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CYSTOMETRIC FINDINGS IN PATIENTS HAVING LUMBAR INTERVERTEBRAL DISK PROTRUSION

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Abstract: Lumbar spinal stenosis usually leads to different degrees of nerve damage, presenting with back and leg pain, and/or neurogenic bladder symptoms. To determine whether lumbar decompression improved urological function, bladder dysfunction was evaluated in this retrospective study of 26 patients with lumbar spinal stenosis who had undergone lumbar decompression surgery. Urodynamic study procedures were performed pre-operatively and 6 months post-operatively. The rating score system and disability index were employed for clinical evaluation. Following surgery, post-voiding residual urine, maximum cystometric capacity and maximum flow rate improved significantly. There was no statistically significant improvement in voided volume, bladder compliance, maximum detrusor pressure or upper urinary tract damage. Urodynamic study was important in the diagnosis of neurogenic bladder dysfunction, prevention of renal deterioration and assessment of postoperative effects after surgical decompression for patients with lumbar spinal stenosis.

Keywords: Urinary bladder; neurogenic; spinal cord injuries; urodynamics.

Introduction

Lumbar spinal stenosis – which results from congenital or degenerative constriction of the spinal canal, lateral recesses or neural foramina and leads to lumbosacral nerve root or cauda equina compression – is acommon disorder that may be present in isolation, with or without disc herniation, or associated with degenerative spondylolisthesis or scoliosis [1,2]. Neurological compression on lumbar nerve roots or cauda equina is often associated with bladder dysfunction, which leads to reflow to the ureter and may result in upper urinary system damage [3]. Studying a patient's urodynamic patterns may, therefore, assistwith the early recognition and evaluation of neurogenic bladder, reveal its prognosis and help to identify appropriate treatments to prevent renal dysfunction, which is the worst complication of this condition. The present study was to explore the urodynamic findings and their association with upper urinary tract lesions in patients with lumbar spinal stenosis, and to reveal whether lumbar decompression can prevent upper urinary system damage.

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Patients And Methods

This retrospective study included adult patients with lumbar spinal stenosis and neurogenic bladder who had undergone lumbar decompression surgery at the Urology Department, University Hospital Centre “Mother Teresa”, Tirana, Albania between 2014 and 2018. Patients were excluded if they had undergone any surgical procedure involving the lower urinary tract. Because this was a retrospective study, no ethical committee review or patient informed consent were required. A diagnosis of lumbar spinal stenosis was made based on each patient’s history, clinical features and physical examination, and confirmed by radiological techniques (namely, computed tomography [CT] and magnetic resonance imaging). The midline osseous sagittal diameter was measured as described by reseachers <12 mm is considered relative stenosis; and <10 mm is considered absolute stenosis. Neurogenic bladder dysfunction was diagnosed pre-operatively according to the standards of the International Continence Society.8 The upper and lower urinary tracts were evaluated. Upper-tract evaluations included renal function tests, renal scans with ultrasound, or CT scans. Lower-tract evaluations included urodynamic study and electromyography (EMG) to determine bladder function, and fluoroscopic video imaging to detect the presence of vesicoureteral reflux and to visualize the bladder anatomy.

Lumbar Spine Surgicalprocedures

All operations were performed with the patient in a prone position, using a standard surgical procedure for posterior lumbar spine surgery. Central and foraminal decompression for relieving the oppression to lumbar nerve roots and/or dural sac was undertaken in all patients. Spinal stabilization was achieved by inserting pedicle screws with a rod system, following decompression. Patients remained in hospital for 10 – 14 days, depending on the requirements of standard surgical care.

Urodynamic Study Procedures

Urodynamic study procedures were conducted pre-operatively and 6 months after lumbar decompression by one urologist who was unaware of the final diagnosis and just conducted the study procedures objectively. At the beginning of the urodynamic study, an 8-F double lumen catheter was introduced into the bladder. After urination, the residual bladder volume was measured by aspirating residual urine through a catheter and measuring the volume using a syringe, after which an intravenous infusion of normal saline (rate of 40 ml/min) was initiated. Abdominal pressure was measured with an intrarectal pressure sensor device (Bonito UDS-600 System; Laborie Medical Technologies, Mississauga, Ontario, Canada).

Maximum cystometric capacity was recorded using the Bonito UDS-600 System before the patient began to void. During the filling and voiding phases, intravesical and abdominal pressures were determined simultaneously. A bladder volume > 400 ml was defined as normal capacity. Normal bladder compliance was determined as > 15 ml/cmH2O and the threshold of maximum detrusor pressure was 40 cmH2O. The Japanese Orthopaedic Association (JOA) score rating system and the Oswestry Disability Index (ODI) were used for clinical evaluations pre-operatively and 6 months post-operatively.9 – 11 Sensory, reflex and motor signs in the lower extremities and saddle area were also assessed.

Statistical Analysis

Data are presented as mean ± SD or as thenumber of patients. All analyses were carried out using the SPSS® statistical package, version 20.0 (SPSS Inc., Chicago, IL, USA) for Windows®. Measurement data were analysed using one-factor analysis of variance and enumeration data were analysed using Fisher's exact test. A P-value < 0.05 was considered to be statistically significant.

Results

Data were collected from 26 patients with lumbar spinal stenosis and neurogenic bladder conditions. Patients' ages ranged from 36 to 72 years (mean ± SD 38.2 ± 12.1 years); 16 patients were male. No patient had previously undergone any surgical procedure involving the lower urinary tract. A diagnosis of lumbar spinal stenosis (at L2/3, L3/4, L4/5 and L5/S1 levels) was confirmed in all patients, using radiological evidence: 15 cases (57.7%) had one-segment involvement, nine cases (34.6%) had twosegment involvement and two cases (7.7%) had three-segment involvement. Fifteen cases (57.7%) were diagnosed as acquired lumbar spinal stenosis, six cases (23.1%) were lumbar disc herniation (LDH) and five cases (19.2%)were degenerative spondylolisthesis. All patients had neurogenic claudication. The mean ± SD history of symptoms was 7.7 ± 5.9 years and symptoms were resistant to conservative treatment. Pre-operatively, the mean ± SD diameter of the lumbar spinal canal was 6.58 ± 2.25 mm. Following decompression surgery, it increased to 14.15 ± 1.28 mm. Pre-operative results of the urodynamic study showed that 16 of the 26 patients (61.5%) were diagnosed with detrusor areflexia, which referred to no detrusor contraction during the voiding phase; the remaining patients (10 of the 26 [38.5%]) had normal detrusor activity. Pre-operatively, six patients with abnormal urodynamic findings had no clinical symptoms involving the lower urinary tract. Following lumbar decompression, seven of the 16 (43.8%) cases with abnormal pre-operative urodynamic findings became normal and no patients with

normal pre-operative urodynamic results developed abnormalities. Detrusor sphincter dyssynergia (DSD) – defined as the presence of an involuntary contraction of the external sphincter during an involuntary detrusor contraction – was identified in eight patients pre-operatively. In five cases, DSD continued after decompression. When the functional results were evaluated with the JOA score rating system, the mean ± SD scores were 15.8 ± 3.9 pre-operatively and 23.2 ± 3.6 post-operatively ($P < 0.05$). For the ODI, the mean ± SD pre-operative score was 47.5 ± 19.6, falling to 20.3 ± 9.7 points postoperatively ($P < 0.05$). Nineteen patients (73.1%) had satisfactory responses following surgery. Table 1 presents the results of the urodynamic study procedures. Values for bladder compliance, voided volume, maximum flow rate and maximum detrusor pressure increased after surgical treatment, although this increase was only statistically significant for maximum flow rate ($P < 0.05$). Post-voiding residual urine ($P < 0.05$) and maximum cystometric capacity ($P < 0.05$) were significantly improved at 6 months after surgery.

Table 1. The results of the urodynamic study procedures

Urodynamic parameter	Pre-operation	Post-operation (6 months)
VV (ml)	132.1 ± 85.0	182.2 ± 52.4
PVR (ml)	214.5 ± 97.9	44.0 ± 23.2*
MCC (ml)	475.8 ± 174.2	386.2 ± 130.6*
Pdet,.max (cmH2O)	23.2 ± 7.7	27.6 ± 8.5
BC (ml/cmH2O)	17.0 ± 7.0	21.0 ± 7.9
Qmax (ml/s)	11.6 ± 5.6	18.5 ± 6.5*

Pre-operatively, upper urinary tract lesions were found in 13 cases (50.0%), consisting of hydronephrosis (two cases), urinary infection (three cases), upper-tract stones (three cases) and abnormal renal function (five cases). Postoperatively, six cases (23.1%) showed upper urinary tract lesions: urinary infections (two cases), upper tract stones (one case) and abnormal renal function (three cases). The data in Table 2 illustrate a significant correlation ($P < 0.05$) between urodynamic results and upper urinary tract lesions, preand post-operatively. Surgical decompression showed significant improvement in bladder function ($P < 0.05$), but no significant effect on improving upper urinary tract lesions.

Discussion

Bladder filling and emptying involve the sympathetic, parasympathetic and somatic innervations of the lower urinary tract [4]. The sacral reflex centre (S2 – S4) participates in

modulating bladder contraction and fullness [5]. The conus medullaris in adults is usually located at the level of the first vertebral body [6]. Thus, lumbar spinal stenosis usually causes lower rather than upper motor neuron lesions. Nerve compression may lead to structural neuronal damage, neuronal ischaemia or oedema, and axonal transport inhibition.13 Signals from the sacral reflex centre are disrupted, and functional disturbance of the neurogenic bladder and urethra may occur. In general, an areflexic neurogenic bladder usually appears in patients with cauda equina injury when the pelvic and pudendal nuclei in the sacral cord are damaged [7]. It was shown in the present study that the results of EMG corresponded to the performance of lower motor neuron lesions defined as detrusor areflexia. Surgery is the best option in patients with lumbar spinal stenosis who have positive radiographic findings and appropriate clinical correlations. The goal of surgical treatment is to provide relief of symptoms by decompression of the spinal canal and neural foramina. Traditionally, the procedure is a decompressive laminectomy, consisting of removal of the spinous processes, lamina, ligamenta flava and medial portions of facet joints [8]. The nerve roots and/or cauda equina are better able to recover physiologically following decompression.12 The many surgical benefits of treating lumbar stenosis have been described and show that the rates of good or excellent results – defined as symptom relief and return to pre-morbid activity levels – range from 50% to 86%.14 In the present study, 19 patients (73.1%) showed satisfactory results following surgical decompression. Most of the patients benefited from surgery in terms of pain reduction, increase in ordinary activities and walking ability. The procedures employed in the present study also yielded significant post-operative improvements in JOA and ODI scores ($P < 0.05$). The urodynamic study showed patients with symptoms of neurogenic bladder such as increased bladder capacity and residual urine volume, decreased bladder compliance, and lower maximum flow rate and maximum detrusor pressure. Changes in bladder function could be predicted on the basis of the urodynamic findings. After decompression, maximum flow rate, post-voiding residual urine and maximum cystometric capacity improved significantly ($P < 0.05$). Seven of the 16 patients (43.8%) with abnormal preoperation urodynamic results achieved recovery in urodynamic findings after surgery. The present study suggests that lumbar decompression contributes to relieving neurogenic bladder dysfunction in patients with lumbar spinal stenosis. Researchers [9] reviewed 22 patients who underwent surgical decompression following a diagnosis of cauda equina syndrome due to LDH, 17 of whom (77%) recovered complete urinary function following surgery. They reported that 89% of patients with cauda equina syndrome, caused by disc herniation,

achieved good or excellent results in bladder function recovery. Researchers [10] described subjective improvements in bladder function in 12 patients (60%) following surgery: residual urine volume and maximum urine flow rates improved, but the results of cytometrography and electromyography, urine flow pattern and bladder capacity were unchanged. No patient with normal urodynamic results in the present study developed abnormalities after surgery, which demonstrated that decompression surgery was an effective treatment with a good safety profile. Urodynamic study identified asymptomatic neurogenic bladder dysfunction in six patients in the present study, demonstrating the occurrence of substantial bladder damage before signs of urinary disorders were evident. Reserachers described the presence of altered urodynamic patterns in 52% of patients without clinical symptoms. This indicates that urodynamic evaluation might play an important role in the early diagnosis of neurogenic bladder dysfunction. The most important types of damage associated with neurogenic bladder dysfunction include upper urinary tract lesions (such as hydronephrosis), urinary infections and renal complications. Data from the present study indicate a significant correlation between urodynamic findings and upper urinary tract lesions, both preoperatively ($P < 0.05$) and post-operatively ($P < 0.05$), in lumbar spinal stenosis patients with neurogenic bladder dysfunction. Detrusor dysfunction might cause decreased bladder compliance, associated with deterioration of the bladder wall, with hypertrophy and stiffness. This leads to increases in intravesical pressure in the storage and voiding phases. Reduced bladder compliance is thought to be a risk factor for upper-tract deterioration in patients with meningomyelocele [12]. In addition, high voiding pressures and prolonged duration of bladder contractions might cause hydronephrosis and renal deterioration. DSD might lead to increased resistance of functional bladder outlet obstruction and damage the mechanism of vesicoureteral antireflux. Researchers [13] that DSD or high-pressure, reducedcompliance bladders were at high risk of upper urinary tract changes. They indicated that DSD was associated with complete injuries, elevated intravesical pressures and upper-tract complications ($P < 0.01$): the subsequent insufficient drainage of the upper urinary tract led to ureter decompensation and recurrent urinary tract infections. Increased intravesical pressures, vesicoureteral reflux and urinary tract infections finally led to serious renal disease. Researchesr [14] found statistically significant relationships between the urodynamic parameters of leak-point pressure, compliance and DSD, and renal deterioration, suggesting that urodynamics assist in identifying patients at risk of such deterioration. In the present study, urodynamic parameters were improved after surgical treatment and, simultaneously, upper urinary tract lesions

improved. There was no significant correlation between decompression surgery and improvement in upper urinary tract lesions, possibly because the 6-month follow-up time was too short to demonstrate recovery of the involved nerves. Additional interventions could be suggested for patients treated with lumbar decompression who continue to indicate abnormal urodynamic parameters. The management of neurogenic bladder dysfunction includes conservative and surgical treatments on the basis of different ur5dynamic results [15]. Serial urodynamic studies should be performed in order to evaluate the efficacy of treatment and monitor the possibility of renal complications. There were some inherent limitations to the present study, including the relatively small number of patients and the short follow-up period. Data from a larger patient population that include long-term outcomes are necessary to determine whether the results remain consistent.

Conclusion

Patients with lumbar spinal stenosis mainly showed signs of detrusor areflexia on urodynamic study. Although significant differences were only found in post-voiding residual urine, maximum cystometric capacity and maximum flow rate, urodynamic study was important in identifying types of neurogenic bladder dysfunction, predicting upper urinary tract lesions during the early disease phase (especially in patients without obvious urological symptoms), and instructing clinical therapy for patients with lumbar spinal stenosis, both pre and postoperatively. No significant correlations between lumbar decompression and upper urinary tract lesion improvement were found; therefore, surveys involving a longer follow-up period may be necessary to reveal the final treatment results. Clinical interventions should be applied early, to prevent upper urinary tract lesions after lumbar decompression surgery if urodynamic parameters remain abnormal.

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