



Factors that influence neurological deficit and recovery in lumbar disc prolapse—a narrative review

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Abstract

Neurodeficit due to lumbar disc herniation (LDH) is a serious complication and can range from sensory hypoesthesia in a single dermatome to a debilitating condition like cauda equina syndrome (CES). Many authors have described variable clinical and radiological risk factors for neurodeficit in LDH. Similarly the prognostic factors influencing recovery have been variable across the studies. This narrative review discusses the pathogenesis, most consistent factors associated with the occurrence of neurodeficit in LDH patients and also the factors which have a prognostic role in recovery. Pathological mechanisms like venous congestion, neuronal ischaemia and multiple root impairment act either individually or in combination to result in neurodeficit in LDH patients. Among the clinical risk factors, diabetes mellitus and acute onset of symptoms have been associated with neurodeficit while pre-existing spinal canal stenosis, non-contained discs (sequestered/migrated) are potential radiological risk factors for developing neurodeficit. When considering prognostic factors for recovery, pre-operative muscle strength is the only significant factor. Knowledge about the causative and prognostic factors in neurodeficit following LDH would help in the successful management of this condition.

Keywords Lumbar disc · Motor deficit · Neurological deficit · Foot drop · Recovery · Review

Introduction

Lumbar disc herniation (LDH) is the most common cause of low back pain and sciatica [1]. A host of biochemical and mechanical factors play a role in the pathogenesis of sciatica by LDH [2]. Hakelius et al., on the natural history of LDH in conservatively managed patients have shown that about 80% of patients have symptomatic relief in six weeks and about 93% in 24 weeks [3]. Indications for surgical intervention in LDH include severe, intractable pain and pain unresponsive to conservative treatment [4]. Various surgical options like open/microscopic discectomy [5, 6], recent minimally invasive procedures including tubular discectomy [7],

endoscopic discectomy [8, 9] and radiofrequency techniques [10] have been advocated in the management of disc herniations. Irrespective of the technique employed, the primary aim of all the procedures is to remove the offending disc and decompress the neural structures. LDH can lead to motor deficit, sensory disturbance as well as bowel and bladder dysfunction. Patients with bowel/bladder dysfunction and/or progressive motor deficit have definite indications for surgery. Neurological deficit due to LDH is a dreadful sequelae and it can lead to long-lasting disabilities. Studies have identified multiple clinical risk factors (age, sex, co morbid conditions, onset of symptoms) and radiological risk factors (canal dimension, level of disc herniation, morphology of the disc, multiple root compression) to be variably associated with the development of neurological deficit in LDH. Similarly, factors like age, co morbid conditions, pre-operative muscle strength, palsy duration/time interval to surgery, and presence of leg pain have been shown to have a predictive role in recovery following surgical intervention for LDH. We present this review on *clinical and radiographic characteristics* associated with the presence of neurological deficit and the *factors which influence recovery* of neurological deficits in LDH.

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Pathophysiology of neurodeficit in LDH

Neurodeficits in LDH may range from simple sensory loss involving a single dermatome to a potentially debilitating condition like foot drop. Features of cauda equina compression such as sacral anaesthesia and bladder/bowel involvement can also be the presenting features. Various pathological mechanisms act either individually or in combination to give rise to neurodeficits in LDH patients. They include:

1. Venous congestion: Obstruction to venous circulation by herniated disc or the migrated disc fragments resulting in venous congestion can cause direct compression of the nerve root leading to impaired nerve root function. Venous congestion is also shown to induce pathologic changes within and around the nerve root complex like oedema, peri- and intra-neural fibrosis or focal demyelination [11]. Orhan Kalemci et al. [12] reported on contralateral deficit in LDH patient caused by venous congestion which resolved with cauterisation of the engorged veins.
2. Neuronal ischemia: Mechanical compression by herniated disc results in neuronal ischemia and this is proven by Shinichi Hida et al. [13] in their *in vivo* study where they found a significant increase in the nerve root blood flow measurements in patients whose neurological deficit or pain resolved shortly after surgery when compared to patients whose neurological deficit did not recover after surgery
3. Impairment of multiple roots: Even though L5 nerve root is most affected due to LDH, patients can also present with weakness of adjacent roots like L4 and S1. This impairment in function of multiple roots is due to upward or downward migration of sequestered fragment from ruptured disc and can possibly explain the genesis of foot drop in single-level LDH [14].
4. Compression at multiple sites: Olmarker et al. [15] in an *in vitro* study on porcine cauda equina showed that double-level compression had a more deleterious effect on nerve function than single-level compression. The above scenario can be seen in LDH with concomitant stenosis.

Risk factors predicting neurological deficit in LDH

The risk factors can be broadly divided into clinical and radiological.

Clinical factors influencing neurological deficit

Diabetes mellitus (DM) DM has been shown to be an independent risk factor for neurodeficit due to LDH. Arinzon et al., [16] in their comparative study on outcomes in diabetic and

non-diabetic patients after decompression surgery for spinal stenosis, found that DM patients experienced more pain, motor weakness, and numbness in the affected limb when compared to non-diabetic patients. Even though pain relief was comparable in both groups, DM patients had more revision surgeries and higher post-operative complications. When analysing DM as a risk factor for neurodeficits in LDH; Krishnan et al. [17] in their prospective comparative study had 14 (20%) DM patients in neurodeficit group and only 3(4%) DM patients in non-deficit group and they found DM as a significant factor contributing to the presence of neurological deficit. Jun Ma et al. [18] in their risk factor analysis study in 236 (52 neurodeficits and 184 normal neurology) patients concluded that DM (19.2% versus 10.3%) is an independent predictor of the presence of foot drop in LDH.

Neuropathic pain is common in diabetic patients and may cause diagnostic difficulty in differentiating pain due to neuropathy and LDH. Diagnosis depends on the presence of neurodeficits in anatomical distribution, clinical correlation with imaging and supplemental neuro-diagnostic tests. The presence of concomitant neuropathy and vasculopathy in DM patients and the diminished ability of the neuronal tissues to sustain mechanical and vascular insults due to LDH is a probable reason behind the association of DM with neurodeficits.

Onset of symptoms Nerve compression due to LDH induces mechanical deformation of the nerve and has effects on the nutritional supply to the nerve tissue [19]. The rate of onset of compression whether acute or insidious can have a bearing on the irreversible mechanical deformation of the nerve tissue. Krishnan et al. [17] categorised the onset of symptoms as acute (<3 months) and chronic (>3 months) and about 84% of patients in their neurodeficit group had acute onset of symptoms. Jun Ma et al. [18] also had a similar finding and 73% patients with foot drop in their study had acute onset of symptoms. The fact that acute compression of the nerve causes significant damage was shown by *in vitro* study [20] on porcine cauda equina where rapid compression of the nerve roots caused more profound effects on nerve conduction, intra-neuronal edema formation, and methyl-glucose transport when compared to gradual onset of compression. This can be extrapolated to explain the presence of neurodeficits in patients with acute or acute on chronic presentation in LDH.

Duration of symptoms This clinical factor may have a prognostic role in neurological recovery following surgery which will be analysed in the later part of the review but all the previous studies [17, 18, 21] are in agreement that duration of symptoms is not a significant factor in predicting the presence of neurodeficits.

Other clinical factors like age, sex, precipitating event, number of previous symptomatic episodes, side of symptoms, unilateral or bilateral, presence or absence of radiculopathy/back pain and nerve root tension signs are insignificant factors across all studies in predicting neurodeficits in LDH.

Radiological factors influencing the presence of neurological deficit

Level of disc herniation Higher level DH even though uncommon, tend to present with neurodeficit. Considering the narrower spinal canal at higher levels, disc herniation at these levels results in greater compromise of the nerve roots when compared to lower lumbar herniations. In the study by Krishnan et al. [17], the most common level of disc herniation in deficit group was L4–L5 with 41 (58.5%) patients followed by L3–L4 level with 17 (24.2%) patients. L3–L4 level was statistically significant in this study in its association with neurodeficits. Even though only 17% of the patients in their study group had higher level DH, neurodeficits were found in 85% of these patients. L4–L5 with 31 (59.6%) patients was the most common level in the neurodeficit group of Jun Ma et al. [18]. Only 18 patients had L3–L4 disc (17 patients with normal neurology, 1 with deficit) and they did not find a statistical significance with respect to level of disc herniation and neurodeficits. Dora et al. [22] in their study on discriminating between symptomatic and asymptomatic discs concluded that symptomatic disc herniation patients had smaller spinal canal dimensions. Lurie et al. [23] in their study on outcomes in LDH at different levels of herniation have demonstrated that higher level lumbar disc herniations were more far-laterally or foraminal located close to the nerve root than lower lumbar disc prolapse which might explain the presence of deficits more commonly in higher level disc herniations.

Morphology of disc herniation

a. Type of herniation: Combined Task Force (CTF) classification defines lumbar discs as normal, focal protrusion, and broad-based protrusion, or extrusion. A sequestered disc is one in which a fragment of a disc gets separated from the parent disc and can migrate freely in any direction [24]. Of these, focal/broad-based protrusions are contained and extrusion/sequestrations are non-contained. Jonsson B et al. [25] in their study on the clinical appearance of contained and non-contained lumbar disc herniations found non-contained LDH to be associated with the incidence of relevant reflex/extensor hallucis longus (EHL) and sensory disturbance. 82% of the patients

in the non-contained group had deficit when compared to 40% in the contained group. Krishnan et al. [17] in their study compared protruded, extruded and sequestered discs in predicting neurodeficits. 11 (15.7%) sequestered and 17 (24.2%) superiorly migrated discs were associated with neurodeficits and they showed that sequestered superiorly migrated discs were more frequently associated with neurological deficit. Izuka et al. [14] correlated intra-operative finding with the presence of foot drop and found that sequestered and migrated fragments were associated with double nerve root compression due to the migrated fragments and hence the presence of foot drop. Suzuki et al. [21] in their study on risk factors for motor weakness and delayed recovery in L4–L5 DH showed on multivariate analysis that non-contained and migrated type of HNP were associated commonly with motor weakness. Postacchini et al. [26] in their study on recovery of motor deficits following LDH found intra-operatively that most of the DH were either extruded or sequestered. Thus there is supporting evidence that non-contained (especially sequestered) and migrated DH is commonly associated with neurodeficits (Fig. 1).

- b) Location of DH: Based on location, LDH is classified into central, paracentral, lateral and foraminal herniations. Jun Ma et al. [18] found lateral recess and foraminal type of herniation to be associated commonly with presence of foot drop. N.E Epstein et al. [27] in their review on foraminal and far lateral disc herniation concluded that far lateral DH are associated with neurodeficits in over 75% of patients. Studies have shown that these far lateral discs are usually supero-laterally migrated fragments from the ruptured parent disc [28, 29]. These migrated fragments exert undue tension on the exiting roots whose mobility is already restricted because of the fibrous bands which attach them to the foramen.
- c) Central disc: Only one study by Krishnan et al. [17] showed higher incidence of deficits in central disc herniations (69%).
- d) Calcified disc: Jun Ma. et al. [18] showed calcified discs to be associated more commonly with deficits (23.2% in deficit versus 4.3% in non-deficit group). It is proven that the osteogenic potential of the degenerated disc is more towards the spinal canal than towards the centre of the disc indicating that there is chronic compression of the nerve roots by the calcified portion of the disc. There is no concrete evidence to show central, calcified discs have an association with neurodeficits.



Fig. 1 Morphological variants of disc herniations which are commonly associated with neurodeficits **a, b** Sagittal and coronal magnetic resonance images (MRI) showing foraminal disc herniation. **c, d** Inferiorly migrated disc. **e, f** Superiorly migrated disc **g, h** sequestered disc

Spinal canal dimension and canal occupancy Spinal canal dimension and canal occupancy by the herniated disc are important factors for predicting neurological deficit in LDH. Dora et al. [22] in their comparative study on the significance of spinal canal dimension in discriminating between symptomatic and asymptomatic individuals measured the spinal canal dimension in both mid-sagittal (at level of pedicles, disc) and axial sections and found that symptomatic patients with LDH had spinal canal dimensions which are significantly smaller than in asymptomatic patients (Fig. 2).

Krishnan et al. [17] also found the mean bony canal dimension significantly smaller in the neurodeficit patients when compared to the non-deficit patients. They found patients with bigger discs occupying larger spinal canal dimensions are more commonly associated with deficits. In their study, the mean antero-posterior (AP) disc dimension was 8.7 mm in the neurodeficit group, in comparison with 7.5 mm in the non-deficit group. Canal compromise was 63.5% in the neurodeficit group, in comparison with 52.3% in the non-deficit group. Jun Ma et al. [18] showed that canal occupancy by the disc which was greater than 50% of the antero-posterior diameter of the canal is a risk factor for developing foot drop. So we can infer that a large disc herniation in a wide spinal canal can remain asymptomatic, while small

disc herniations in a narrow spinal canal can cause severe radicular pain and deficits (Fig. 3).

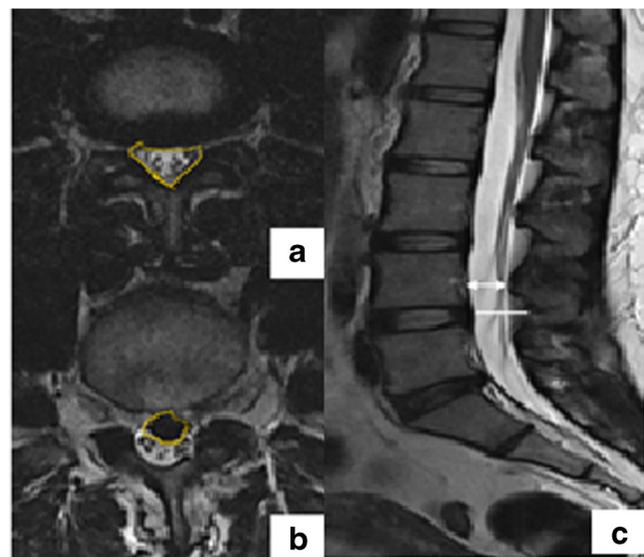


Fig. 2 Spinal canal and disc dimension measurement. **a** Spinal canal dimension measurement on axial T2 weighted (T2W) magnetic resonance image (MRI); **b** disc dimension measurement on axial image; **c** mid sagittal measurement of spinal canal dimension at the level of pedicle (double arrow) and at the level of the disc (white line). Dora et al. [22]

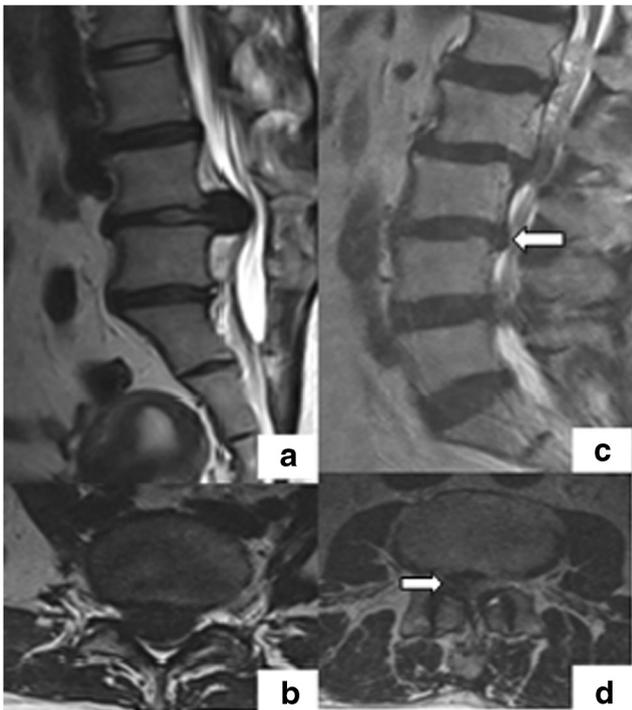


Fig. 3 Disc dimension and canal occupancy. **a, b:** Sagittal and axial magnetic resonance images (MRI) showing a huge disc herniation occupying majority of the canal. **c, d:** Sagittal and axial MRI showing primary canal stenosis with small disc herniation which is susceptible to develop deficit

Other factors like multiple level DH, Pfirrmann's grading, Modic changes and pre-existing foraminal stenosis are not predictors of neurological deficit in LDH patients.

Prognostic factors for recovery of neurological deficit in LDH

In this section, various factors which have been thought to have a bearing on the recovery of neurodeficits in LDH patients will be discussed.

Surgery versus conservative management Most often the presence of neurological deficit in LDH is seen as an indication for surgery. On the contrary, Weber et al. [30] in their randomised study used objective measurement of muscle power [31] to document weakness and they concluded that operative treatment did not give better prognosis for the recovery of motor deficit when compared to conservative management. They subjected their patients to conservative management of two weeks duration with the aim of giving pain relief as they felt radicular pain would alter the measurements of muscle function. One drawback of the study is that about 80% or more paresis (severe deficit) was seen in only eight (2.8%) out of 280 patients included in the study. Duborg et al. [32] in their observational pilot study reported a recovery of 32% in the non-operative group and 25% in the surgical

group and they did not find any statistical difference between the two groups. The surgical group in their study included patients with more severe weakness than the conservatively managed patients which is a major confounding factor. Buttermann et al. [33] in their prospective randomised study found significant improvement in the severe motor deficit patients with operative management when compared to the group which was given epidural steroid injection. The patients in the crossover group who were operated after trial of epidural injection also recovered significantly even though offered late surgical intervention. All the other studies show a recovery rate ranging from 16 to 76% [14, 26, 34, 35] after surgical management which is higher than that of Duborg et al. [32]. Considering the recovery rates with surgical management, it should be offered to patients with neurodeficits; especially in patients with severe neurodeficit (< 3/5MRC).

Time to surgery following motor deficit While prognosticating recovery, time to surgical intervention should be considered with utmost importance. In the prospective study on recovery of motor deficits by Postacchini et al. [26], the average duration to surgery from the time of weakness was 45 days (7–553 days) and they showed that patients who were operated upon within 35 days of onset of weakness had complete recovery than those operated upon after 69 days. Patients who had surgery after 70 days of motor deficit had no recovery. This includes patients with severe (< 3/5) and very severe weakness. Aono et al. [36] on their analysis of recovery of foot drop caused by degenerative lumbar conditions reported a recovery rate of tibialis anterior (TA) by at least 2 grades in 61% of their patients and they found a significant difference in the surgical outcomes in patients with varying duration of foot drop. Patients with shorter duration of weakness had better recovery rates and the odds ratio almost doubled (1.81) when the duration of foot drop doubled and they could prove this association even on multivariate analysis. However they included cases with either LDH (60%) or LSS (40%) in their analysis. Bhargava et al. [37] in their retrospective study which included patients with both LDH (88%) and LSS (12%) showed complete recovery of TA function in patients with weakness of a mean of 1.1 weeks, some recovery in patients with mean weakness of 5.6 weeks and no improvement in patients with a mean weakness of 18.3 weeks.

The retrospective study of Ghahreman et al. [34] on recovery of foot drop following lumbar decompression surgery showed no correlation between the duration of weakness to recovery of motor deficit. The average time interval to surgery following weakness was 14 days (range: 1–180 days) in their study and comparing to other studies they had a shorter time interval to surgery. Girardi et al. [38] in their retrospective study did not find any significance between the duration of symptoms and the recovery of foot drop. The mean duration was in fact more in patients who recovered (17.6 weeks) when

compared to patients who had some post-operative weakness (8.4 weeks). All their patients were treated non-surgically for a mean of 3.7 months (range 6 days to 2.5 years) before surgery. Lonne et al. [39] in their prospective study on the recovery of deficits in discectomy patients had a mean duration of paresis of 30 days (median 6 days). They evaluated several time frames for the duration of paresis, which could indicate a good or bad prognosis for recovery like two days, median (6 days), two weeks, one month, but did not find a statistically significant threshold. Eysel et al. [35] in their prognostic study on lumbar paresis had a mean duration of weakness of seven days and they did not find duration of paresis a significant predictor of outcome. With regard to the time to surgery, only Postacchini et al. [26] have conclusively told the time period (70 days) after deficit when no recovery can be expected. All other studies have compared their average time to surgery with the two groups (recovery versus no recovery) and we cannot come to a conclusion that early surgery has a favourable prognosis on recovery; however, in patients who present with acute motor deficits, Ondra et al. [40] concluded that patients with moderate (3/5 MRC) and severe (< 3/5 MRC) deficit surgery within 48 hours of the onset of deficit had better outcomes when compared to surgery after 48 h.

Pre-operative muscle strength Most of the studies [21, 26, 34, 37, 39, 41] on the prognostic factors of recovery of motor deficits conclude that pre-operative muscle strength is a significant factor in predicting recovery. Izuka et al. [14] showed no association between pre-operative motor power and recovery in disc herniation patients but they could find significance in the LSS patients in the same study. Girardi et al. [38] also did not find a correlation between pre-operative muscle strength and motor recovery but their conclusion cannot be considered as they used a different grading system for muscle power. Considering the data above, pre-operative muscle strength is a significant predictor of motor recovery.

Age of the patient It is a well-known fact that in spinal cord injury (SCI), younger individuals have a better recovery potential when compared to older individuals. When considering age as a prognostic factor in LDH patients with neurodeficit, Duborg et al. [32] found average age in completely recovered patients significantly lower compared to those with no recovery (43 vs. 51 years); however, age did not achieve significance when comparing those improving one grade of power compared to those with no improvement in the same study. Aono et al. [36], comparing age at surgery found better surgical results with younger patients and concluded that age at surgery significantly affected post-operative tibialis anterior strength but they could not prove the same association on multivariate analysis. Ghahreman et al. [34] also found that younger age group patients showed better recovery than elderly patients; however, they considered a wide range of age

groups for comparison (25–40 years, 45–60 years, > 60 years). Barring the above studies, all the other studies [14, 26, 35, 37, 38, 42] are in agreement that age is not a prognostic factor in neurological recovery following LDH.

A brief description of the prognostic factors evaluated by different authors; with their potential drawbacks are shown in Table 1.

Other factors including gender, presence or absence of radicular pain, multiple muscle group involvement, smoking, diabetes, alcohol consumption and obesity have been shown to have no association with recovery of motor deficits in LDH.

Cauda equina syndrome (CES)

Since clinical manifestations and outcomes vary in patients with CES due to LDH in comparison to patients with isolated motor/sensory deficits, we are discussing it as a separate entity. CES as a complication of LDH is seen in 1–10% of all cases of LDH [43–46]. It manifests as a combination of sensory loss of the saddle area, motor deficit and/or loss of reflexes of the lower limbs, micturition dysfunction, defecation complaints, and/or sexual dysfunction [47, 48]. CES has been clinically divided into CESS (Suspected), CESI (Incomplete) and CESR (Retention). CESI is a patient with urinary difficulties of neurogenic origin, including altered urinary sensation, loss of desire to void, poor urinary stream and a need to strain in order to micturate [49]. CESR is a patient with painless urinary retention with overflow incontinence where the bladder is no longer under control. CESC is complete loss of all cauda equina function with absent perineal sensation, a patulous anus and a paralysed insensate bladder and bowel. A CESS patient is one with unilateral or bilateral radicular pain at risk of, but does not have sphincteric involvement [50].

Literature review on predictive factors for presence of CES in LDH shows no clinico-radiological correlation. Korse et al. [42] did not find any association between MRI findings (level of LDH, severity of cauda equina compression and side of compression) and the clinical presentation (bladder/bowel involvement, saddle hypo/anaesthesia or sciatica) in CES. In the study, authors also compared spinal canal dimensions in patients with CES due to LDH with patients operated for sciatica secondary to LDH and they concluded that patients with CES had smaller AP canal dimension when compared to patients with LDH and sciatica. When considering gender as a variable in CES, Korse et al. [48] found female patients more likely to present with defecation dysfunction than their male counterparts (OR = 4.11).

On the mode of presentation and outcomes in CES patients, Korse et al. [51] in their systematic review reported that the pre-operative presenting complaints of the patient like micturition dysfunction, defecation dysfunction and saddle hypo/anaesthesia had no prognostic effect on the outcome of micturition, defecation and sexual function. Possible factors

Table 1 Summary of the studies included in the review with the prognostic factors and potential drawbacks (*) of the study

Sl.no	Author	Type of study	Study population(n)	No association	Positive association	Comments
1.	Postacchini [20]	Prospective	LDH (n = 116)	1. Radiological(contained versus non-contained) 2. Site of LDH-foraminal or extra-foraminal 3. Age 4. Comorbidities	1. Preop muscle strength 2. Duration of weakness	*No patients with complete paralysis
2.	Duborg [26]	Prospective multi-centre	LDH (n = 67) 39 (58.2%) surgery, 28 (41.7%) conservative	1. Type of DH (contained versus non contained) 2. Surgery versus conservative 3. Duration of weakness 4. Gender	1. Age (completely recovered)	*No significance when comparing one grade improvement with no recovery *Greater no of paretic muscle, longer duration of sciatica in surgical group *Patients with LSS were included *Early surgery (mean 14 days) after onset of weakness *Only patients with L4–L5 LDH included. *Most patients (n = 29) with < 4/5 power included *Both LDH and LSS patients included *No sub group analysis
3.	Ghahreman [28]	Retrospective	LDH(49)+LSS(7)	1. Duration of weakness 2. Gender 3. LDH versus LSS	1. Age	
4.	Suzuki [15]	Retrospective	LDH (n = 43)	1. Age 2. Gender 3. Percentage occupancy by disc 4. Migration of disc 5. Duration of weakness	1. Non contained disc 2. Pre-operative muscle strength	
5.	Anno [30]	Retrospective	LDH (n = 24) + LSS (n = 16)	1. LDH versus LSS 2. Age 3. Gender 4. Single-level versus multiple-level 5. Leg pain versus no leg pain	1. Pre-operative muscle strength 2. Duration of weakness	
6.	D. Bhargava [31]	Retrospective	LDH (n = 23) + Lateral recess stenosis (n = 3)	1. Pre-operative muscle strength 2. Level of disc 3. Type of DH 4. Size of DH 5. Presence or absence of radicular pain	1. Duration of weakness	*Both LDH and stenosis patients included *No subgroup analysis
7.	Girardi [32]	Retrospective	LDH (n = 40) + LSS (n = 15)	1. Age 2. LDH versus LSS 3. Comorbidities 4. Pre-operative muscle strength 5. Duration of weakness	Nil	*Both LDH and stenosis patients included *No subgroup analysis
8.	Izuka [8]	Retrospective	LDH (n = 26) + LSS (n = 12)	1. Age 2. Gender 3. Pre-operative muscle strength 4. Diabetes mellitus 5. Duration of weakness	1. LDH versus LSS °Pre-operative muscle strength and duration of weakness significant in LSS patients	*Both LDH and stenosis patients included *For analysis muscle strength divided into severe paresis (< 3/5), Mild paresis (< 4/5) for analysis
9.	Lomme [33]	Prospective	LDH (n = 91)	1. Age 2. Gender 3. BMI 4. Duration of weakness 5. Smoking	1. Pre-operative muscle strength	*Shorter duration between onset of motor deficit and surgery *260 patients operated < 4 weeks and only 30 operated > 4 weeks after onset of deficit.
10.	Eysel [29]	Prospective	LDH(n = 295)	1. Age 2. Sex 3. BMI 4. Type of DH (contained versus non contained)	1. Pre-operative muscle strength	

influencing outcome in CES have been evaluated before, of which time to decompression is the most frequently studied parameter. Korse et al. [48] on predictors of outcome in CES patients involving factors like age, gender, duration of complaints of herniated disc, duration of CES complaints, time to decompression (stratified into < 36 hours and > 36 hours), found that patients decompressed within 36 hours experienced more sciatica than patients decompressed after 36 hours (79.4% versus 37.9) at first follow-up (mean 75 hours), but no such association between timing to decompression and outcome of sciatica was observed at second follow-up (mean 63 days). No other factors were significant predictors of outcome in their study. Qureshi et al. [52] stratified time to decompression in their study into three groups (< 24 hours, 24–48 hours, > 48 hours) and they did not find significant correlation between the outcomes and time to decompression. Another study by Oliver et al. [43] showed a recovery of bladder function in 90% of patients and recovery of function was not related to the time to surgical intervention (< 48 hours versus > 48 hours). Similar to the above studies, there are studies that advise for early surgery in CES. Srikantharajah et al. [45] in their study on bladder outcomes in CES patients reported that decompression surgery within 24 hours of onset of autonomic symptoms in CESI reduces bladder dysfunction at initial follow-up, but no statistically significant difference was observed in CESR patients with regard to time of surgery. Delong et al. [53] in their meta-analysis of observational studies concluded that early surgery (< 48 hours) is beneficial for CES and patients with CESR and those with CESI should not be combined while analysing the results of surgical timing. Todd et al. [50] in their review article have concluded that emergency surgical decompression is required in patients with CES with deterioration, CESI, CESR (uncertain/early with preserved cauda equina function). Even though time to decompression is a controversial topic, it is widely accepted that delay in the time to surgery yields poor outcomes.

Conclusion

Neurodeficit in LDH is a serious complication and it can give rise to long lasting disabilities. Clinical risk factors like DM and acute onset of symptoms were found to be associated with the presence of neurodeficit. Among radiological parameters, pre-existing spinal canal stenosis, large discs occupying greater spinal canal dimensions, sequestered/migrated disc fragments and foraminal type of discs are predisposed to development of neurological deficits. On analysing the prognostic factors for recovery, pre-operative muscle strength is the only strong indicator. The variable conclusions reported in studies with respect to duration of weakness can be explained by the differences in the pre-operative muscle weakness, gradation of weakness and time intervals chosen to prognosticate recovery.

Knowledge of the predictive and prognostic factors is essential in management of patients with neurodeficit in LDH.

Compliance with ethical standards

Conflict of interest On behalf of all authors, the corresponding author states that there is no conflict of interest.

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