Original Article

Urodynamic Study Findings Prior to Myelomeningocele Repair in Neonates

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Abstract

Introduction: Myelomeningocele (MMC) is the most common cause of neurogenic bladder dysfunction in children. Neurogenic bladder dysfunction is developed before birth due to autonomous nervous system affected prenatally in patients with MMC. The aim of this study was to share urodynamic study findings before MMC repair and to discuss the correlation with neurological evaluation. **Materials and Methods:** We prospectively studied 37 patients who underwent surgery for MMC repair in our institution in the first 20 h of their lives between 2013 and 2016. All patients were evaluated by a neurosurgeon, neonatologist, and pediatric surgeon. Urodynamic study was performed in first 18 h of life before MMC repair in all patients. Lesion level, occurrence of hydrocephalus, neurological functions, spinal deformities, and urodynamic study results were analyzed. **Results:** The study included 18 female and 19 male patients. Overactive detrusor was detected in 22 patients, and hypoactive detrusor was detected in 5 patients. Overactive sphincter muscle was detected in 32 patients, and hypoactive sphincter was detected in 2 patients. Detrusor–sphincter dyssynergia was present in 34 patients. **Conclusion:** Detailed analysis of urodynamic study findings in larger patient groups may be important to understand the physiopathology of prenatal damage in patients with MMC.

Keywords: *Detrusor–sphincter dyssynergia, myelomeningocele, neurogenic bladder dysfunction, overactive detrusor, urodynamic study*

Introduction

Myelomeningocele (MMC) is the most common cause of neurogenic bladder dysfunction (NBD) in children.[1] NBD is developed before birth due to autonomous nervous system, which is affected prenatally in patients with MMC. Although upper urinary tract is not affected in most patients, vesicoureteric reflux, progression of reflux, and formation of renal scars may occur due to NBD in early childhood.[2] Urodynamic study is the most effective method to evaluate and follow-up bladder functions in patients with MMC, but timing and type of urodynamic studies are still controversial.[3]

Although many literature about the evaluation of NBD in patients with MMC are available, lesion levels, associated spinal deformities, occurrence of hydrocephalus, and neurological examination of patients are not clearly mentioned.[1,4,5] The aim of this study was to share urodynamic study findings before MMC repair and to discuss the correlation with neurological evaluation.

Materials and Methods

We prospectively studied 37 patients who underwent surgery for MMC repair in our institution in the first 20 h of their lives between 2013 and 2016. Our study included both the newborns delivered at our institution and those referred immediately after birth for the management of MMC. Patients with thoracolumbar and lumbar lesions were included in the study. Patients

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with sacral lesions were not included. All patients were evaluated by a neurosurgeon, neonatologist, and pediatric surgeon. Urodynamic study was performed in the first 18 h of life before MMC repair in all patients. Lesion level, occurrence of hydrocephalus, neurological functions, spinal deformities, and urodynamic study results were analyzed. Urodynamic study consists of cystometry and sphincter electromyography (EMG). Urodynamic study parameters were functional bladder capacity, leak point pressure (maximum detrusor pressure), compliance, post-micturition residue, detrusor–sphincter activity, and EMG.

All the patients were prenatally diagnosed with MMC, and they underwent cesarean section for delivery. No patients had been diagnosed with hydronephrosis on the prenatal studies. All urodynamic studies were performed by the same team, which consists of two experienced nurses and a pediatric surgeon. The urodynamic study technique was same as described in the literature.[6]

Results

The study included 18 female and 19 male patients. The level of lesions was lumbar in 21 patients and thoracolumbar in 16 patients. Hydrocephalus was present in 20 patients at the time of delivery. Spinal deformities were diagnosed in 12 patients (lumbar kyphosis in five, thoracolumbar kyphosis in three, thoracolumbar scoliosis in one, thoracolumbar kyphoscoliosis in one, and decreased lumbar lordosis in two). Thirteen patients had paraparesis and 11 had paraplegia in neurological evaluation. Thirteen patients had normal neurological examination [Table 1]. The mean leak point pressure was 64.4 cm H_2O (±28.68). The mean functional bladder capacity was 25 mL (±15.05). Detrusor muscle activity was normal in 10 patients. Overactive detrusor was detected in 22 patients, and hypoactive detrusor was detected in 5 patients. Sphincter muscle activity was normal in three patients. Overactive sphincter muscle was detected in 32 patients, and hypoactive sphincter was detected in 2 patients. Detrusor–sphincter dyssynergia (DSD) was present in 34 patients. Compliance was normal in 36 patients and was decreased in 1 patient. The mean post-micturition residue volume was 11.108 mL (±13.93) [Table 1].

Discussion

Micturition requires a coordinated activation and deactivation of somatic and autonomic reflex pathways. Pontine and suprapontine centers provide this coordination in terms of inhibiting somatic and sympathetic guarding reflexes before attempted micturition.[7]

Lesions of sacral spinal cord or the spinal pathways to the higher centers may cause NBD. NBD may cause deficiency in the storage of urine or voiding or any combination. Three categories of lower urinary tract dynamics have been defined according to detrusor muscle contractility and external sphincter function during voiding: dyssynergic, synergic, and completely denervated.[8]

NBD and nocturnal enuresis are most often confronted bladder dysfunction in infants and children. Pathophysiology of bladder dysfunction in many aspects is still unclear. The dynamic feature

Table 1: Patients and findings

Patient No	Lesion Level	Hydrocephalus	Spine Deformity	Neurologic Evaluation	Functional Bladder Capacity (ml)	LPP (cm H2O)	Compliance	PMR	Detrusor	Sphincter	EMG
1	Lumbar	None	None	Paraplegia	60	60	Normal	37	Normal	Overactive	Dyssynergic
2	Thoracolumbar	Yes	Lumbar kyphosis	Paraplegia	18	14	Normal	10	Hypoactive	Hypoactive	Dyssynergic
3	Thoracolumbar	Yes	None	Paraplegia	40	36	Normal	30	Normal	Overactive	Dyssynergic
4	Thoracolumbar	Yes	None	Paraparesis	17	100	Decreased	8	Overactive	Overactive	Dyssynergic
5	Thoracolumbar	Yes	Decreased Lumbar Lordosis	Normal	24	85	Normal	3	Normal	Overactive	Dyssynergic
6	Lumbar	Yes	Lumbar kyphosis	Paraparesis	15	85	Normal	0	Overactive	Overactive	Dyssynergic
7	Lumbar	None	None	Normal	28	60	Normal	15	Overactive	Overactive	Dyssynergic
8	Lumbar	Yes	None	Paraparesis	13	90	Normal	5	Overactive	Overactive	Dyssynergic
9	Lumbar	None	None	Normal	10	30	Normal	0	Overactive	Overactive	Dyssynergic
10	Lumbar	None	None	Normal	21	80	Normal	0	Overactive	Overactive	Dyssynergic
11	Thoracolumbar	Yes	Thoracolumbar kyphosis	Paraplegia	14	60	Normal	0	Hypoactive	Overactive	Dyssynergic
12	Thoracolumbar	Yes	Thoracolumbar kyphosis	Paraplegia	11	25	Normal	6	Overactive	Overactive	Dyssynergic
13	Lumbar	None	None	Normal	17	60	Normal	0	Overactive	Normal	Synergic
14	Lumbar	None	None	Normal	23	40	Normal	0	Overactive	Overactive	Dyssynergic
15	Thoracolumbar	Yes	Thoracolumbar kyphosis	Paraparesis	12	100	Normal	0	Overactive	Overactive	Dyssynergic
16	Lumbar	Yes	None	Paraparesis	12	53	Normal	2	Overactive	Overactive	Dyssynergic
17	Lumbar	None	None	Normal	29	100	Normal	0	Normal	Normal	Synergic
18	Lumbar	None	None	Normal	13	70	Normal	0	Overactive	Overactive	Dyssynergic
19	Thoracolumbar	Yes	None	Paraparesis	33	90	Normal	15	Normal	Overactive	Dyssynergic
20	Thoracolumbar	Yes	Decreased Lumbar Lordosis	Paraparesis	59	35	Normal	15	Hypoactive	Overactive	Dyssynergic
21	Lumbar	None	None	Normal	21	80	Normal	9	Overactive	Overactive	Dyssynergic
22	Lumbar	None	None	Normal	23	17	Normal	0	Overactive	Normal	Synergic
23	Thoracolumbar	Yes	None	Paraparesis	30	38	Normal	25	Hypoactive	Overactive	Dyssynergic
24	Thoracolumbar	Yes	Thoracolumbar scoliosis	Paraplegia	13	17	Normal	5	Hypoactive	Hypoactive	Dyssynergic
25	Thoracolumbar	Yes	Lumbar kyphosis	Paraplegia	13	56	Normal	10	Overactive	Overactive	Dyssynergic
26	Lumbar	Yes	None	Paraplegia	45	75	Normal	38	Normal	Overactive	Dyssynergic
27	Lumbar	None	None	Paraparesis	10	55	Normal	0	Overactive	Overactive	Dyssynergic
28	Thoracolumbar	Yes	None	Paraparesis	55	110	Normal	45	Overactive	Overactive	Dyssynergic
29	Thoracolumbar	None	Lumbar kyphosis	Paraplegia	41	70	Normal	20	Normal	Overactive	Dyssynergic
30	Lumbar	None	None	Normal	18	100	Normal	0	Overactive	Overactive	Dyssynergic
31	Lumbar	None	None	Normal	32	75	Normal	20	Normal	Overactive	Dyssynergic
32	Thoracolumbar	Yes	Thoracolumbar kyphoscoliosis	Paraplegia	17	30	Normal	15	Overactive	Overactive	Dyssynergic
33	Lumbar	None	None	Paraparesis	32	130	Normal	13	Normal	Overactive	Dyssynergic
34	Lumbar	Yes	None	Paraparesis	19	60	Normal	7	Overactive	Overactive	Dyssynergic
35	Lumbar	None	None	Paraparesis	8	74	Normal	0	Overactive	Overactive	Dyssynergic
36	Lumbar	None	None	Normal	61	60	Normal	53	Normal	Overactive	Dyssynergic
37	Thoracolumbar	Yes	Lumbar kyphosis	Paraplegia	18	60	Normal	5	Overactive	Overactive	Dyssynergic

LPP = leak point pressure, EMG = electromyography

of lower urinary tract is complex as mature bladder function takes place with the growth of the child. In addition, physical growth of the sphincter unit differs from gaining neurological control over the bladder.[9] Urodynamic studies provide a better evaluation of the pathophysiological processes of bladder dysfunction.[10]

Previously, it was thought that voiding was inducted by a particular bladder volume, complete and without effect of the cerebral structures, and thus, independent of sleep, consciousness, or any other disturbing event. In the last decade, reports showed us that the brain is implicated in the modulation of bladder function in neonatal period. Small infants wake up to void when asleep. That does not mean voiding is voluntary or conscious, but the voiding reflex pathway connection to the cerebral cortex is developed anatomically, although function is immature and the signal only disturbs the neonate. Another example of neonatal cortical connections of the voiding reflex is that infants stop voiding when they are disturbed.[11]

Normal detrusor function contributes to bladder filling with little or no change in pressure. Involuntary contractions do not appear despite provocation. Detrusor overactivity is characterized by involuntary detrusor contractions during the filling phase, which may be spontaneous or provoked.[12] Isolated detrusor overactivity may be a result of suprapontine lesions. Detrusor overactivity was present in 22 patients (59.4%), and it was associated with DSD in 21 patients. Detrusor function was normal in 10 patients (27%), and 9 of them also had DSD. Detrusor hypoactivity was diagnosed in five patients (13.5%). Although hypoactive detrusor is expected to be seen with the injury of sacral levels, lesions of our patients with hypoactive detrusor were in thoracolumbar levels. In addition, hypoactive detrusor was associated with overactive sphincter muscle in three patients. Spina bifida causes abnormal development of corticospinal tracts.[13] There are some hypothesis about neurophysiological pathways that coordinate contractions of detrusor muscle and external sphincter. But the influence of these pathways is unclear in patients with MMC.[3] These findings show that pathophysiology of spinal injury in patients with MMC is more complex because of abnormal embryology and unpredictable intensity of intrauterine damage.

DSD is defined as the presence of an involuntary contraction of the external urethral sphincter during an involuntary detrusor contraction. DSD has been originally understood as a result of unconditional execution and coordination of the somatic guarding reflex by the external urethral sphincter, resulting from a loss of input from the pontine micturition center. DSD leads to several complications, which increase morbidity in patients with MMC. Poor bladder emptying and high bladder pressures can cause recurrent urinary tract infections, structural bladder damage, and vesicoureteric reflux, which finally causes hydronephrosis and renal failure.[5,14] DSD was present in 34 patients (92%) at the time of birth in our series. Damage of spinal cord in affected levels may vary in patients with MMC.[5] In addition, several supratentorial abnormalities, including hydrocephalus, exist in this group of patients. Spine deformities are another important condition, which deteriorates the clinical outcome. Hydrocephalus was detected in 20 patients (54%), and spine deformities were defined in 25 patients (67.5%) in our series. Occurrence of many abnormalities makes the pathophysiology and management of NBD more sophisticated in patients with MMC. Although impairment of bladder compliance is a component of NBD in the following years, it was detected in only one patient (2.7%) in our series.

Conclusion

Urodynamic study findings are important in patients with MMC because of not only their predictive value for kidney diseases in following years but also changes in them can be an early sign for tethered cord, which is a real challenge in both prenatally and postnatally operated patients with MMC. Underlying pathological processes of NBD in patients with MMC are still unclear. Detailed analysis of urodynamic study findings in larger patient groups may be important to understand the physiopathology of prenatal damage in patients with MMC.

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Conflicts of interest

There are no conflicts of interest.

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