

Review Paper

Neuourogenital Aspects of Lumbosacral Spinal Stenosis



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ABSTRACT

Background and Aim: Lumbosacral spinal stenosis (LSS) is a disease in elderly cases and in its advanced forms gives rise to intractable leg pain and in many cases various degrees of bladder dysfunction. In LSS bladder dysfunction can occur following compression of lumbar nerve roots or cauda equine. Urological manifestations like lower urinary tract complaints (LUTC) and sexual dysfunction of LSS are rare and reviewed in this study. From neuro-urological aspect urodynamic study (UDS) is vital in LSS and assesses the function of lower urinary tract, provides informations about diagnosis and prognosis, and permits planing a rational therapeutic pain to prevent renal dysfunction.

Methods and Materials/Patients: Using the keywords lumbar stenosis, neurogenic bladder, surgical decompression, and UDS, all the corresponding surveys were held on Google Scholar, and PubMed Medline.

Results: Urological presentations of LSS are rare; however, many patients present with various degrees of bladder malfunction. UDS is crucial in detecting neurogenic bladder malfunction, preventing renal function aggravation, and evaluating the neuro-urological effects of LSS surgery.

Conclusion: LSS may present with genitourinary symptoms. In contrast to conservative therapy, surgical decompression yields an adequate improvement in the neuourological aspects of LSS, with no significant relationship between the operation for LSS and the resolution of the upper urinary tract lesions (UUTL). More conclusive studies with a long follow-up period and a large case population are required to explore the final treatment results.

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Highlights

- Bladder dysfunction can occur in association with LSS and can create urinary reflow to the ureter and UUTL.
- UDS can aid in the primary diagnosis and assessment of cases with neurogenic bladder, as well as in targeting therapeutic modalities, especially for preventing renal dysfunction.
- There is a drastic improvement in maximum flow rate, post-voiding residual urine, and maximum cystometric capacity following surgical decompression.

Plain Language Summary

Lumbosacral spinal stenosis (LSS) is a disease of elderly cases with intractable leg pain in advanced forms and, in many cases, with various degrees of bladder dysfunction and rarely occurring genitourinary manifestations. Urodynamic study (UDS) is crucial in detecting neurogenic bladder dysfunction, preventing renal function deterioration, and evaluating the surgical results on the neurourological effects of LSS. Operation is the best choice in LSS cases with positive radiologic findings and proper clinical correlations. The operation has been shown to create an appropriate improvement of the neuro urological aspects of LSS.

1. Introduction

The stenosis of the spinal canal, nerve root canals, or intervertebral foramina can create spinal stenosis. Neuronal injury, ischemia, or edema can occur following nerve roots or cord compression, and the resulting symptoms can vary depending on the affected cord segment. Neuro-urological manifestations of lumbosacral spinal stenosis (LSS) are rare; however, many patients present with various degrees of bladder dysfunction. A correlation is observed between the urodynamic findings and the area and degree of injury [1]. For the treatment of neuro-urological sequelae of LSS, an urodynamic study (UDS) is obligatory in determining the appropriate intervention and often in guiding neurosurgical management. Surgical decompression as laminectomy brings about subjective improvement in fifty percent of LSS cases with associated voiding symptoms [2].

2. Methods and Materials

For writing this narrative article, we provided up-to-date information on the neurological manifestations of LSS with a precise review of articles about these findings in LSS. Using the keywords "lumbar stenosis," "neurogenic bladder," "surgical decompression," and "urodynamic study," all corresponding studies were retrieved from [Google Scholar](#), [PubMed](#), and [Medline](#), and were precisely studied.

3. Results

The peripheral sympathetic, parasympathetic, and somatic innervations of the lower urinary tract control bladder filling and emptying [3], and the sacral reflex center (S2–S4) modulates bladder contraction and fullness [3]. The anatomic location of the conus medullaris in adults is at the level of L1-L2, and below this level is the cauda equina [4]. Therefore, LSS is usually created lower than upper motor neuron symptoms and signs. Nerve compression can lead structural neuronal injury, neuronal ischemia, and/or edema, resulting in axonal transport restriction [1-4], disruption of signals from the sacral reflex center, and functional impairment of the neurogenic bladder and urethra. In cases involving cauda equina injury in association with damage to the pelvic and pudendal nuclei in the sacral cord, an areflexic neurogenic bladder typically develops [1-4].

4. Discussion

Neuro-uro-genital aspects of LSS

I-neuro-urological malfunction

Lower urinary tract complaints (LUTC) can occur in about half of the cases referred for treatment of intractable leg pain or neurogenic claudication with LSS [5]. These complaints may be overlooked or attributed to disorders, such as benign prostatic hyperplasia [2-5]. Acute central LSS may present concomitantly with

cauda equina syndrome (CES), which is frequently presented with sphincter malfunction, incontinence, disordered anal tone, saddle anesthesia and bilateral sciatica and warranting surgical therapy [6, 7]. In LSS and lumbar disc herniation (LDH), bladder malfunction, such as neurogenic underactive bladder, is a frequent finding, and also urinary incontinence can occur in both conditions. LSS, tethered cord syndrome, LDH, trauma, lumbar spondylosis, and intraspinal tumors can create neuropathic bladder [6-8]. Sekido et al. based on a rat model of LSS, showed complaints of hypoactive bladder or detrusor hypoactivity, low voiding effectiveness, and high post-void residual volume (PVR) [9-12]. Sone et al. surveyed cases with LSS, showing the occurrence of urological symptoms, such as dysuria and urinary retention in 73.9% of cases, underactive or acontractile detrusor, in 40.6% of patients, and overactive detrusor in 10.6% of patients [13]. Yamanishi et al. studied 80 cases of cauda equina impairment due to LSS and showed that 21% experienced detrusor areflexia and overactivity, while 4% experienced detrusor-sphincter dyssynergia. Cases with lower LSS may present with urge incontinence upon walking due to detrusor overactivity [14]. Shi et al. showed that bilateral radiculopathy and sciatica were the initial phases of CES, and these findings are red flags for developing an advanced clinical condition [15]. Yamazaki et al. presented a case of urinary incontinence that deteriorated with walking, associated with LSS at the L4/L5 level, and showed an overactive bladder on a cystometrogram [16]. They proposed that a hyperactive bladder may be a probable complaint of LSS and a precursor to urinary retention [16]. Mitra et al. presented an elderly female case with chronic back pain and urinary urgency with a final diagnosis of hyperactive bladder in association with serious central canal L4-L5 LSS and with significant improvement of low back pain and urgency following epidural steroid injections; therefore, they concluded that severe central LSS created the patient's overactive bladder [17]. In LSS, due to the unknown acceptance of bowel and bladder dysfunction as consequences of aging, they do not always complain of these symptoms [12-17]. Therefore, the correlation between these symptoms and LSS is difficult for both patients and doctors. Surveys have failed to show a significant relationship between PVR and the subjective complaints of neurogenic bladder dysfunction in LSS cases [12-17].

II-renal osteodystrophy

A case report showed a probable relationship between renal osteodystrophy and LSS [18]. In this case report study, a woman with achondroplasia and ad-

vanced renal disorder with severe renal osteodystrophy presented with symptoms of LSS, and the authors concluded that renal osteodystrophy might have aggravated the LSS, which is a customary character of achondroplasia [18]. In another case report of simultaneous LSS and renal osteodystrophy, neurological symptoms began following renal osteodystrophy [19], and a probable etiologic correlation between these two disorders [19]. Moreland et al. also showed a correlation between renal osteodystrophy and LSS [20]. Also, a link is observed between thoracic spinal stenosis and renal osteodystrophy [21].

III-sexual impairment

Sexual dysfunction is a rare probable presentation of LSS, and there are a few case reports on repetitive priapism, otherwise named spontaneous and painful penile erection, in association with LSS [22, 23]. Pressure effect on the sacral nerve roots of S2-4 can result in erectile dysfunction (ED) [24]; however, ED has a propensity to be a neglected complaint in LSS. Gempt et al. conducted a retrospective study, showing a prevalence of 89.5% of ED in LSS cases [24]. They reported a higher prevalence of ED in LSS cases in compared to the prevalence of age-dependent ED and other medical conditions associated with ED [24]. In a particular case report on a patient with degenerative LSS, authors reported a recurrent priapism with exacerbation during walking, even in short distances, that was attributable to LSS [25].

UDS

Bladder dysfunction can occur in association with LSS and may lead to urinary reflux to the ureter and upper urinary tract lesions (UUTL). UDS can aid in the primary diagnosis and assessment of cases with neurogenic bladder, as well as in targeting therapeutic modalities, especially for the prevention of renal dysfunction [26]. Additionally, UDS can detect bladder overdistension in LSS [27]. Before operation, to prioritize at-risk cases and prevent injury to the detrusor muscle, a urological consultation should be performed. In UDS, an 8F double-lumen catheter is placed inside the bladder for measuring PVR. For measuring abdominal pressure, an intrarectal pressure sensor is also placed. During the filling and voiding stages, intravesical and abdominal pressures are measured simultaneously. Maximum cystometric capacity is assessed before voiding [26, 27]. Cong et al. studied cases with LSS and neurogenic bladder and showed high bladder capacity, high PVR, low bladder compliance, decreased maximum flow rate, and maximum detrusor pressure, with incidental findings of

bladder malfunction in asymptomatic cases. Therefore, the modality's utility for early detection can be used in asymptomatic cases [26, 27]. During this study, the operation and urodynamic studies were performed pre- and post-operatively and significant improvement was shown in the maximum flow rate, maximum cystometric capacity, and PVR [26, 27].

Effects of surgical decompression on neuro-uro-genital manifestations of LSS

Operation is the best choice in LSS cases with positive radiologic findings and proper clinical correlations. The real aim of the operation, such as decompressive laminectomy, is the achievement of symptomatic relief and better recovery of nerve roots and/or cauda equina [28, 29]. Operation in LSS has numerous beneficial effects, with 50% to 86% of outcomes being considered good or excellent [30]. The UDS findings in LSS can detect and predict alterations in bladder function in cases with or without complaints of neurogenic bladder, respectively, and also explore drastic improvement in maximum flow rate, post-voiding residual urine, and maximum cystometric capacity following decompression. Deen et al. showed that laminectomy in advanced LSS had an appropriate effect on bladder function in 60% of the cases and no effect in 40% of the cases [2] and revealed improvement of residual urine volume and maximum urine flow rates without any changes in cytometrography and electromyography results, urine flow pattern and bladder capacity [2]. Other surveys have also confirmed the beneficial effects of operations such as laminectomy, on urge incontinence and erection issues. Buchner and Schiltewolf [31] showed that surgical decompression in the cases with CES due to LDH had a recovery rate of urinary function of 77% following surgery. Radulović et al. [32] showed a recovery rate of 89% of bladder function in cases with CES due to LDH. In some patients with LSS, UDS identified asymptomatic neurogenic bladder dysfunction that demonstrated the creature of significant bladder injury before obvious signs of urinary disorders. Bemelmans et al. [33] showed the disordered urodynamic findings in 52% of asymptomatic cases. This suggests that urodynamic assessment may play a significant role in the initial detection of neurogenic bladder dysfunction.

UUTL, like hydronephrosis, is a significant complication associated with neurogenic bladder. In LSS cases with neurogenic bladder dysfunction, a significant relationship is observed between urodynamic findings and UUTL, both pre- and post-operatively. Detrusor malfunction can lead to low bladder compliance, char-

acterized by the aggravation of the bladder wall, resulting in hypertrophy and stiffness, which in turn creates increasing intravesical pressure during both storage and voiding stages. Decreased bladder compliance can create UUTL, like in cases with myelomeningocele [34]. Hydronephrosis and renal deterioration may occur due to elevated voiding pressures and increased bladder contraction time [35]. Detrusor sphincter dyssynergia (DSD) might create high resistance of functional bladder outlet obstruction and harm the vesicoureteral antireflux system. Sidi et al. [36] showed an increased risk of UUTL in association with DSD or low-compliance bladders with elevated pressure. Weld et al. [37] showed that ureter decompensation and repeated urinary tract infections can occur in cases with DSD and create significant renal disease. Kurzrock and Polse [38] showed a significant relationship between the urodynamic characteristics of leak-point pressure, compliance, and DSD, and renal function aggravation, recommending that UDS can aid in the diagnosis of cases at risk of such aggravations. However, no significant relationship between decompression surgery and the resolution of UUTL was observed, likely due to the short follow-up time in this survey [38].

If abnormal urodynamic parameters persist after lumbar decompression early additional interventions should be suggested for the prevention of upper tract lesions. Based on the various urodynamic findings, conservative and/or surgical therapy can be recommended for the management of neurogenic bladder [35-40]. For evaluating the beneficial effects of therapy and monitoring the risk of renal complications, serial UDS is obligatory. The presented studies have some limitations, like a low number of cases and short follow-up times; therefore, data from a huge number of cases with longer follow-up time are required. UDS is crucial in detecting the types of neurogenic bladder malfunction, predicting UUTL in the initial disease stage (especially in cases without frank urological complaints), and guiding clinical treatment for cases with LSS, both before and after operation [35-40].

5. Conclusion

The classic presentation of LSS cases is neurogenic claudication symptoms. However, these cases may also present with genitourinary symptoms. In contrast to medical therapy, the operation has been shown to create an appropriate improvement in neuro-urological aspects of LSS. No significant relationships are observed between the operation of LSS and UUTL resolution; therefore, more conclusive studies involving longer

follow-up times and increased cases population size are required to explore the final treatment results.

Ethical Considerations

Compliance with ethical guidelines

There were no ethical considerations to be considered in this research.

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Authors' contributions

Conceptualization, study design, review and editing: Navid Kalani; Data collection: Navid Kalani and Ali Kazeminezhad; Writing the original draft: Ali Kazeminezhad; Data analysis, interpretation and final approval: Hossein Hakimellahi and Ali Kazeminezhad.

Conflict of interest

The authors declared no conflict of interest.

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