



Epidural Steroid Injection Might Advance Clinical and Electrophysiology Outcomes among Drop Foot Patients due to LDH Underwent of Transforaminal Microdiscectomy: A Prospective Study

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Abstract

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BACKGROUND: Drop foot can be caused by degenerative disorders affected at L4-5 (20%), L5-S1 (41.5%), or isolated L5 (25.2%), whereas 68% of cases are caused by peripheral neuropathy (18.3%) and peroneal neuropathy (31%). The Lumbar disc herniation (LDH) might cause compression and/or neuropathic problems.

AIM: We aimed to investigate how epidural steroid injection might advance clinical and electrophysiology outcomes among drop foot patients due to LDH underwent of transforaminal microdiscectomy.

METHODS: Nine subjects (4 females, 5 males), aged between 35 and 77 years old (mean 56 + 4), suffered from drop feet due to LDH for 5–18 months following traumatic experiences. Four subjects had prescribed diabetic problems, four were normal or overweight, and one was obese.

RESULTS: After 3 months of OS study, the NRS score improved (29–58.5%), while the Manual motor test varied. Subject #8, whose age is 77 years old, showed clinically bilateral drop feet; and four subjects (#2, #5, #6, #8) suffered from diabetes. Regarding the BMI in the presence of overweight or obesity, there are four subjects each, whereas the normal weight has only one (subject #5). Obese subjects showed no sign of motor improvement (20–25%), whereas pain improvement was in the range of 25–50%. The removal of disc volume is about 0.5–1 cc (mean 0.722 + 0.2), while the diameter is reduced in the range 4.3–10.99% (mean 8.63% + 2.36).

DISCUSSION: Drop feet due to LDH in accordance to bring inflammation and structural problems, so transforaminal microdiscectomy is aimed at nerve decompression and managing the internal annular layers. Blocked dorsal root ganglions might improve the clinical syndromes with regard to the nociceptive and neuropathic pain that interfere. Electromyography might examine the radiculopathies but cannot distinguish between neuropathies and myopathies clearly. Age, body weight, onset, presurgical motor strengths, level of involvement, either neuropathy or diabetes in association with the prognosis.

CONCLUSION: Decompression procedures are important for treating drop foot patients when herniated discs are the main problem.

Introduction

Drop foot is characterized by the weakness of ankle and foot dorsiflexion, which is served by the tibialis anterior, extensor digitorum longus, and extensor hallucis longus [1]. Drop foot can be caused by degenerated disc disease at L4-5 (20%), L5-S1 (41.5%), or isolated L5 (25.2%), whereas 68% of cases might be underlined with peripheral neuropathy (18.3%) or peroneal nerve lesion (31%) [2]. Drop foot might not be so usually found among lumbar disc(s) herniation Lumbar disc herniation (LDH) or stenosis patients [3], [4], unless the upper lumbar discs are involved [1]. Epidural steroids injection (ESI) following endoscopic lumbar dissection contributed to an improvement in pain and clinical condition within 6 months. Steroids improved the inflammation of the lesion, while lidocaine might improve the nerve membrane and nerve conductivity [5].

Magnetic resonance imaging (MRI) is the standard examination, even without guaranteeing proper identification. The drop foot is associated with various etiologies of neuroforamen or proximal lesion sites, and around 60–70% follow compressed nerves. The positive straight leg raise and loss of the patella tendon reflex (PTR) or achilles tendon reflex (ATR) should be found accompanying the drop foot. Lumbar plexus or sciatic nerve disorders might initially present as drop feet but involve tibialis posterior or gastrocnemius muscle weakness. The electromyography (EMG) and nerve conduction study (NCS) examinations, or electrodiagnostic examination (EDX), have the ability to differentiate between fibular or sciatic neuropathy, plexopathy, or lumbar radiculopathy. So it might be useful for neurophysiological progression evaluation [1], [6]. Both discectomy and transforaminal microdiscectomy (TFMD) yield the same results, except the shorter hospitalization went to microdiscectomy or TFMD

[7], [8]. The ESI, favorable for improving pain [8] and disc herniation resorption, underwent percutaneous endoscopic lumbar discectomy [9]. But sometimes the recurrence symptoms might emerge when the fibrosis tissues remain nearby the lesion site or are driven by residual hematoma inside the epidural space that underwent surgeries.

LDH is often accompanied by neuropathic pain symptoms due to compression of adjacent nerves. Or by following the chronic inflammation process of nearby vessels, congestion might lead to hyperacidity, so hypersensitivity occurs. The inflammation cascades promoted both peripherally and centrally T-cells and macrophage accumulation, again current in the dorsal root ganglion (DRG). When those inflammation mediators persist, the DRG might lead to chronic stimulation of the neurons, followed by the prolonged sensation of pain. Neurotropic factors released by glial cells are accompanied by peripheral nerve injury, which induces of the cell body of sensory neurons to compress. Moreover, that compression will irritate the DRG's nerve as the persistent inflammation contributes [10].

When the inflammation went into a chronic state, it might promote fibrosis in tissues adjacent to the herniated site. It might lead to neuroforamen narrowing due to inflamed edema, vascular congestion leading to local ischemic appearance, aggravating pain, and/or neurological deficits. The mid-zone is a foraminal region where the nerve root and DRG pass. The lumbar DRG, lacking a protective capsule, is commonly located in the intraforaminal area. Approximately 48% of the DRG lies intraforaminal, which is easily compressed by a narrowing neuroforamen and limited movement. This can cause radicular pain, numbness, feeling tight or heavy, motor weakness, or muscle spasms along the back region [11]. Established on this occurrence, we thought that steroid injection into the DRG might lead to the significance of TFMD utilization among drop patients better. Even without utilizing 3D visualization for endoscopic spine surgery, we believed that TFMD might lead to good results. That would not only promote clinical results but also the neurophysiology study, which might show some improvements.

Methods

The whole study was carried out in the Department of Neurology at Diponegoro University and Dr. Kariadi Hospital in Semarang, Indonesia, with the informed consent of the subjects. It was approved by the Ethical Clearance Committee: No. 753/EC/KEPK-RSDK/2021. Nine drop-foot subjects who underwent TFMD were analyzed for the NRS and EDX before surgery, as well as after 1 week and 3 months. The EDX was re-performed at 1 week, whereas the MRI was at

3 months. Subject #1 (SM) is a 58-year-old female with a BMI of 32.4 kg/m², NRS = 5 for 12 months, posterocentral protrusion (L4-5), right ligamentum flavum hypertrophied and mild listhesis (L5-S1), paresthesia, and left drop foot (Manual motor test [MMT] 5-5-5/4-4-3). Subject #2 (SD) is a 60-year-old male with a BMI of 25.8 kg/m², right drop foot (MMT 4-4-3/5-5-5), NRS = 4 for 11 months, right L3-4 and L4-5 protrusion, hypertension, and diabetes confounded. Subject #3 (IS) is a 53-year-old male with a BMI of 26.2 kg/m² who suffered from L3-4 protrusion and mild L4-5 listhesis, NRS = 6, numbness, and drop foot (4-4-3/5-5-5) for 18 months. Subject #4 (BH) is a 54-year-old male with a BMI of 31.3 kg/m², right drop foot (5-4-3/5-5-5), NRS = 4), paresthesia and numbness for 5 months, and L2-3, L3-4 posterocentral protruded disc while L4-5 in advanced. Subject #5 (MR) is a 77-year-old male with a BMI of 21.1 kg/m², NRS = 5, bilateral drop foot (4-4-3/5-5-4) for 12 months, and confounding diabetes and hypertension. MRI has shown a severe posterocentral protruded disc at L4-5 and a bulging disc at L2-3. Subject #6 (SA) is a 53-year-old female with a BMI 29.76 kg/m², who suffered from left drop foot (MMT 5-5-5/4-3-3), NRS = 4 for 7 months, confounded diabetes, and right posterolateral L4-5 right posterolateral protruded. Subject #7 (AS) is a 50-year-old female with a BMI of 31.4 kg/m², left drop foot (MMT 5-5-5/4-4-3) since 12 months ago, lower limb numbness, NRS = 4, and left posterolateral L4-5 extruded and stenosis. Subject #8 (SU) is a 53-year-old female with a BMI 31.9 kg/m², NRS = 4, paresthesia on both lower limbs, decreased PTR and ATR, bilateral foot drop (MMT 4-3-2/4-4-4) for 12 months, confounded diabetes, and hypertension, L2-3 posterocentral protrusion and L4-5, L5-S1 bulged, and L5-S1 listhesis. Subject #9 (MA) is a 35-year-old male with a BMI of 28.9 kg/m² who complained of left drop foot (MMT 5-5-5/4-4-3), NRS = 5 for 7 months, L1-2 left posterocentral bulge, L2-3 posterolateral extrusion, and L4-5 posterocentral extrusion (Table 1). The MRI re-examination might be ordered 3 months later to evaluate disc diameter, so the EDX would be 2 weeks post-surgery.

Subjects hospitalized at Dr. Kariadi Hospital Semarang with MRI and EDX examinations were ordered for 1-day surgical care. MMT follows the rules: hip flexion, calf extension, calf flexion, plantar flexion, and dorsiflexion of the ankle. It observed the improvement after surgery at 1 week and 3 months when they visited an outpatient department. The TFMD procedure was undergone by TB, a neurologist, pain specialist, and minimally invasive physician who experienced managing more than 250 patients (since 2017) with LDH or stenosis, and more than 2500 chronic pain cases (since 2012) by interventional procedures. The TFMD was done in a prone position under local anesthesia, so the awake state remains to communicate, for avoiding the potential complication. The depth and direction of insertion follow the appropriate line of the process disc(s) by posteroanterior (PA) view and the

Table 1: Subjects improvement

Subjects	Sex	Age (year old)	BMI (kg/m ²)	Onset (months)	Comorbid	Discs lesion		Motor performances		Pain intensity (NRS)		
						Lesionsites	Dissectedsites	Pre-right/left	Post-right/left	Pre	Post	Δ (%)
#1	Female	58	32.4	12	N/A	L4-5, L5-S1 listhesis	L4-5	555/543	555/544	5	3	40
#2	Male	60	25.8	11	DM, HT	L3-4, L4-5	L4-5	443/555	544/555	4	2	50
#3	Male	53	26.1	11	N/A	L3-4, L4-5 listhesis	L3-4	443/555	444/555	6	3	50
#4	Male	54	31.1	5	N/A	L2-3, L3-4, L4-5	L4-5	543/555	554/555	4	2	50
#5	Male	77	21.1	12	DM, HT	L2-3, L4-5	L4-5	555/433	555/443	5	3	40
#6	Female	53	29.76	7	DM	L4-5	L4-5	433/555	443/555	4	2	50
#7	Female	50	31.4	12	HT	L4-5	L4-5	443/555	544/555	4	3	25
#8	Female	53	31.9	12	DM, HT	L2-3, L4-5, L5-S1 listhesis	L2-3	432/444	433/444	4	2	50
#9	Male	35	28.9	7	N/A	L1-2, L2-3, L4-5	L2-3	555/443	555/444	5	3	40

Post: 3 months post-surgeries, Ø: Diameter of the disc, Δ: Improvement percentages, DM: Diabetes mellitus, HT: Hypertension, NRS: Numeric Rating Scale, N/A: Not available.

anteroposterior line by lateral view. The 1.5% lidocaine injection was followed by 18G of the spinal needle insertion to the lesion sites carefully, and then PA view shifted to observe endplates so advanced insertion penetrated the discs. It showed laterally at one-third of the posterior part of the disc, so 1 mL of dye contrast was injected to evaluate the annular tears (discography). A mini grasper plugged and removed the posterior part of the inner annular by grabbing the “disc tail” to reduce the outsider. The bipolar coagulation set up 10–15 volts applied in a circle direction for 3–45 s and coagulated the nociceptors. Cefazoline 1 g was injected inside the disc for 2–3 mL and sutured to the skin after the tube was pulled out. Disc diameter in line with the lesion sites measured and compared with the previous one to know improvement percentages. The triamcinolone 15–20 mg in dilution with lidocaine 1.5%, applied through the caudal epidural steroid (CESI) approach and transforaminal ESI (TFESI), are intended for DRG block at the surgical sites. The subjects referred to the recovery room at least stayed for 30 min to evaluate their conditions.

Results

Subjects consisted of 3 females and 6 males aged 35–77 years old (mean 56 ± 4), with pain improvement within 3 months in the range of 25–50% (Table 1), and the MMT varies. Subject #8, 77 years old, was accompanied by bilateral drop feet, and four subjects (#2, #5, #6, #8) suffered from diabetes. Regarding the BMI, four subjects were overweight, four others were obese, and only one was normal weight (#5). The EDX observed improvements in latencies, amplitudes, and NCS among all subjects. Dissected materials were about 0.5–1 cc (mean 0.722), while the surgical site diameter reductions were 4.3–10.99% (mean 8.63%) (Figure 1). Obesity subjects without pain improvement above 50%, as compared with normal or overweight subjects, at least 50%. However, it still requires large numbers of subject involvement in the study.

The EDX showed variable types of neuropathy, such as true demyelination, which affects 4 subjects (#1, #5, #6, #9), and 5 subjects (#2, #3, #4, #7, #8)

with mixed demyelination and axonal degeneration (Figure 2). The latencies, amplitude, and either NCS observed improvement among 4 diabetic patients, but the long-term onset of drop foot might interfere with the recovery. The EDX is consistent with peripheral lesions causing drop feet, but multiple site lesions require more detailed examination using needle EMG. Obese or overweight subjects were observed to have motor improvement of at least 20–25%; this spondylolisthesis or stages might interfere with the improvement. Upper and lower lumbar involvement together are required for careful determination of topical sites associated with drop foot. Tibial and peroneal nerve examination distinguished the upper lumbar (Th12-L1-2, L2-3, L3-4) from the lower (L4-5, L5-S1). The ESI accompanying TFMD might advance the outcomes.

Discussion

Chronic degenerative disc disorders are clinical diagnoses with a complex point of view due to structural changes such as calcification formation, annular tears, nuclear damage, or ligamentum hypertrophy. The inflammation mediators and neovascularization at the lesion site and nearby tissues. It leads to adhesion formation, and then neuronal sensitization and hyperalgesia appear [10]. Subjects observed with a mild improvement on motor and NCS examination following TFMD and ESI without any post-surgical complications such as spondylodiscitis are 0.12% among 9000 patients [5]. Our results are similar to those of other spinal surgical techniques, where the outcomes with improvement and satisfaction rates are beyond 75% [12]. Intradiscal bipolar coagulation can dehydrate the nucleus, shrink the annular collagen matrix, and ablate the nociceptors at the posterior annular [13]. Thus, in this study, according to the direction around the eight points surrounding the taken disc site, the work was done in 3–4 s for each point. We took more concern for the site of lesions at the inner annular layers, which could potentially be leaky locus minoris and prone to the penetration of sprouted fibers inside. In approximately 52% of cases, disc herniation might be associated with drop foot, and in 35%, with spinal canal stenosis. It is a challenge when affected by

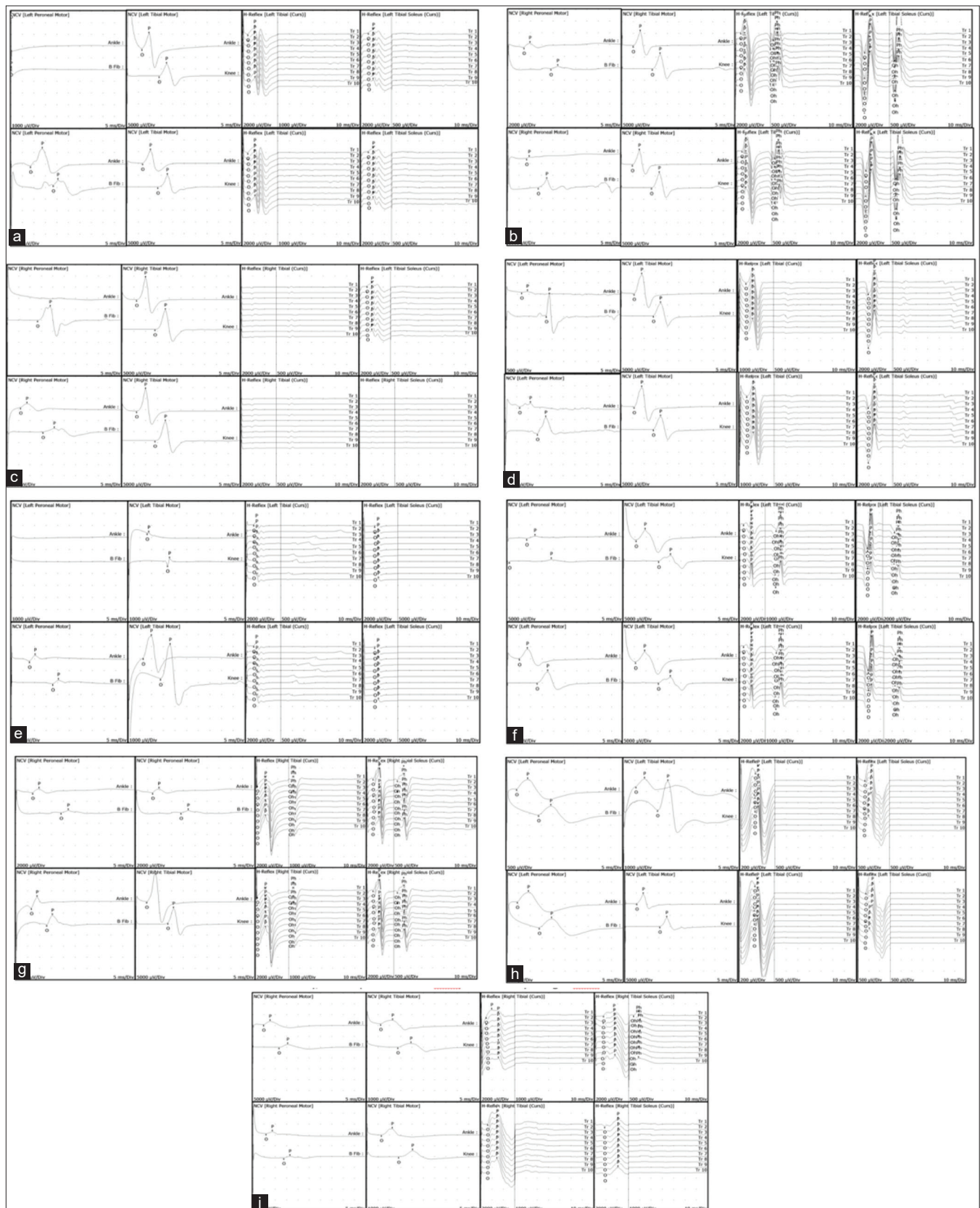


Figure 1: Electrodiagnostic examination performed on pre and post microdiscectomy: (a) Subject #1 wave formation appears, (b) Subject #2 amplitude and NCS improvement, (c) Subject #3 NCS, latency, and amplitude improvement, (d) Subject #4 NCS improvement, (e) Subject #5 latencies and NCS improvement, (f) Subject #6 amplitudes and NCS improvement, (g) Subject #7 NCS improvement, (h) Subject #8 wave productivity and NCS improvement, (i) Subject #9 amplitudes improvement of sensory nerves velocity

both a herniated disc and stenosis. The post-surgical recovery of drop foot might happen gradually in 6 weeks,

6 months later, or within 2 years. Presurgical mild motor weakness will more quickly recover than those

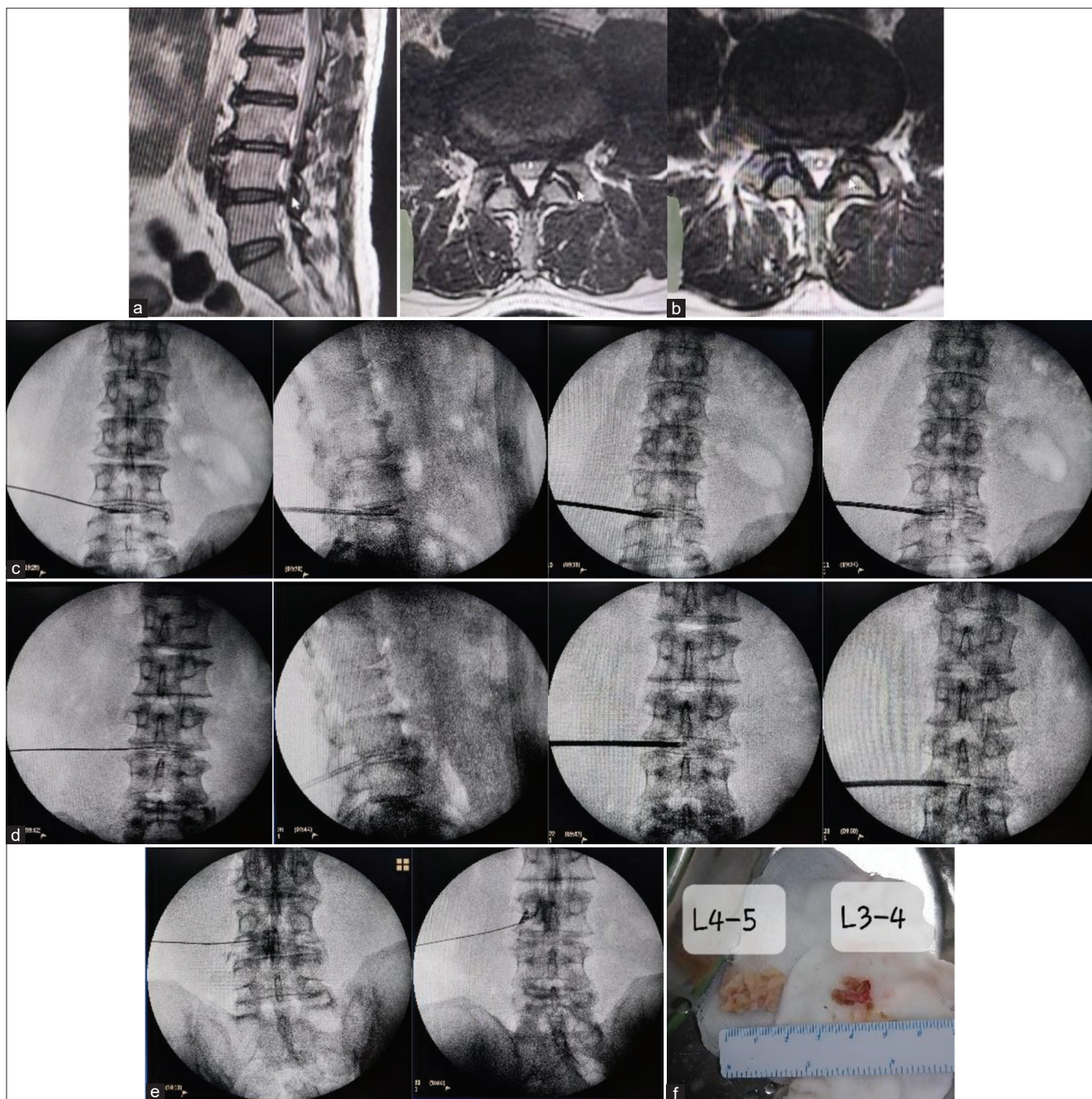


Figure 2: TFMD procedures: (a) sagittal view showed protruded discs at L3-4 and L4-5; (b) axial view showed L3-4 and L4-5 posterocentral protrusion; (c) and (d) observed of TFMD steps: guide wire inserted into the targeted disc, microtube cannula placed at one-third posterior disc near to inner layer of AF, TFESI at left DRG of L3-4 and L4-5

with severe weakness, wherein MMT ≥ 3 brings for full recovery or MMT ≥ 4 as a rate in the range 68–70%, partial recovery approximately 16–51%, <30% with remained motor weakness, surgery, and around 49% with nonresponse. While MMT <3 with fully recovering is <30% [14].

Triamcinolone is intended to treat the local inflammatory process, while lidocaine is an antinociceptive agent. Lidocaine might inhibit ectopic discharges, suppress inflammation, or modulate both inhibitory and excitatory neurotransmission [15]. Triamcinolone is an intermediate-acting steroid that showed better pain relief than the long-acting steroid

betamethasone or dexamethasone [16]. Low-dose TFESI improves the pain from disc herniation better than CESI, with effectiveness at least for 6 months [17], [18], while it also reduces the herniated volume [19]. Lidocaine can block the sodium channel and nociceptive transmission by raising the membrane depolarization threshold [20]. Thus, diluted preparations of lidocaine and steroids might reduce the nerve sensitivity caused by irritant materials [21]. We purposed TFESI here for blocking the DRG, which is involved in neuropathic pain development through the expression of ion channels or receptors, which play a role in transduction, transmission, and modulation of afferent

impulses. The sympathetic nerve fibers pass through to the DRG myelin sheath. The transduction electrical impulse is exhibited by the order of transient receptor potential voltage channels, Na⁺ channels, acid-sensing ion channels, and adenosine-5'-triphosphate (ATP)-sensitive receptor involvement. When injury occurs, the DRG becomes hyperexcitable by increasing glial cells within and producing ectopic firing. Glial cells contain cytokines, bradykinin, chemokines, ATP, and others, which play a role in the signal transmission phase to DRG. Calcium channels and glutamate receptors might cause dorsal horn presynaptic neurotransmitter release and modulate transmission. This fact supports the role of DRG in the onset of neuropathic pain as well as the chronicity of the pain that occurs. Injured tissues lead to peripheral nociceptors activation and sensitization, which activates glial cells within the DRG [22]. The DRG lies within the zone formed by the neuroforamen and vertebral pedicles, so it is able to connect with the spinal canal. A herniated disc, ligamentum flavum, blood vessels, or other adjacent tissues might cause mechanical compression of the DRG. Based on it, the posterolateral herniated disc might be followed by severe pain intensity [23]. The compressed roots are more sensitive to mechanical and thermal stimulation, so the excitatory threshold decreases. The root action potential extends up to 50%, and the pain intensity increases. Approximately 48–70% of the lumbar DRG lies within the neuroforamen, and the transforaminal approach can reach it by going slightly deeper. The sympathetic nerve might increase the intra-radicular, sciatic nerve, and cauda equina vascularization of the DRG. When the sympathetic nerve circulation improves, then mechanical allodynia, TNF- α expression, and DRG neuron apoptosis due to nerve injury might be inhibited [24], [25].

Two kinds of nerves with an important role for the intervertebral discs and surrounded tissues are: the sinuvertebral nerve (SVN) and basivertebral nerve (BVN). The SVN is derived from the spinal nerve and has a connection to sympathetic nerves, and the innervation has an extended distribution to posterior annulus fibrosis. When the disc degeneration happened, the sprouted nerves from the SVN might penetrate inside the disc through the annulus layers and promote discogenic pain. Thus, a structural lesion might also lead to radicular pain without clearly causing structural inflammation. SVN originated from ventral rami for somatic branches and united with autonomic roots. Spinal nerves appeared more distal than the DRG and supplied both proprioceptive and nociceptive fibers. The BVN, which plays an important role in vertebral endplate nociceptive pain transmission, together with the SVN, enter the vertebral body through the central vascular foramen around the endplate [26]. By flushing normal saline, it might develop a mechanical force to wash out the inflammatory mediators lying adjacent to the DRG. Hence, it can be helped to lose the sticky or

surrounding adhesion tissues.

Neuropathies are found in approximately 2–3% of the population, starting at 55 years old, with the potential to raise the number of cases. An action potential originates from activated muscles recorded by EDX and is known as a compound muscle action potential (CMAP). Whereas sensory nerve conduction is figured out by the innervated skin distal to the targeted nerves and is known as sensory nerve action potential. Three major features are observed: amplitude, distal latency, and conduction velocity, so the problems from the myelin sheath or axons can be distinguished [27]. Nerve conduction seems to decline starting at 30–40 years old, and a 60-year-old might be below 10 m/s. Or the decreasing rate of an average of 0.41 m/s in every advanced age. Aging might influence either the ATP energy metabolism, neuromuscular junction, or demyelination changes [28] and correlate with the decreasing of NCS while the distal latency increases. When the CMAP amplitude is reduced and the distal latency and conduction are normal, axonal neuropathy might be suspected. When the fibers are lost and the conduction velocity is getting milder or slower, it can be thought of as mixed axonal demyelination [27]. Some subjects in this study suffered from diabetes mellitus, so the neurophysiological studies were done following axonal neuropathies.

The radiculopathies examination might be applied by inserting a small electrode needle into the muscle to distinguish a myopathy or neuropathy from motor weakness. A needle EMG examination might show no abnormality if the onset is <3 weeks, whereas fibrillation or fasciculation will be found if the patient has suffered from radiculopathies for more than 3 weeks. The motor unit action potential (MUAP) can be presented with a long duration and large amplitude polyphasic when the disorder happened for 3 or 4 months. Axonal Wallerian degeneration is formed when the disorders happen beyond 4 or 6 weeks after onset. The subjects have already experienced the demyelination phase based on the onset time and EDX outcomes. When the radiculopathies went to chronic or even severe polyneuropathies, the MUAP was observed to elongate while the nerve amplitudes might reduce. Age factors also play a role in the outcomes, which cause the declination to be around 0.5–4 m/s per decade, either due to the body heights [27]. Overweight or obesity might cause spinal mechanical loading, an increased share of spinal lumbar, or annular tears in advance, so back pain increases. Metabolic syndromes have been associated with abdominal obesity, so TNF- α and IL-6 expression address systemic inflammation. It leads to peripheral and central sensitization associated with positive and negative symptoms of neuropathic pain [29], [30]. Degeneration processes are caused by inappropriate or lack of nourishment, so further denervation of the endplates and disc happened [31].

EDX is an important tool to evaluate regional nerve innervation disorders, because it is able to specifically examine the anatomic location. Drop foot might be caused by problems in the sciatic and tibial nerves together or by a single peroneal nerve injury. If the EMG shows any sciatic lesion or radiculopathy accompanied by abnormal tibial EDX, it can be concluded that the drop foot is the cause. When the tibial EDX result showed no abnormality, the drop foot might have originated from peroneal lesions, which means the lesion site is at the proximal or upper lumbar levels. The Babinski sign and PTR and/or ATR hyperreflexes appearance might be important signs for a central lesion. In elderly people or osteoporotic patients, attention should be paid to sacrum region deformities such as fractures, trauma, radiotherapies, malignancies, or diabetic inflammation, especially if there are no impinged nerve roots [1]. We argue that ESI might help with a better clinical and EDX examination. In addition to the MRI study with a sensitivity of 87.5% and a specificity of around 57.1%, NCS has a 65.2% sensitivity and 28.6% specificity for lumbar radiculopathy [32]. Combining EDX, needle EMG, and MRI examinations makes the diagnosis and evaluation more accurate.

Conclusion

The long-term duration of the lesion and precisely the indication and sites of the lesion, age, presurgical motor strengthening, anatomy levels of involvement, confounding neuropathy disorders, and overweight are associated with the prognosis. Posterolateral herniation or stenosis with more severe pain, caused by compression or DRG affected. Thus, it might interfere with local circulation and affect oxygenation, otherwise causing the formation of local adhesion. By blocking at the DRG as high as the lesion site, it might bring on pain improvement due to the compressed event, either postsurgical pain syndromes or TFMD. However, because of the small number of subjects involved and the lack of needle EMG, the monitoring has limitations in distinguishing neuropathies or myopathies. We have not understood enough about the outcomes related to decompression or steroid administration.

Author Contributions

TB planned the study and performed the experiment assisted by RP. Together with AH, EK and DP, we analyzed the data and wrote the article for

publication purposes. All authors have read and agreed to the published version of the manuscript. TB designed the study by assisting with RP perform the experiments. EK was performing and analyze of electrophysiology AH, and DP analyze the data then finalized the draft. All authors have read and agreed to the submitted manuscript. analyzed the data and wrote the first draft of the manuscript.

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