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Urodynamic Profile of Patients with Neurogenic Bladder after Intervention

Dr. Herion Dredha[1], Arsen Seferi[2]

[1]Urology Department, University Hospital Centre "Mother Teresa", Tirana, Albania (Correponding author)

[2]Neuroscience Department, University Hospital "Mother Teresa", Tirana, Albania

Abstract: ***The normal function of the urinary bladder is to store and expel urine in a coordinated, controlled fashion. This coordinated activity is regulated by the central and peripheral nervous systems. Neurogenic bladder is a term applied to urinary bladder malfunction due to neurologic dysfunction emanating from internal or external trauma, disease, or injury. Symptoms of neurogenic bladder range from detrusor underactivity to overactivity, depending on the site of neurologic insult. The urinary sphincter also may be affected, resulting in sphincter underactivity or overactivity and loss of sphincter coordination with bladder function. The appropriate therapy for neurogenic bladder and a successful treatment outcome are predicated upon an accurate diagnosis through a careful medical and voiding history, together with a variety of clinical examinations, including urodynamics and selective radiographic imaging studies.***

Keywords: neural disturbance of micturition, bladder dysfunction, intervention

1. Introduction

Spinal cord lesions are well known to cause neurogenic bladder dysfunction. [1] Significant association exists between the level of spinal cord lesion and its correlating bladder and sphincter behavior. Lesions above the spinal micturition center may lead to neurogenic detrusor overactivity (NDO) and detrusor-sphincter dyssynergia (DSD), inducing reflex micturition with increased detrusor leak point pressures, causing incontinence and consequent renal damage if untreated. [2,3] The role of urodynamic study in assessment and management of neurogenic bladder following myelopathies is well established. A number of studies in the past have reported the significance of such a practice to manage bladder effectively following non-traumatic myelopathies (NTMs) and to avoid complications in lower and upper urinary tract in the short- and long-term run. [4-7] The present study was conducted to observe detrusor behavior in patients following monophasic NTM (a spinal disorder/ lesion with a single phase) by performing urodynamic study (UDS) and to manage bladder accordingly. Another objective was to see if there is any correlation between detrusor behavior according to UDS and the level and severity of spinal cord lesion in these patients.

2. Material and Methods

We retrospectively reviewed the clinical data of 31 patients who had decompressive surgery for LDH and were diagnosed with CES between January 2015 and December 2019 at [1]Urology Department, University Hospital Centre "Mother Teresa", Tirana, Albania. All patients complained of urinary dysfunction by acute or chronic LDH but other symptoms due to LDH, such as motor weakness, radiating pain, rectal dysfunction, and sexual dysfunction were or were not presented in enrolled patients. Acute and chronic LDH were defined that any symptom except for urinary dysfunction such as radiating pain, weakness was presented in patients within less than6 weeks from onset of urinary dysfunction or more than 6 weeks from urinary dysfunction. An LDH below L2 level was included in this study and the improvement of initial symptoms after surgery were evaluated over a follow-up between 12-96 months. After surgery, patients had been classified into a normal bladder function group and an abnormal bladder function group, according to their bladder function improvement. Abnormal bladder function was defined as requiring clean intermittent catheterization because of urinary difficulty. We also assessed whether the time to operation from the onset of autonomic symptom, rectal dysfunction, sexual dysfunction (male only), main level of disc herniation and degree of spinal canal compression correlated with surgical outcome. The data used to evaluate the surgical outcomes were collected from clinical charts, out-patient department surveys and interview call. We defined the time to operation as the duration between the onset of urinary dysfunction and decompression surgery. Three categories were identified: less than 24 hours, 24-48 hours, and more than 48 hours. The symptoms were defined as follows: rectal dysfunction, as having difficulty defecating with decreased perianal sense; sexual dysfunction, as having difficulty achieving erection (in males only); level of herniated disc, as the level at which most severe spinal canal compression occurred; and degree of spinal canal compression, as the ratio of herniated disc anteroposterior (AP) diameter of the spinal canal to AP diameter of a normal spinal canal. The severity of urinary dysfunction at admission was categorized as mild (did not require Foley catheterization) and severe (required Foley catheterization). The data were statistically analyzed by MedCalc Statistical Software version 18.11.3 (MedCalc Software bvba, Ostend, Belgium) and the odds ratio determined using Pearson chi-square test.

3. Results

A total of 31 patients (mean age, 50.7 years; median, 51years; range, 19-83 years), comprising 26 male and 5 female patients were assessed during a 12- to 96-month follow-up. Fourteen patients were evaluated for outcome on within 3 years after operation. 10 patients were evaluated on 3-6 years after operation and 7 patients were evaluated on 6-

9 years after operation. A total of 8 patients (22.6%) were operated on within 24 hours. After decompression, 7 of these patients had normal bladder function and one had abnormal bladder function. Of the 23 patients with decompression after 24 hours, 12 had a normal bladder function outcome and 11 had an abnormal bladder function outcome. The proportion of patients with normal bladder function outcome was higher when decompression was performed within 24 hours compared to after 24 hours but there was not statistically significant difference. Of the 16 patients operated on within 48 hours, 13 and 3 patients had normal and abnormal bladder function outcome, respectively. Of the 15 patients with decompression after 48 hours, 6 and 9 had normal and abnormal bladder function outcome, respectively. Patients operated on within 48 hours compared to after 48 hours, had a significantly better outcome after decompression. The group operated on within 48 hours were 6.5 times more likely to have normal bladder function after decompression compared to the group with decompression after 48 hours (odds ratio, 6.5; 95% confidence interval, 1.27 to 33.03; Fisher exact test 2-sided, p=0.02). There were no complications, such as dural tears and infection, during the perioperative period.The degree of spinal canal compression, which ranged between 10% and 80%, was categorized into less than 50% (16 patients) and more than 50% (15 patients). After decompression, a respective 11 and 5 patients had normal and abnormalbladder function for patients with less than 50% compression. For patients with more than 50% compression, 8 and 7 patients had normal and abnormal bladder function, respectively, after decompression. The proportion of patients with normal bladder function outcome was higher in patients with less than 50% compression (68.7%) compared to more than 50% (53.3%). But there was no statistically significant difference (p=0.3) in the degree of spinal canal compression to the surgical outcome. There were 18 and 13 patients with and without rectal dysfunction, respectively. After decompression, respective 11 and 7 patients had normal and abnormal bladder function for patients with rectal dysfunction. For patients without rectal dysfunction, 8 and 5 patients had normal and abnormal bladder function, respectively, after decompression. The proportion of patients with normal bladder function outcome was almost same in patients with rectal dysfunction (61.1%) and without rectal dysfunction (61.5%). There was no statistically signifi cant difference (p=0.9) in the rectal dysfunction to the surgical outcome. There were eight and 18 male patients with and without sexual dysfunction, respectively. After decompression, respective 6 and 2 patients had normal and abnormal bladder function for patients with sexual dysfunction. For patients without sexual dysfunction, 12 and 6 patients had normal and abnormal bladder function, respectively, after decompression. The proportion of patients with normal bladder function outcome was higher in patients with sexual dysfunction (75.0 %) compared to patients without sexual dysfunction (66.6%). But there was no statistically significant difference (p=0.6) in the sexual dysfunction to the surgical outcome. There were 11 and 20 patients with L234 LDH and L45S1 LDH, respectively. After decompression, respective 4 and 7 patients had normal and abnormal bladder function for patients with L234 LDH. For patients with L45S1, 13 and 7 patients had normal and abnormal bladder function, respectively, after decompression. The proportion of patients with normal bladder function outcome was higher in patients with L45S1 LDH(65.0%) compared to patients with L234 LDH(36.3%). But there was no statistically significant difference (p=0.1) in the main level of LDH to the surgical outcome. Of the 21 patients with mild bladder dysfunction at admission, a respective 16 and 5 had normal and abnormal bladder function after decompression. Among the 10 patients with severe bladder dysfunction at admission, 3 and 7 had normal and abnormal bladder function after decompression. The proportion of patients with normal bladder function outcome was higher about 2.5 times in patients with mild bladder dysfunction at admission (76.0%) compared to patients with severe bladder dysfunction (30.0%). There was a statistically significant difference (p=0.02) in the severity of bladder dysfunction at admission to the surgical outcome . Consequently decompression resulted in a significantly impro ved outcome and there is a statistically significant difference in this study for patients on operated within 48 hours and with mild bladder dysfunction at admission.

4. Discussion

CES can occur due to LDH, tumor, or epidural abscess, and so on. [8] But CES is a rare and severe condition and is considered the absolute surgical indication. [9] The pathological CES process is debated but the mechanism of injury to the nerves in CES is suggested to be due to the combination of mechanical pressure, arterial ischemia, and venous congestion3). [10] Therefore, decompressive surgery is essential to improve the disability of this condition. The timing of surgical intervention for CES by decompression is debatable. Researchers demonstrated that treating all patients with CES within 48 hours of autonomic symptoms led to significant improvement in sensory and motor deficits, and urinary and rectal function. [11] However, reseachers suggested that a significant improvement in outcome was seen with within 24 hours. [12] Interestingly, one prospective study analyzed 33 patients undergoing surgery for CES due to LDH7), and found no statistical significance between timing of surgery (less than 24 hours, 24-48 hours, more than 48 hours) but a better outcome after decompression was found for patients who were continent of urine at onset of symptom than for patients who were incontinent of urine. This result supports the theory that the severity of bladder dysfunction at the onset of symptom may be a factor determining the outcome after decompression. Our study shows that operating within 48 hours on CES patients with bladder dysfunction caused by LDH can results in a significantly improved postsurgical outcome (6.5 fold) compared to decompression after 48 hours. Although the proportion of patients presenting a better postsurgery when operated on within 24 hours was higher than the patients with decompression after 24 hours, it was not statistical significant difference because p-value is 0.1. Furthermore, patients with mild bladder dysfunction at admission had an improved outcome after operation compared with patients with severe bladder dysfunction. But our study did not show that other factors, level of LDH, degree of spinal canal compression, sexual dysfunction, rectal dysfunction, are related to outcome. There was a little improved outcome after operation in patients with sexual dysfunction, lower level of LDH(L45S1), and less than 50%

spinal canal compression but there was no statistically significant difference ($p>0.05$). And there was no difference of outcome between with and without rectal dysfunction. There were several limitations to this study. This study is small, total 31 cases. It was a retrospective study; recent information regarding the surgical outcome was obtained by phone interview but most data were collected from medical records, including time to operation and severity of symptoms. In addition, because most patients with urinary dysfunction were operated on immediately or one day after arrival, the evaluation of bladder function was assessed by objective tests, such as uroflowmetry and postvoid residual urine scans were only performed in some patients. Furthermore, and in contrast to the objective evaluation, the outcome of bladder dysfunction after surgery was evaluated subjectively through a statement and survey. Follow-up period also was various from 12 months to 96 months. Longer follow-up period can result in confused outcome after operation because of other disease affecting the outcome such as benign prostatic hyperplasia, urethral stricture. If follow-up period for patients is equal, more reliable result can be showed.

5. Conclusion

Our study suggests that decompression surgery should be performed for patients with a neurogenic bladder within 48 hours from the onset of bladder dysfunction to improve bladder dys function after decompression. In addition, patients with mild bladder dysfunction at admission showed a significantly improved outcome after decompression.

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