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Common diseases mimicking lumbar disc herniation and their treatment

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ABSTRACT

Lumbar disc herniation (LDH) is a common disease characterized by leg pain, numbness, and low back pain, which are also encountered in peripheral nerve and paralumbar spine disease. This study describes other diseases with symptoms similar to LDH. Patients with paralumbar spine diseases such as superior cluneal nerve entrapment neuropathy (NEN), gluteus medius muscle pain, piriformis syndrome, and sacroiliac joint pain experience lowback, buttock, and leg pain. Peripheral nerve diseases of the leg including lateral femoral cutaneous NEN, common and superficial peroneal NEN, and tarsal tunnel syndrome also cause leg symptoms. These diseases can produce intermittent claudication, thought to be specific to lumbar spine disease, and can be misdiagnosed as LDH. They are rather common and can be treated less invasively. As a misdiagnosis may result in failed back-surgery syndrome, it is important to differentiate between LDH and the diseases described here.

INTRODUCTION

Lumbar disc herniation (LDH) is common; its symptoms of leg pain, numbness, and lowback pain (LBP), are also reported by patients with peripheral nerve and paralumbar spine diseases. Since they can cause intermittent claudication, thought to be specific to lumbar spine disease, differential diagnosis can be difficult. As misdiagnosis can result in failed back surgery syndrome (FBSS), spinal surgeons must be able to differentiate them from LDH.

PARA-LUMBAR SPINE DISEASES

Superior cluneal nerve entrapment neuropathy

Definition and symptoms

The superior cluneal nerve (SCN) is a sensory nerve that originates from the lower thoracic and lumbar posterior nerve. It is comprised of 4 to 6 nerves, runs around the paraspinal muscle, penetrates the thoracolumbar fascia near the iliac crest, and ends at the buttock [Figure 1]. The clinical features and etiology

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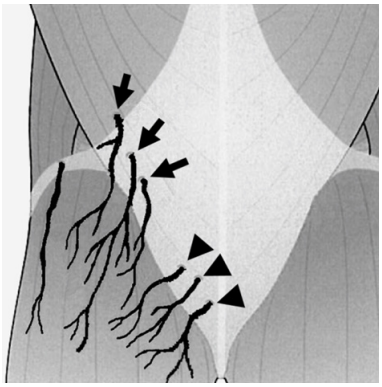

Figure 1: The superior cluneal nerve (arrows) consists of 4-6 nerves; it runs around the paraspinal muscle and penetrates the thoraco-lumbar fascia near the iliac crest before it arrives at the buttock. The middle cluneal nerve is identified by arrowheads

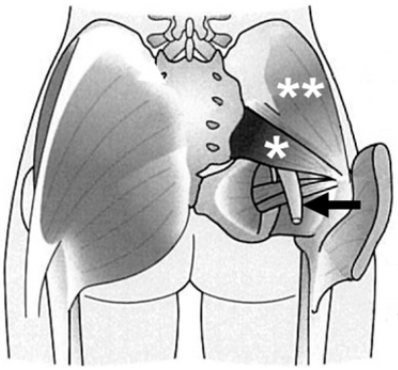

Figure 2: The location of the gluteus medius muscle (GMeM) (**) and piriformis muscle (*). The GMeM (**) is located in the buttock over the gluteus minimus and partially under the gluteus maximus muscle; it is covered by a tight gluteal aponeurosis. The piriformis muscle (*) connects the sacrum and greater trochanter. Loading of this muscle results in buttock pain and affects the adjacent sciatic nerve (arrow)

of SCN-entrapment neuropathy (EN) remain poorly understood. LBP occurs when the SCN is entrapped where it penetrates the thoraco-lumbar fascia. LBP attributable to SCN-EN involves the iliac crest and buttocks and can be misdiagnosed as a lumbar disorder. The reported incidence of SCN-EN ranges 1.6-14%.[1,2]

The most common symptom of SCN-EN is LBP around the iliac crest. It is exacerbated by lumbar movements involving flexion, extension, bending, rotation, standing, and walking. It can produce intermittent claudication, with 50% of patients reporting leg symptoms.[1,3] As these symptoms are similar to those of lumbar disease, their differentiation is important for treatment planning.

The pathogenesis of SCN-EN remains unknown. It is seen in patients with vertebral compression fractures, LDH, lumbar spinal canal stenosis, FBSS, and Parkinson's disease.[1,3-6] As it is also encountered in the elderly, soldiers, and athletes, age-related spondylotic changes, sports-related activities, high body training, and trunk rotation may be related to the manifestation of SCN-EN.[1,5-8]

Diagnosis and treatment

The SCN is thin and difficult to identify through the skin surface. As SCN-EN cannot be identified with radiological and electrophysiological studies, its diagnosis is based on clinical symptoms.[2,9] When we suspect SCN-EN because patients report LBP involving the iliac crest and buttocks, we identify the trigger point that elicits radiating pain over the posterior iliac crest located approximately 7 cm from the midline where the SCN penetrates the thoraco-lumbar fascia to confirm entrapment. The trigger point has been localized in earlier reports and is not affected by patient age, height, gender, or race. When SCN block successfully decreases pain, we make a diagnosis of SCN-EN.

SCN-EN can be treated by less invasive procedures such as local SCN block and SCN neurolysis under local anesthesia. We usually perform peripheral nerve surgery under local anesthesia without nerve block using no special techniques because we want to observe symptom changes and monitor the affected nerve during surgery. Approximately 28-100% of patients with SCN-EN respond to SCN-EN blocking.[1,2,7] In some instances, SCN block is useful for treating refractory severe LBP. If only transient pain amelioration is achieved, SCN blockage can be repeated. When SCN-EN cannot be controlled by observation therapy including SCN blocks, it can be treated by less invasive SCN neurolysis under local anesthesia.[9-11]

Gluteus medius muscle pain

Definition and symptoms

The gluteus medius muscle (GMeM) is located in the buttock over the gluteus minimus and partially under the gluteus maximus muscle; it is covered by a tight gluteal aponeurosis [Figure 2]. The GMeM supports the pelvis and femur when standing on one leg, walking, and running. GMeM pain results in buttock pain.[6,12] It is elicited by walking, prolonged sitting, standing, and standing on one leg. Lateral and posterior femoral pain is reported by 80% of patients.[12] The symptoms are similar to those of lumbar disease, and differentiation of GMeM from LBP is important for treatment planning. Given its size, the GMeM generates an exceptionally large force, and this background may be related to GMeM pain severity.[13] The GMeM plays a significant role in chronic LBP.[14-16]

Diagnosis and treatment

GMeM pain cannot be identified radiologically, so its diagnosis relies on clinical symptoms.[6,12] In our

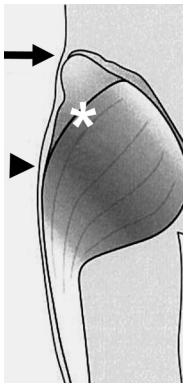

Figure 3: The trigger point (*) for gluteus medius muscle pain is located at the edge of the gluteus maximus muscle at the midpoint between the iliac crest (arrow head) and greater trochanter (arrow)

practice, we consider GMeM involvement in buttock pain when it is located around this muscle. The trigger point is located on the GMeM at the edge of the gluteus maximus muscle equidistant from the iliac crest and greater trochanter [Figure 3]. Some patients report pain radiation to the lateral-posterior thigh. When transient pain amelioration is obtained by local GMeM block, we diagnose GMeM pain.

When GMeM pain cannot be controlled by medication and physiotherapy, GMeM block may be useful. Some patients experience gait disturbance due to transient leg paralysis after blockage. Non-responders to conservative therapy may require less invasive GMeM decompression surgery under local anesthesia.[6,12] This treatment can benefit even very old patients with intractable buttock and leg pain due to the GMeM. Peripheral block and less invasive surgery under local anesthesia are other treatment options.[6]

Piriformis syndrome

Definition and symptoms

The piriformis muscle connects the sacrum and greater trochanter. When it is overburdened, buttock pain also involving the adjacent sciatic nerve with nerve pain down to the lower thigh may be experienced. The pain is similar to that elicited by S1 radiculopathy and may be attributable to anatomic anomalies of the piriformis and sciatic nerve. It is more common in women than men.[17,18]

There are no specific symptoms. Patients report lower buttock pain and S1-like sciatic pain that rarely involves the ankle. The symptoms are exacerbated by prolonged sitting, stairclimbing, and walking.[19,20] Some patients experience decreased pain with walking. The etiology of piriformis syndrome involves exercise load, trauma, and tumor; lumbar spine disease may be an idiopathic cause.

The accurate diagnosis of piriformis syndrome avoids FBSS and insufficient decompression after surgery. Among patients with LBP, 5-17% manifest piriformis syndrome.[17,19,21-23] Some patients develop piriformis syndrome after percutaneous endoscopic lumbar discectomy (PELD). Kim and Kim[22] reported that the incidence of piriformis syndrome was 13.7%. Within 3 months of PELD, 40.4% of operated patients presented with piriformis syndrome; its incidence was highest in the first postoperative month. Their observations suggest that heightened anxiety in patients undergoing PELD under local anesthesia may increase the incidence of piriformis syndrome elicited by walking. They suggested that general anesthesia may reduce the incidence of piriformis syndrome after PELD, although local anesthesia is preferable because it allows for intraoperative monitoring. Anxiolytic administration makes intraoperative patient cooperation difficult, particularly in older patients, and their use may have adverse effects. A proper preoperative period stretching of the piriformis muscle may be useful in locally anesthetized patients.

Diagnosis and treatment

Piriformis syndrome cannot be identified by radiological and electrophysiological studies; its diagnosis is based on clinical symptoms and palpation.[18-22] During palpation, the swollen, stiff piriformis muscle is identified as a sausage-shaped mass over the piriformis muscle. There is tenderness, and some patients report radiating pain along the sciatic nerve. Symptom alleviation obtained by piriformis muscle block is diagnostic.[18,20-22]

Piriformis muscle stretching is useful in addition to medication and rehabilitation [Figure 4]. Some patients experience pain alleviation upon piriformis muscle stretching, but this exercise must be continued for more than 2 weeks. When these methods fail, piriformis muscle block may be useful. Piriformis muscle block may elicit transient leg paralysis 30-60 min after injection when the anesthetic reaches the sciatic nerve. Non-responders may require piriformis muscle dissection.

Sacroiliac joint pain

Definition and symptoms

The sacroiliac joint (SIJ) connects the spine and pelvis; it is comprised of articular and posterior ligamentous compartments. It is reinforced with hard ligaments and moves only slightly. SIJ pain can be elicited by everyday activities and involves both articular and posterior ligament regions. It is felt not only in the lower back and buttocks but also in the groin and lower extremities, it can be difficult to discern from pain secondary to other disorders. The major pathological factor in SIJ pain is joint dysfunction.[24] Repetitive movements and/or accidental minor subluxation of the SIJ may damage

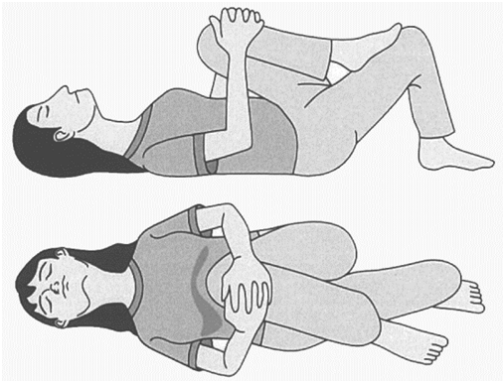

Figure 4: Piriformis muscle stretching

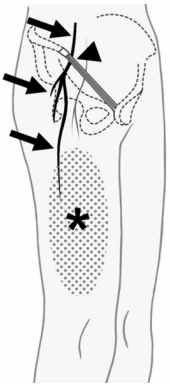

Figure 5: The course and entrapment point (arrowhead) of the lateral femoral cutaneous nerve (arrow). This sensory nerve branches off the L2 and L3 nerve roots, merges, passes through the inguinal ligament inside the superior iliac spine, and then distributes subcutaneously through the femoral fascia (*). From Clinical diagnosis for low back pain by palpation (2015), Isu T & Kim K, CHUGAIIGAKU. CO., LTD

SIJ-related structures such as the joint capsule and the posterior ligament.

SIJ pain is commonly perceived in the gluteal region; it can be referred to the lower limbs and groin region, and is similar to symptoms due to lumbar diseases.[24-26] The pain area tends to be located around or within 2 cm of the posterosuperior iliac spine (PSIS). SIJ pain should be considered in patients reporting lowerback and buttock pain.[24,27] Approximately 50% of patients with SIJ pain experience groin pain,[24,28,29] which is exacerbated by sitting on a backless chair.[24]

While SIJ pain may occur alone, 39% of patients also manifested LDH and LSS.[30] It has been reported after lumbar fusion and lumbar decompression surgery.[31-33] In patients with lumbogluteal and/or lower extremity pain and a high SIJ-related score,[24] SIJ pain should be considered even in the absence of lumbar disease or prior lumbar surgery.

Diagnosis and treatment

As it is difficult to identify SIJ pain radiologically, its diagnosis is based on clinical symptoms and the effect of SIJ block. Kurosawa *et al*.[24] proposed a score for diagnosing SIJ pain to distinguish it from pain elicited by other lumbar diseases [Table 1]. Their scoring system includes six items and is useful for both diagnosing and understanding SIJ pain. Patients often point to an area within 2 cm around the PSIS as the most painful area when instructed to identify the affected area with one finger (one-finger test). The SIJ shear test is the most useful provocation test. With the patient in the prone position, the examiner places a palm over the patient's posterior iliac wing and thrusts the palm inferiorly to produce a shearing force across the SIJ.

Besides medication and rehabilitation, conservative approaches include a pelvic belt and SIJ block. Blocking the posterior ligament and periarticular region of the SIJ under fluoroscopic guidance yielded more effective pain relief than intra-articular SIJ injection.[24,27,28,34] Injecting a local anesthetic into the posterior ligament can also relieve SIJ pain. While intra-articular SIJ injection is not recommended as a definitive diagnostic tool for pelvic girdle pain, it can be combined with the injection of a local anesthetic into the extra-articular SIJ ligaments to alleviate pain.[34,35] SIJ denervation or fixation is a treatment option in non-responders.

PERIPHERAL NERVE DISEASES

Lateral femoral cutaneous nerve EN

Definition and symptoms

The lateral femoral cutaneous nerve (LFCN) is a sensory nerve that branches off the L2 and L3 nerve roots, merges, passes through the inguinal ligament inside the superior iliac spine, and is then distributed subcutaneously through the femoral fascia [Figure 5]. The incidence of LFCN-EN is 33-43 individuals per 100,000; the site where the nerve penetrates the inguinal ligament is often involved.[36,37]

Obesity, pregnancy, compression by tight undergarments and corsets, lower abdominal surgery, autogenous iliac bone, and nerve compression due to posterior spinal surgery in the prone position have been reported to be implicated; 77% of LFCN-EN is idiopathic.[36-38] Diabetes and alcoholism are metabolic risk factors for LFCN-EN, which can be unilateral or bilateral.[38-40] The symptoms are pain, abnormal perception, numbness, and a burning sensation in the

Table 1: Sacroiliac joint pain scoring[24]

Item	Score	Odds ratio
1. One-finger test	3	25.9
2. Groin pain	2	14.5
3. Pain while sitting on achair	1	1.4
4. Sacroiliac joint shear test	1	1.8
5. PSIS tenderness	1	2.2
6. STL tenderness	1	2.2
Total score	9	

PSIS: posterosuperior iliac spine; STL: sacrotuberous ligament. Scores above 4 are considered high SIJ pain scores

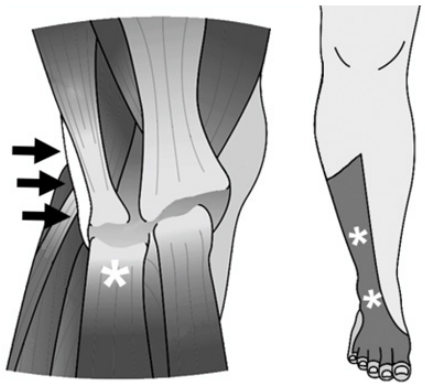

Figure 6: The common peroneal nerve (arrow) runs around the fibular head, then between the soleus and the peroneus longus muscle (PLM) (*), and then into the PLM. Patients experience pain and paresthesia of the affected area, the lateral aspect of the lower calf, and the dorsum of the foot (**)

anterior lateral region of the thigh; they are elicited by hip joint movement and alleviated by squatting. Some patients complain of intermittent claudication.[38,41,42]

Diagnosis and treatment

The symptoms above and Tinel-like signs at the nerve penetration site inside the superior iliac spine are diagnostically relevant. In some patients without clear Tinel-like symptoms, the disappearance of symptoms after nerve block is useful for a diagnosis. Electrophysiological studies can also be helpful. Patients with symptoms clearly attributable to LFCN-EN may report perception anomalies on the outside of the thigh when the nerve is compressed in the pelvis or in the presence of a retroperitoneal tumor. Consequently, pelvic lesions must be ruled out when blocking fails to be effective.

Conservative therapy and nerve block are effective in 90% of patients.[42-44] Tagliafico *et al*.[42] reported that 80% of patients improved after a single block; others required 2 blocks to decrease symptoms. The nerve block is applied at the site with Tinel-like symptoms, 2 cm inside and 2 cm below the anterior superior iliac spine. As the anesthetic infiltrates the femoral nerve running on the inside, approximately 5% of patients experience transient femoral nerve paralysis.[43,45] Non-responders to conservative therapy may require neurolysis or neurectomy under local anesthesia.[38,46,47]

Common peroneal nerve EN

Definition and symptoms

Common peroneal nerve (CPN)-EN is the most common peripheral entrapment neuropathy eliciting leg symptoms. The CPN runs around the fibular head and then between the soleus and peroneus longus muscle (PLM) to the inner PLM [Figure 6]; it can become entrapped in this area. As the nerve runs a shallow course on the bone, external compression neuropathy is not infrequent. However, EN has been reported in patients whose daily activities failed to account for its elicitation.[48-50]

The symptoms are pain and paresthesia of the affected area on the lateral aspect of the lower calf and the dorsum of the foot. Drop foot is a severe symptom, although some patients report only pain and paresthesia without severe paresis.[48-52] Walking and prolonged standing may lead to symptom exacerbation and intermittent claudication.

Diagnosis and treatment

CPN-EN cannot be diagnosed radiologically. While nerve conduction studies may be useful, the anomaly may not be detectable in patients with dynamic neuropathy-like intermittent claudication.[48-51,53] In these situations, it can be difficult to distinguish CPN-EN from lumbar spine disease because the symptomatic area is similar to L5 radiculopathy with intermittent claudication.[48-50] CPN-EN diagnosis is based on clinical symptoms. Although the Tinel-like sign is useful diagnostic information, it may be absent.[51]

Repetitive plantar flexion of the ankle joint is a useful provocation test because the CPN is entrapped by the PLM and soleus muscle;[48,49] these muscles are most heavily loaded during maximum plantar flexion. CPN-EN results in intermittent claudication. At a cutoff of 110 s, sensitivity and specificity were 94.1%, suggesting that the repetitive plantar flexion test is diagnostically useful.[48]

When conservative treatment fails, surgical neurolysis around the fibular head under local anesthesia is a useful treatment. It is important to intraoperatively confirm sufficient decompression by ankle movement because dynamic neuropathy is an important factor in CP-NEN etiology.

Superficial peroneal nerve EN

Definition and symptoms

CPN-EN is more common than superficial peroneal nerve (SPN)-EN. The SPN bifurcates from the CPN around the fibular head and runs along the peroneal tunnel between the peroneus longus/brevis muscles and the extensor digitorum longus muscle. The SPN can be entrapped in this area.

Patients with SPN entrapment report pain and paresthesia in the affected area, the lateral aspect of the lower calf, and the dorsum of the foot. Styf and Morbergfound SPN entrapment in 17 of 480 (3.5%) patients with chronic leg pain.[54] According to

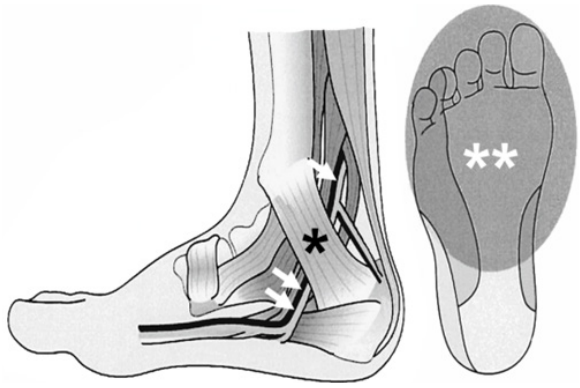

Figure 7: Tarsal tunnel syndrome is an entrapment neuropathy of the posterior tibial nerve (arrow) in the tarsal tunnel (*). Only the sole of the foot has symptoms (**); there is no heel pain

others,[55-57] SPN-EN was attributable to entrapment due to muscle herniation, trauma, compression by a mass lesion, (e.g. varicose veins or lipoma), or an idiopathic origin.

Diagnosis and treatment

SPN-EN diagnosis is based on its symptomatology because it is difficult to diagnose on radiological and nerve conduction studies.[54-57] Its symptoms tend to be exacerbated by walking and exercise, and SPN-EN must be differentiated from other lumbar diseases. The Tinel-like sign is diagnostically useful.[54-57] Tinel-like signs are occasionally observed at multiple compressed points along the SPN.[54] SPN block with lidocaine may provide transient pain relief. SPN-EN can co-exist with CPN-EN; Franco *et al*.[56] reported that 78% of patients who underwent SPN-EN surgery had undergone CPN decompression surgery. In patients with muscle herniation, static palpitation and radiological studies may not identify the lesion, and the loaded posture may be necessary for a correct diagnosis.

The decision to intervene surgically depends on SPN-EN etiology. Neurolysis may be effective in patients with idiopathic origins. The area requiring decompression is not necessarily limited to the part with the Tinel sign. Some patients may require decompression involving the area from the PLM to the SPN exit point along the SPN.

Tarsal tunnel syndrome: posterior tibial nerve EN

Definition and symptoms

The tarsal tunnel is a fibro-osseous tunnel under the flexor retinaculum below the medial malleolus. The posterior tibial nerve bifurcates to the medial and lateral plantar nerve and passes inside the tarsal tunnel together with the posterior tibial artery and vein. Tarsal tunnel syndrome (TTS) is an entrapment neuropathy of the posterior tibial nerve within the tarsal tunnel [Figure 7].

The etiology of TTS has been ascribed to tumors; changes in anatomical structures due to trauma; and idiopathic factors such as tortuous vessels, hypertrophy of the flexor retinaculum, and fibrosis with a variety of origins. Compression of tortuous arteries and veins can elicit TTS, although a tortuous vein may be a normal variation. TTS was idiopathic in 18-69% of patients.[58-60]

The symptoms are sensory disturbance in the sole of the affected foot, paresthesia, a foreign-body sensation like walking on gravel, cold sensation, and burning or tingling. They are exacerbated by prolonged standing or walking; they do not involve the heel of the affected foot (heel sparing) because the branch to the heel bifurcates proximal to the tarsal tunnel. Heel sparing is therefore useful for the diagnosis of TTS.[58,59,61-63]

Diagnosis and treatment

An accurate diagnosis is difficult, and TSS is regularly underdiagnosed based on clinical symptoms affecting the plantar aspect of the foot.[61-64] Although sonography, computed tomography, and magnetic resonance imaging studies are diagnostically useful in patients with space-occupying lesions, the identification of idiopathic TTS remains difficult. False-positive and false-negative findings make diagnosis of TTS based on electrophysiological means alone difficult.[61,65-67] A positive Tinel-like sign and radiating pain on the entrapment point of the tibial nerve in the tarsal tunnel are diagnostically useful. A positive Tinel sign is the best indicator of a favorable outcome after decompression surgery.[58,59,62,64]

TTS symptoms may be misdiagnosed as part of the symptomatology of spinal disease and as sequelae after spinal surgery. TTS was found in 4.8% of patients with lumbosacral radiculopathy and tends to complicate lumbar spine disease. FBSS should be considered in the absence of clear evidence of TTS.[68,69] TTS must be ruled out or addressed when patients treated by spinal surgery continue to experience anterior sole numbness and/or pain.

When TTS fails to respond to observation therapy, we perform neurolysis under local anesthesia.[62,63] We make a 3-4-cm bow-like incision 1.5 cm below the medial malleolus over the point of the Tinel-like sign without using a tourniquet. In some cases, besides cutting the flexor retinaculum and opening the tarsal tunnel, sufficient decompression from the neurovascular band and transposition may be necessary. Although the outcome of surgery for idiopathic TTS tends to be good, some patients experience only partial or no improvement.[60,61,64,67] Significant pain alleviation after tarsal tunnel decompression surgery has been

reported by 44-90% of patients. An incorrect diagnosis, incomplete surgical decompression, adhesive neuritis, neural trauma or damage, systemic disease, double crush syndrome, and prolonged symptoms are factors that must be considered in patients with failed TTS surgery.[60,62,64,67]

CONCLUSION

We described typical peripheral nerve and paralumbar spine diseases with symptoms similar to those of LDH. These diseases are common, and unless they are diagnosed and treated correctly, patients may progress to FBSS. In some instances, these diseases respond well to less invasive treatment methods, and some patients experience dramatic improvement. The surgical procedures described herein are less invasive and do not require sophisticated techniques.

It is important to recognize that the diseases we discussed may be associated with LDH. Therefore, a better understanding of specific diseases other than LDH and their treatment is necessary. Symptoms in patients with LDH may be attributable to such diseases, and they may accompany symptoms elicited by LDH. The careful analysis of factors that contribute to the patients' symptoms is important for making a correct diagnosis and may broaden the range of beneficial treatments and improve their quality of life.

Authors' contributions

Concept and design: K. Kim, T. Isu
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Literature search: D. Morimoto, N. Iwamoto, R. Kokubo, J. Matsumoto, T. Kitamura
Manuscript review: A. Sugawara, A. Morita

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Conflicts of interest

There are no conflicts of interest and no financial disclosures.

Patient consent

Not applicable.

Ethics approval

This review article is waived for ethical approval.

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