59-year-old woman was admitted with a 2-month history of left-side buttock and posterior thigh pain. The patient had an antalgic gait with knee flexed and hip adducted and was not able to walk further than 10 m

Fig. 3. Axial fat saturated T2-weighted lumbar MRI showing hyperintensity at the left sciatic nerve.

Fig. 4. (A) Coronal STIR-weighted and (B) axial postcontrast fat-saturated T1-weighted sacroiliac joint MRIs showing inflammation on the left side.
because of increasing symptom intensity. At physical examination the SLR test was negative. Freiberg’s sign was positive. The range of trunk motion was not limited. The left leg was paresthetic. Tenderness in the piriformis muscle and ischium pubis was noted on deep palpation. MRI of the lumbar spine yielded no abnormal findings. Magnetic resonance neurography (MRN) revealed piriformis asymmetry on the left side, high signal at the sciatic nerve at this location and bone marrow edema at the ischium pubis at the hamstring muscle insertion site (Fig. 6). A physical therapy protocol for piriformis syndrome (PS) was applied. At the end of the physical therapy, the VAS score decreased from 10 to 3 points. The antalgic gate was corrected and Freiberg’s sign became negative. Tenderness of the ischium pubis remained due to hamstring tendinopathy.

2.2.6. Lumbar disc hernia and piriformis syndrome (Patient 10)

A 48-year-old male was admitted with a 6-week history of LBP and left leg pain. MRI of the lumbar spine showed L5–S1 disc herniation compressing the left S1 nerve root. Electromyography showed S1 radiculopathy. He underwent L5 hemipartial laminectomy, and left L5–S1 microdiscectomy. After surgery, the symptoms resolved partially but the patient still complained of left thigh pain after prolonged sitting and hip internal rotation. MRI neurography showed no abnormal signal at the sciatic nerve. The patient underwent a tetracosactrin injection protocol but received no benefit. A new examination revealed negative SLR. Freiberg’s sign was positive at the left side. Although MRN showed no abnormality, given the results of a physical examination, the patient was diagnosed as having PS and the physical therapy protocol for PS was applied. The patient received 15 sessions of physical therapy. At the end of the physical therapy regimen, the VAS score decreased from 9 to 2 points. The patient was comfortable after prolonged sitting and internal rotation of the hip.

2.2.7. Degenerative lumbar spine and coxarthrosis (Patient 11)

A 65-year-old male was admitted with bilateral buttock pain and neurogenic claudication. The FABER test was also positive at the left side. A lumbar MRI showed L3–L4 disc herniation, lumbar spinal stenosis, and L4–L5 spondylolisthesis. Pelvic radiographs showed grade IV osteoarthritis of the left hip. After discussion with the patient regarding a treatment plan, the patient underwent L3–4–5 decompression and instrumentation, and intertransverse fusion. Two months after spinal intervention the patient underwent total hip arthroplasty. The symptoms resolved after surgery.

3. Discussion

Many intraspinal and/or extraspinal pathologic processes along the lumbar nerve roots and sciatic nerve can cause sciatica. In 20% of cases, the sciatica is of both discogenic and non-discogenic origin. However, in practice, causes of NDS are often overlooked, partly due to the high sensitivity of lumbar spine MRI in asymptomatic patients.

The causes of NDS may be classified into two major categories: intraspinal and extraspinal. Differential diagnosis
requires a careful and detailed physical and radiological examination based on a schema (Fig. 7).

3.1. Intraspinal causes of sciatica

Many intraspinal disorders can cause sciatica. There may also be multiple processes, for instance intradural or extradural cysts and tumors (mainly schwannomas), adult tethered cord syndrome, spinal epidural abscesses and hematomas, facet syndrome, lumbosacral deformities and instabilities. The pain pattern, and the presence or absence of accompanying symptoms, are the most important components of the differential diagnosis.

The tumor-related pains commonly cause patients to wake in the night. Whereas small schwannomas lead to radicular pain, larger tumors lead to accompanying symptoms due to multiple nerve root and spinal cord compressions. Epidural abscesses and hematomas may present with symptoms similar to intradural tumors. Adult tethered cord syndrome may lead to sciatica associated with a stretched thigh, resulting in gait disturbance. The pain of facet origin is located commonly in the low back, buttock and thigh. It rarely extends to the lower levels. The pain secondary to deformities and instabilities is commonly associated with LBP and increases in spine loading.

Fortunately, because lumbar MRI shows anatomical details of the lumbar spine, and other neural structures and soft tissues, making a diagnosis of spinal NDS is relatively easy. Coincidental spinal and extraspinal disorders, however, may complicate the symptomatology, and require careful evaluation.

3.2. Extraspinal causes of sciatica

Detection of extraspinal causes of sciatica is much more difficult than detection of intraspinal ones, and requires a high degree of caution. The main causes of extraspinal sciatica include sacroilitis,5,15 PS, intrapelvic processes,1,4 and hip arthrosis.17,18

3.3. Sacroilitis and seronegative spondyloarthropathies

Sacroilitis, as one of the major causes of sciatica,5,15 should be considered during the differential diagnosis of LDHs, particularly when there is posterior thigh pain.

There are two potential mechanisms by which sacroilitis can generate sciatica: (i) referred pain (e.g. patient 6), and (ii) direct involvement of the nerve by inflammatory mediators released from the sacroiliac joint (e.g. patient 7).6

Sacroilitis is a common feature of seronegative spondyloarthropathies; the others include spondylitis, morning stiffness, LBP, decreased mobility of the lumbar spine, and peripheral arthritis.

The history of the patient and characteristics of the pain allow diagnosis of sacroilitis and its etiology. The pain in sacroilitis has an insidious onset, is commonly localized in the deep gluteal region and may refer to the posterior thigh. The pain decreases with activity and increases in the late evening.
The presence of LBP in patients with sacroiliitis makes differential diagnosis complex. This is because LBP may indicate both ankylosing spondylitis and degenerative lumbar spine. The patient needs to be questioned carefully to determine the symptoms that indicate the disease. Disc herniation-related LBP increases after activity and decreases after rest, whereas spondylitis-related LBP decreases after activity, and increases during the latter half of the night. Morning stiffness is another characteristic of spondylitis-related LBP.19

Physical examinations should include a Sacroiliac Compression Test, Gaenslen’s test and a FABER test. The location of the aggravating pain after the FABER test may reflect sacroiliitis, whereas pain in the groin may reflect coxarthrosis.19

The early clinical diagnosis of sacroiliitis may be difficult and the diagnosis should be proven radiologically. Both CT and MRI are sensitive methods of demonstrating sacroiliitis. The early CT scan findings include cortical erosions and subchondral sclerosis of the sacroiliac joints. The later CT scan findings include sacroiliac joint narrowing and ankylosis.20 MRI has a similar capacity to CT for detecting the disease; however, MRI can detect the early stages of sacroiliitis because it can reveal bone marrow edema before morphologic changes can be determined by a CT scan.21,22

3.4. Intrapelvic compressive processes

Intrapelvic compressive processes may affect the nerve as it passes from the neural foramina to the greater sciatic notch. Reported intrapelvic compressing processes include tumors,3,23 hematoma,24 endometriosis,25 tubo-ovarian abscess,26 presacral abscess,27 and aneurysms.28

Pelvic and femoral bone tumors may compress the sciatic nerve. Bickel et al. analyzed a surgical database of 32 patients with bone tumors that caused sciatica. According to their analysis, the characteristics of the patient’s pain are very important in the differential diagnosis of bone tumors. Sciatica due to bone tumors generally has an insidious onset, causes constant pain, awakening at night, and is progressive and unresponsive to position changes.1 Local compression may reveal local tenderness. As most bone tumors occur in the pelvis or proximal femur, initial pelvic radiography is recommended for patients with atypical sciatica. A simple pelvic radiograph may show tumor-related bony destruction, as in patient 8. Three-phase bone scintigraphy, CT scanning, and MRI seem to be sensitive in detecting bone tumors. MRI and MRN may show the relationship between the tumor and the sciatic nerve in detail.

3.5. Piriformis syndrome

Sciatica is caused by PS in 6% of patients.29 Hypertrophy, inflammation, anatomic variations, myositis ossificans and traumatic injury of the piriformis muscle may compress the sciatic nerve.16,30 Any increase in the piriformis muscle tone not only produces local muscle pain but also may result in sciatica.

Patients with PS exhibit significantly different symptoms and results of physical examination to those of patients with discogenic sciatica.31 Pain is the predominant symptom in both, but there are some differences in pain patterns. There is usually sciatic notch tenderness or pain at the buttock in both patient groups. The pain of discogenic origin refers to the buttock and posterior thigh without any specific focal tenderness in the buttock. Unlike patients with LDH, patients with PS typically experience symptoms in multiple dermatomes rather than either the lateral (S1 radiculopathy) or the medial (L5 radiculopathy) dermatome alone. Numbness or weakness is quite uncommon in PS. Unlike in LDH, the SLR test is generally negative in patients with PS. Most patients with PS report that prolonged sitting and walking exacerbate their pain and that their symptoms reagggravate after internal rotation of the hip.

The differential diagnosis of PS requires special tests including Pace’s sign, Freiberg’s sign and deep digital palpation of the piriformis muscle. If some of these tests are positive, advanced radiological tests such as pelvic MRI and MRN should be performed since the pelvic MRI alone may fail to show the extraspinal parts of the sciatic nerve. MRN may identify the sciatic nerve and its relationship with the surrounding structures.

In one study, MRN had 93% specificity and 64% sensitivity for distinguishing patients with PS from those with similar symptoms by presenting piriformis muscle asymmetry and sciatic nerve hyperintensity at the sciatic notch.27 Although piriformis muscle hypertrophy has been demonstrated by MRI32 Filler et al. also observed ipsilateral muscle atrophy in some patients.31 This finding may be secondary to disuse of the muscle in the chronic stage. In the present study, MRN of patient 9 showed piriformis muscle atrophy and asymmetry, and sciatic nerve hyperintensity. However, the MRI of patient 11 (who had PS), similar to Lee et al.33 showed that the piriformis muscle was not compressing the sciatic nerve. The absence of the positive finding in the neutral (static) position can be explained by the dynamic nature of the piriformis muscle: as the pain is positive in Freiberg’s position, MRI and MRN should be performed in both static and dynamic positions (i.e. Freiberg’s position).

3.6. Hip disorders

The pattern of pain distribution in hip disorders may create difficulty in determining the source of pain (spine vs. hip).17 Swezy et al. reviewed patients who were treated for lumbar spinal stenosis while the primary source of the pain was osteoarthrosis of the hip, and patients who were treated for osteoarthrosis of the hip while the primary source of pain was lumbar spinal stenosis. Symptoms for both disorders are frequently present in the same patients.15 The presence of limb or groin pain, and limited
internal rotation of the hips is predictive of a hip disorder rather than a spine disorder. The occurrence of groin pain during the FABER test may suggest a hip disorder. Pelvic radiography may show hip osteoarthritis.

However, both hip and lumbar spine pathologies may exist together, as in patient 12. The radicular pain associated with progressive neurogenic claudication and neurologic deficit may dictate a lumbar decompression procedure before a hip procedure.

LDH is not the only cause of sciatica. A detailed patient history, especially focused on pain characteristics, is an important component of patient evaluation. Physical examination of patients with sciatica should include inspection, palpation, and all physical tests to exclude NDS.

References