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Micturition disturbance in acute transverse myelitis

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In ten patients with acute transverse myelitis (ATM), seven patients had urinary retention, and the other three patients had difficulty in voiding within 1 month from the onset of the disease. Five of the patients with retention became able to urinate. After the mean follow-up period of 40 months, nine still had urinary symptoms including difficulty in voiding in five and urinary frequency, urgency and incontinence in four patients. Four patients had urinary disturbance as the sole sequel of ATM. Urodynamic studies performed on nine patients revealed that all of the three patients with the urgent incontinence had detrusor hyperreflexia, all of the four patients with retention had an areflexic cystometrogram as well as sphincter hyperreflexia, and three of five patients with voiding difficulty had detrusor-sphincter dyssynergia. An areflexic cystometrogram tended to change to a low compliance bladder, followed by detrusor hyperreflexia or a normal cystometrogram. Analysis of the motor unit potentials of the external sphincter revealed that two of the three patients had high amplitude or polyphasic neurogenic changes. Supranuclear as well as nuclear types of parasympathetic and somatic nerve dysfunctions seemed to be responsible for micturition disturbance in our patients with ATM.

Keywords: acute transverse myelitis; micturition disturbance; urodynamic study

Introduction

Acute transverse myelitis (ATM) is a rare but well documented condition.[1–4] It usually develops rapidly with paraparesis and sensory disturbance with a nonprogressive course stabilizing within 2 weeks, and thereafter recovery ensues with some residual neurological deficits. ATM is recognized as a heterogeneous entity[3], although occurrence after vaccination or exanthematous infections[5] and serological[2] or pathological studies[4] suggested a parainfectious origin. Radiological investigations such as myelography and magnetic resonance imaging (MRI) enable us to rule out arteriovenous malformation, tumor and other causes of spinal cord compression from ATM. Although many patients with ATM have micturition disturbance, there are only few reports[6] about the urodynamic findings to our knowledge, although urinary incontinence or urinary retention has been described occasionally.[4,5] We report the results of micturitional disturbances and of urodynamic studies in ten patients with ATM.

Patients and methods

This is a retrospective study, reviewing the records of ten patients diagnosed as having ATM seen in our department during the past 8 years. There were six male and four female patients. The mean age was 38 years ranging from 15–57 years. Patients with spinal cord compression were excluded from the study. Patients with recurrent episodes or with encephalitic signs suggesting multiple sclerosis or acute disseminated encephalomyelitis were also exlcuded. The clinical features are summarized in Table 1. All patients had paraparesis which was mild in two, moderate in four and severe in four. The upper limit of disturbed sensation was cervical in two and thoracic in eight. Six patients had a mild mononuclear pleocytosis and increased protein content in the cerebrospinal fluid. Spinal MRI studies disclosed, in one patient, mild swelling of the cord. Seven patients underwent oral or intravenous steroid therapy. One patient was placed on the α-adrenoreceptor blocking agent phenoxybenzamine, which was discontinued before the urodynamic studies were started. We followed up these patients, with a mean interval of 40 months, ranging from 3–123 months. No male patient had apparent prostate hypertrophy on rectal examination.

Histories of micturition symptoms were taken, being divided into irritative and obstructive. Irritative symptoms consisted of nocturnal or diurnal urinary frequency, sensation of urgency, urinary incontinence and enuresis. Obstructive symptoms consisted of difficulty of voiding (including urinary hesitation and prolongation) and urinary retention. The results of the findings of the neurological and micturition status after the follow-up period are summarized in Table 2.

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Urodynamic studies consisted of the measurement of the residual urine, urethral pressure profilometry, water cystometry and sphincter electromyography. Three patients underwent the study repeatedly. The methods have been described previously.[7] All patients provided informed consent before we carried out the urodynamic studies.

Results

Micturition histories (Tables 1 and 2)

Within 1 month from the onset of the disease, seven patients (70%) (cases 1–6, 10) were found to have urinary retention. Three other patients (cases 7–9) had difficutly in voiding, and patient number eight also had nocturnal and diurnal urinary frequency, urgency, enuresis and urgencywith urinary incontinence (Table 1). During the follow-up period of 3–123 months, five of seven patients with retention became able to urinate. Five of them had difficulty in voiding, and four developed irritative symptoms including nocturnal urinary frequency in four, diurnal frequency in four, urgency in three, urgent incontinence in three, and enuresis in one patient (Table 2). The two other patients (patients two and three) had urinary retention within 3 months from the onset of the disease. Nine patients (90%) had urinary symptoms after the mean

Table 1 Patients and results of micturition histories in the acute stage

No.	*Age (years)*	*Sex*	*Spinal symptoms*					*Micturition symptons*						
			Paraparesis	*Reflex*	*Babinski sign*	**Sensory Level*	*Sacral Sparing*	*Noct*	*Poll*	*Urgency*	*Inco*	*En*	*Difficulty of voiding*	*Retention*
1	44	M	moderate	H	–	T10		–	–	–		–		+
2	48	M	mild	H	+	T7	+	–	–	–	–	–	–	+
3	57	F	severe	absent	+	T1	–	–	–	–	–	–	–	+
4	37	F	severe	absent	+	C6	–	–	–		–		–	+
5	16	M	moderate	N	+	T4	–	–	–	–	–	–	–	+
6	39	M	moderate	H	+	C7	–		–	–	–	–	–	+
7	40	F	moderate	H	+	T3	+	–	–	–	–	–	+	–
8	15	M	severe	H	+	T8	+	+	+	+	+	+	+	–
9	50	M	mild	H	+	T8	–	–	–	–	–	–	+	–
10	35	F	severe	H	+	T7	–	–	–	–	–	–	–	+

*upper limit of disturbed superficial sensation. H: hyperreflexia; N: normal; noct: nocturia; poll: pollakisuria; inco: incontinence; en: enuresis

Table 2 Follow up results of micturition status

No.	*Age (years)*	*Sex*	*Therapy*	*Follow-up periods (months)*	*Neurological status*	*Micturition status*
1	44	M		6	complete recovery other than urinary disturbance	able to urinate, but difficulty of voiding persisted and pollakisuria, nocturia appeared
2	48	M	*steroid pulse	3	complete recovery other than urinary disturbance	urinary retention unchanged and overflow incontinence appeared
3	57	F	dexamethasone, *steroid pulse, OHP	3	partial recovery	urinary retention unchanged
4	37	F	prednisolone	12	partial recovery	able to urinate, but difficulty of voiding persisted and pollakisuria, nocturia, urge incontinence appeared
5	16	M	prednisolone	5	partial recovery	able to urinate, but difficulty of voiding persisted
6	39	M	prednisolone POB	48	partial recovery	able to urinate, but difficulty of voiding persisted and pollakisuria, nocturia, urge incontinence appeared
7	40	F	prednisolone	8	partial recovery	difficulty of voiding disappeared
8	15	M		92	almost complete recovery other than urinary disturbance	difficulty of voiding improved, but pollakisuria, nocturia, urge incontinence and enuresis persisted
9	50	M		98	partial recovery	difficulty of voiding persisted
10	35	F	prednisolone	123	almost complete recovery other than urinary disturbance	able to urinate, but difficulty of voiding persisted and enuresis, pollakisuria, nocturia, urge incontinence appeared

steroid pulse: methylprednisolone 1g*3 succeeding days; OHP: oxygen under hyperbaric pressure; POB: phenoxybenzamine

follow-up period of 40 months. Urinary incontinence occurred in four patients in the overall follow-up periods, and this appeared to be more common in those with severe paraparesis (3 of 4; 75%), than in those with a mild or moderate paraparesis (1 of 6; 17%), although there was no statistical significance. Of four patients who had almost complete neurological recovery, all had only slight recovery of micturition. In the other six patients, three had only partial recovery of both their neurological state, and of micturition. One patient (case 7) had complete recovery of micturition.

Urodynamic studies (Table 3)
Urodynamic studies were performed in all of the patients except for one (case 6), and three (cases 1, 4 and 8) underwent the study repeatedly. Five patients underwent the study within 3 months from the onset and six patients after 5–125 months from the onset of the disease. Measurement of the residual urine was performed on seven patients, except for those with urinary retention, five had a residual urine of over 30 ml, with the mean volume of 78 ml, ranging from 30–200 ml. The volume of residual urine in three patients (case 1, 4 and 8) decreased along with the course of the disease. Urethral pressure profilometry was performed on eight patients. The maximum urethral closure pressure was decreased in three patients, and increased in one. Water cystometry was performed on all of the patients, and revealed that the bladder volume at the first desire to void and/or the maximum desire to void was decreased in three and increased in four patients. Five patients had an areflexic cystometrogram only within 2 months from the onset of the disease, which changed to low compliance at 3 months and then detrusor hyperreflexia at 5 months (case 4), or changed to normal at 2 months (case 1) from the onset. Four patients had detrusor hyperreflexia only after 5 months from the onset of the disease. External sphincter electromyography was performed on all of the patients, and revealed a brisk bulbocavernosus reflex in two, detrusor-sphincter dyssynergia (overactive sphincter on voiding) in four and sphincter hyperreflexia (overactive sphincter with areflexic bladder) in four patients. Analysis of the motor unit potentials of the external urethral sphincter was performed on three patients, and two patients (cases, 9 and 10) had high amplitude or polyphasic neurogenic changes. All of the four patients with urinary retention (cases 1–4) had an areflexic cystometrogram and sphincter hyperreflexia, and three of the five with difficulty of voiding had detrusor-sphincter dyssynergia. All of the three patients with urgent urinary incontinence (cases 4, 8 and 10) had detrusor hyperreflexia.

Discussion

The present study revealed ten patients had urinary symptoms, and seven of them (70%) had urinary retention in the acute stage. The incidence of micturition disturbance in our patients with ATM is very common, which may be attributable to its transverse nature. All of the patients with retention became able to urinate after 4 months from the onset of the disease. Urodynamic studies revealed that five patients with urinary retention had an areflexic cystometrogram at 1–2 months, which changed to a low compliance bladder at 3 months and then to detrusor hyperreflexia at 5 months, or changed to normal at 2 months from the onset in the follow-up studies. Berger *et al*[6] also found an areflexic cystometrogram at 3 weeks, which changed to detrusor hyperreflexia at 10 weeks from the onset in a patient with ATM. These results indicate that urinary retention and an areflexic cystometrogram in the acute stage of ATM could be considered as the 'spinal shock phase' of spinal cord injury. Four of our patients also had sphincter hyperreflexia associated with an areflexic cystometrogram. These evacuating disorders in the acute stage gradually improved. Seven patients in the chronic stage had difficulty in voiding, and four also had nocturnal urinary frequency, diurnal frequency in four, urgency in three, and enuresis in one patient; altogether nine patients (90%) had urinary symptoms after the mean follow-up period of 40 months. In the chronic stage four patients had detrusor hyperreflexia and four had detrusor-sphincter dyssynergia, indicating the major causes of storage as well as evacuating disorders. Berger *et al*[6] also reported that all of seven patients had detrusor hyperreflexia, and four of six had detrusor sphincter dyssynergia. Five of our patients obtained only partial recovery of both their neurological and micturition states. The other four patients recovered almost completely regarding their neurological status, but only slightly in their micturition status, indicating that micturition disturbance is the sole sequel of ATM. Urinary incontinence was noted in only four patients in the overall follow-up periods, and seemed to be more common in patients with severe paraparesis (three of four; 75%) than in those with mild or moderate paraparesis (one of six; 17%) although there was no statistical significance. The finding indicates that urinary incontinence seems to be related to the pyramidal tract involvement in ATM.

Urodynamic findings in ATM can also indicate the extent of the lesion concerning micturition disturbance. Detrusor hyperreflexia and detrusor-sphincter dyssynergia indicate supranuclear types of pelvic and pudendal nerve dysfunction,[8,9] corresponding to the transverse lesion of the spinal cord above the lumbosacral area. Analysis of the motor unit potentials of the external sphincter disclosed high amplitude or polyphasic neurogenic changes in two of three patients. This finding indicates the presence of sacral cord involvement.[10] Ropper *et al*[4] noted lymphocytic infiltration and demyelination from the midcervical to the lumbosacral regions to a variable extent in the postmortem study of a patient with ATM who had a T3 sensory level, indicating that subclinical

Table 3 Results of urodynamic studies

No. age sex (years)	*Duration* (months)*	*Urinary symptoms*	*Residual urine (ml)*	*UPmax (cmH_2O)*	*FDV (ml)*	*MDV (ml)*	*Detrusor hyperreflexia*	*Low compliance*	*Areflexic*	*Brisk BCR*	*DSD*	*Sphincter hyperreflexia*	*Motor unit potentials*
1 44 M	1	Retention	np	54	100	500	–	–	+	–	–	+	np
	2	Difficulty of voiding	40	np	250	450	–	–	–	–	–	–	np
	5	Difficulty of voiding, pollakisuria, nocturia	50	73	400↑	600↑	–	–	–	–	–	–	np
	6	Difficulty of voiding, pollakisuria, nocturia	0	56	290	480	–	–	–	–	–	–	np
2 48 M	1	Retention, overflow incontinence	np	> 100↑	194	> 600↑	–	–	+	–	–	+	normal
3 57 F	1	Retention	np	54	160	510	–	–	+	–	–	+	np
4 37 F	2	Rention	np	np	130	500	–	–	+	–	–	+	np
	3	Difficulty of voiding	190	37↓	450↑	500	–	–	–	–	+	–	np
	5	Difficulty of voiding, pollakisuria, urgent incontinence	200	33↓	200	270	+	–	–	np	np	np	np
	9	Difficulty, nocturia, pollakisuria, urgent incontinence	60	37↓	120	150↓	+	–	–	–	+	–	np
5 16 M	2	Difficulty of voiding	50	57	160	440	–	–	+	–	–	–	np
7 40 F	7	Normal	0	33↓	230	600↑	–	–	–	–	+	–	np
8 15 M	85	Noctura, pollakisuria, urgent incontinence, enuresis	30	47	30↓	30↓	+	–	–	+	–	–	np
	92	Pollakisuria, urgent incontinence, enuresis	0	37↓	50↓	50↓	+	–	–	+	–	–	np
9 50 M	96	Difficulty of voiding	70	np	170	229	+	–	–	+	+	–	polyphasic
10 35 F	120	Difficulty, nocturia, pollakisuria, urgent incontinence	0	78	80↓	200	+	–	–	–	+	–	high amplitude

*: duration of the disease before urodynamic study. Downward-pointing arrows indicate a value below normal range, and upward-pointing arrows indicate a value over normal range. np: not performed; UPmax: maximum urethral closure pressure; FDV: first desire to void; MDV: maximum desire to void; BCR: bulbocavernosus reflex; DSD: detrusor-sphincter dyssynergia

damages of the Onuf nucleus[10] may also occur in ATM. The above findings indicate that supranuclear as well as nuclear types of pelvic and pudendal nerve dysfunction seem to be responsible for micturition disturbances in our patients with ATM.

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