Research article

Monitoring development of autonomic dysreflexia during urodynamic investigation in patients with spinal cord injury

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Objectives: We aimed to investigate whether autonomic dysreflexia (AD) develops during urodynamic investigation in patients with spinal cord injury (SCI) with neurological level below thoracic (T) 6 together with the frequency and related factors for AD development.

Study Design: Prospective study.

Methods: The systolic blood pressure (SBP), diastolic blood pressure (DBP) and heart rate (HR) of 51 SCI patients with a neurological level below T6 were measured and recorded at the beginning and every two minutes during the filling phase of the urodynamic study. The changes between the SBP, DBP and HR values at the beginning and end of the filling phase were analyzed.

Results: Autonomic dysreflexia developed only in one of the 51 patients included into the study. The BP of this patient increased from 105/76 mmHg to 145/102 mmHg and the HR dropped from 88 beats/minute (bpm) to 69 bpm together with development of the AD symptoms. The patient was a 47-year-old male with a neurological level at T8. A significant difference was found between the mean SBP and the mean DBP values at the beginning and end of the filling phase.

Conclusions: Although rare, AD can be seen during urodynamic investigation in patients with a neurological level below T6, especially when close to the T6 level. Therefore, we suggest that the patients with a neurological level below T6 and especially closer to T6 level should be followed-up in terms of development of AD. The clinicians should take into account the HR values in addition to the the SBP and DBP values at follow-ups.

Keywords: Spinal cord injury, Autonomic dysreflexia, Urodynamics, Blood pressure

Introduction

Autonomic dysreflexia (AD) is an acute symptom developing as a result of excessive and uncontrolled sympathetic activity increase in patients with spinal cord injury (SCI) at and above the neurological level thoracic (T) 6.¹ Although it is mildly symptomatic, AD has been rarely reported in patients with a neurological level below T6.²⁻⁴

Autonomic dysreflexia may cause symptoms and signs such as rapid increase of arterial blood pressure, bradycardia, tachycardia, headache, blurred vision, nasal congestion, nausea, piloerection, sweating and flushing over the lesion level, together with coldness

and pallor below the lesion level.¹ AD can be asymptomatic or severe enough to cause life-threatening complications.^{1,5-8}

One must take into account that a blood pressure (BP) measured as 120/80 mmHg could be high in SCI patients as their BP value at rest is usually 90/60 mmHg. Systolic blood pressure (SBP) can increase to 250–300 mmHg and diastolic blood pressure (DBP) to 200–220 mmHg during an AD episode.¹ While bradycardia is frequently seen during an AD episode, tachycardia or atrial and ventricular fibrillation can also be observed.^{1,9,10}

Any noxious stimulant acting below the lesion level can trigger an AD episode. Triggers that cause AD are most commonly of lower urinary tract origin. Bladder distention is responsible for 75% to 85% of AD episodes.^{1,11}

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In this study, we aimed to investigate whether AD develops during urodynamic investigation in SCI patients whose neurological level was below T6 together with the frequency and related factors for AD development.

Materials and methods

Patients

A total of 51 consecutive patients (aged between 18–65 years) with SCI at a single region and below the T6 neurological level and who were hospitalized for rehabilitation and has been sent to the urodynamics laboratory for lower urinary tract dysfunction were included into this study. Exclusion criteria were the presence of additional neurological disorder or history of cardiovascular or genitourinary system disorder.

The sex, age, duration of injury, the etiology of SCI and SCI level and class according to the American Spinal Cord Injury Association (ASIA) Impairment Scale (AIS) classification of the patients were recorded. AD and its symptoms were explained to the patients and they were monitored for these symptoms.

Systolic blood pressure, DBP and heart rate (HR) values were measured and recorded at the beginning of the filling phase and then every two minutes during the filling phase. BP and HR measurements were performed from the right arm with an automatic measurement device (Microlife BP-3BTO-A Blood Pressure Device, Microlife AG Swiss Corporation, Widnau, Switzerland). Each measurement took about 30 seconds. The changes between SBP, DBP and HR values at the beginning and end of the filling phase was used in the analysis. We instructed the patients not to move or talk during BP and HR measurements. Hospital ethical committee approval was obtained.

Urodynamic investigation

Urodynamic investigations were performed with the MMS Solar (MMS, Enschede, The Netherlands)

urodynamic measurement system by using 7 F sterile urethral double-lumen catheters and sterile physiological saline at room temperature at a 20 ml/min constant filling rate. The filling phase was terminated and the bladder capacity at that moment was recorded as the maximum cystometric capacity (MCC) when the patient expressed a strong need for urination, a significant leakage developed, the intravesical pressure exceeded 40 cmH₂O or AD developed during urodynamic investigations. The filling phase was ended in patients with a minimum increase of 20 mmHg in SBP after evaluating the symptoms, DBP and HR values. Detrusor activity, compliance and bladder filling sensation were evaluated and recorded from the urodynamics reports.

Statistical analysis

Statistical analyses were performed by using SPSS version 15 software (SPSS Inc., Chicago, IL, USA). Descriptive statistics were shown as mean $\pm$ standard deviation, range and medians for variables with continuous measurement while nominal and ordinal variables were shown as number of cases. The paired *t* test was used in the comparison of filling beginning and end SBP, DBP and HR values of the patients. A *P*-value below 0.05 was considered statistically significant.

Results

Demographic and clinical features of the patients in the study were presented in Table 1. Of the 51 patients, 12 were subacute and 39 chronic period SCI patients (0–1 months was evaluated as acute, 1–6 months subacute, and more than 6 months as chronic).

Mean SBP, DBP and HR at the beginning of filling phase was 117 $\pm$ 12 mmHg, 74 $\pm$ 9 mmHg and 76 $\pm$ 11 bpm respectively. Mean SBP, DBP and HR at the end of the filling phase was 121 $\pm$ 13 mmHg, 78 $\pm$ 11 mmHg, 75 $\pm$ 12 bpm respectively. Statistically

Table 1 Demographic and clinical features of the patients in the study (n = 51)

Mean age (years)	35.4 $\pm$ 12
Mean duration between the injury and the urodynamic investigation (months)	24 (1–123)
Sex (female/male)	9/42
Level of lesion (T/L)	37/14
T7/T8/T9/T10/T11/T12/L1/L2/L3	2/5/2/8/2/18/6/4/4
AIS class (A/B/C/D)	31/8/8/4
Etiologic factors	
Traffic accident/Fall/Being trapped under a weight/Firearm injury/Past surgery	16/26/5/3/1
Mean maximum cystometric capacity (ml)	444 $\pm$ 223
Hypoactive/Hyperactive detrusor	15/36
Hypocompliant detrusor (yes/no)	31/20
Bladder filling sensation	
Absent/partially preserved/fully preserved	20/28/3
Anticholinergic-antimuscarinic drug use (yes/no)	24/27

T: Thoracic, L: Lumbar, AIS: ASIA (American Spinal Cord Injury Association) Impairment Scale

Table 2 Comparison of the blood pressure and heart rate values at the beginning and end of the filling phase

	Beginning of the filling phase	End of the filling phase	P value
Systolic blood pressure (mmHg) (mean $\pm$ SD, range)	117 $\pm$ 12, 81–139	121 $\pm$ 13, 83–146	0.005
Diastolic blood pressure (mmHg) (mean $\pm$ SD, range)	74 $\pm$ 9, 48–91	78 $\pm$ 11, 47–102	0.001
Heart rate (beats/minute) (mean $\pm$ SD, range)	76 $\pm$ 11, 59–113	75 $\pm$ 12, 50–108	0.488

significant differences were detected between the mean SBP and DBP values at the beginning and end of the filling phase ($P = 0.005$, $P = 0.001$, respectively). However, there was no statistically significant difference between the mean HR values at the beginning and end of the filling phase ($P = 0.488$) (Table 2).

Autonomic dysreflexia developed only in one of the 51 patients included in the study. The BP of this patient increased from 105/76 mmHg to 145/102 mmHg and the HR dropped from 88 bpm to 69 bpm together with development of the AD symptoms. The patient was a 47-year-old male with a neurological level of T8 and AIS grade of C suffered the injury 51 months ago. The MCC was 508 mL with a hyperactive-hypocompliant detrusor where bladder-filling sensation was partially preserved and he was using an anticholinergic drug. There were two other patients with a BP increase of 20 mmHg from the baseline value. The SBP increased 22 mmHg and DBP increased 8 mm Hg while the HR decreased 1 bpm in one patient with a neurological level at T10. The DBP and HR did not change in other patient who had a neurological level at T12. However, AD symptoms were not observed in both of these two patients.

Discussion

To know in which patient group and with which stimulants AD will develop and its pathophysiology in SCI patients is an important issue in terms of preventing and treating this potentially life-threatening condition.

In the present study, we evaluated the patients with SCI below T6 neurological level in terms of the development of AD during urodynamic investigation. The SBP, DBP and HR values of the patients were examined for AD development during the filling phase and the patients were monitored for AD symptoms. Using an automatic BP device that can also record HR in our study provided ease of use as well as more objective results as it was independent of the individual who was performing the measurements.

Although a statistically significant increase was observed between the SBP and DBP values at the beginning and end of the filling phase in our study population, AD developed in only one of these patients. There was no significant change in HR value in the

study population. An increase of more than 20 mmHg in both systolic and diastolic blood pressure and a significant decrease in HR, and occurrence of AD symptoms were detected only in the patient who developed AD. We feel that the significant difference between the beginning and ending mean SBP and DBP is not surprising because the significant increase of SBP and DBP in the patient who developed AD and the ending mean SBP values of 2 patients with 20 mmHg or more increase in SBP increased the mean ending BP values. We think that the significant difference between the beginning and ending mean SBP and DBP observed in our study population may be an indicator of the occurrence of AD and possibly silent AD in SCI patients below T6 level. However new studies conducted in normal population in which SBP, DBP and HR monitoring carried out during urodynamic study will better clarify the changes in these parameters in urodynamic examinations.

Autonomic dysreflexia is a life-threatening complication characterized by a sympathetic discharge reflex resulting from the preganglionic neurons in the thoracolumbar spinal cord. It is caused by a deafferented spinal cord stimulated by a stimulus triggered mostly by the distention of visceral organs in the pelvic region in injuries over the T5–6 sympathetic splanchnic exit level of the spinal cord.¹²

Although AD is described as a symptom complex that develops as a response to a noxious stimulant below the lesion level and is characterized by bradycardia and sudden increase in BP,¹³ how much the increase in BP should be and whether a DBP increase is required together with SBP and whether HR changes should accompany the BP increase in order to identify BP elevation as AD are controversial. Some authors describe the SBP increasing more than 20–30 mmHg or 20–30% as a dysreflexic episode^{14–17} while other authors have reported that a decrease of 15% in the HR together with a 30% increase in SBP and DBP may indicate AD,¹⁶ that a BP exceeding 160/90 mmHg¹⁸ or 150/100 mmHg may be a finding of AD,¹⁹ and that a sudden increase in SBP and DBP of 20–40 mmHg and accompanying bradycardia may be a finding of AD.²⁰ According to the Consortium for Spinal Cord Medicine, an increase in BP of 20–40 mmHg can be a sign of AD.²¹

Bradycardia usually accompanies AD but some authors report that tachycardia is seen more commonly during an AD episode and there are even those who report that AD may induce tachycardia in those whose lesion level is at T1–5 as the sympathetic nerve innervation of the heart can be protected.³

Of the 51 patients included in our study, an increase of more than 20 mmHg in the DBP and bradycardia accompanying the SBP increase was observed only in 1 of the 3 patients in whom an increase of more than 20 mmHg in SBP was detected. AD symptoms were also observed only in this patient. While SBP increased 40 mmHg and DBP 26 mmHg in this patient, the HR decreased 19 bpm. This patient who developed AD was a 47-year-old male with a neurological level at T8 and AIS grade of C and disorder duration of 51 months. The MCC was 508 mL, with a hyperactive-hypocompliant detrusor and partially preserved bladder filling sensation. He was using an anticholinergic drug.

Autonomic dysreflexia frequency in patients with a neurological level at T6 and above varies between 19% and 20%.^{1,9,22} Although the AD episode in SCI patients with a high level and complete injury is more severe,¹⁶ AD has also been reported to develop in patients with a neurological level below T6.^{2–4} Evaluation of SCI patients with a neurological level below T6 who develop AD has shown that the neurological levels of these patients are closer to the T6 level in general.^{3,4} The AD course may be milder as the neurological level moves away from the T6 since the unopposed sympathetic discharge will decrease. When AD cases causing complications were investigated, these cases were found to be high neurological level SCI patients.^{5,7,8} AD cases causing life-threatening complications or death were investigated in a review published in 2014 and it was detected that the neurological level of all 32 cases was at T6 and above.⁶

The neurological level of the patient who developed AD in our study was T8. The neurological level of the patient whose SBP increased 22 mmHg and DBP increased 8 mmHg without the development of the AD symptoms was T10. The neurological level of the patient whose SBP increased 20 mmHg without a DBP increase or AD symptoms was T12. Although AD is more commonly seen in the chronic period, it can also be seen in the first few days or weeks after the injury.^{11,17,23,24} The duration of SCI in our patient who developed AD was 51 months.

The trigger in 75% to 85% of AD episodes is bladder distention.^{1,11} AD or SBP and DBP elevation and bradycardia are reported to develop in patients especially with a neurological level at T6 and above during

urodynamic studies.^{3,25–27} Therefore, the patients should be informed regarding AD, especially before interventional procedures such as urodynamic investigation, urethral catheter insertion and cystoscopy. BP should be followed up in this patient group. According to the results we obtained from our study and the results in the literature, autonomic dysreflexia, although rare, can be seen during urodynamic investigation in patients with a neurological level below T6, especially when close to the T6 level. Therefore, we suggest that the patients with a neurological level below T6 and especially closer to T6 level should be followed-up in terms of development of AD. The clinicians should take into account the HR values in addition to the SBP and DBP values at follow-ups.

Disclaimer statements

Contributors EK and ME: planned and coordinated the study, made the statistical analysis and wrote the manuscript. ME: supervised the study period and revised the manuscript. All of the study participants were evaluated by both of the authors. All urodynamic studies were conducted by the 1st and 2nd author.

Funding None.

Conflicts of interest The authors declare no conflict of interest.

Ethics approval The study was approved by our hospital's ethics committee.

References

- Blackmer J. Rehabilitation medicine: 1. Autonomic dysreflexia. *CMAJ* 2003;169(9):931–5.
- Moeller BA, Jr, Scheinberg D. Autonomic dysreflexia in injuries below the sixth thoracic segment. *JAMA* 1973;224(9):1295.
- Huang YH, Bih LI, Chen GD, Lin CC, Chen SL, Chen WW. Autonomic dysreflexia during urodynamic examinations in patients with suprasacral spinal cord injury. *Arch Phys Med Rehabil* 2011;92(9):1450–4.
- Gimovsky ML, Ojeda A, Ozaki R, Zerme S. Management of autonomic hyperreflexia associated with a low thoracic spinal cord lesion. *Am J Obstet Gynecol* 1985;153(2):223–4.
- Sumiya T. Hypertensive intracerebral hemorrhage due to autonomic dysreflexia in a young man with cervical cord injury. *J UOEH* 2013;35(2):159–64.
- Wan D, Krassioukov AV. Life-threatening outcomes associated with autonomic dysreflexia: a clinical review. *J Spinal Cord Med* 2014;37(1):2–10.
- Eker A, Yigitoglu PH, Ipekdağ HI, Tosun A. Acute onset of intracerebral hemorrhage due to autonomic dysreflexia. *J Korean Neurosurg Soc* 2014;55(5):277–9.
- Dolinak D, Balraj E. Autonomic dysreflexia and sudden death in people with traumatic spinal cord injury. *Am J Forensic Med Pathol* 2007;28(2):95–8.
- Grigorean VT, Sandu AM, Popescu M, Iacobini MA, Stoian R, Neascu C, et al. Cardiac dysfunctions following spinal cord injury. *J Med Life* 2009;2(2):133–45.
- Pine ZM, Miller SD, Alonso JA. Atrial fibrillation associated with autonomic dysreflexia. *Am J Phys Med Rehabil* 1991;70(5):271–3.

- 11 Lindan R, Joiner E, Freehafer AA, Hazel C. Incidence and clinical features of autonomic dysreflexia in patients with spinal cord injury. *Paraplegia* 1980;18(5):285–92.
- 12 Gondim FA, Lopes AC, Jr, Oliveira GR, Rodrigues CL, Leal PR, Santos AA, *et al.* Cardiovascular control after spinal cord injury. *Curr Vasc Pharmacol* 2004;2(1):71–9.
- 13 Teasell RW, Arnold JM, Krassioukov A, Delaney GA. Cardiovascular consequences of loss of supraspinal control of the sympathetic nervous system after spinal cord injury. *Arch Phys Med Rehabil* 2000;81(4):506–16.
- 14 Krassioukov A, Warburton DE, Teasell R, Eng JJ. Spinal Cord Injury Rehabilitation Evidence Research Team. a systematic review of the management of autonomic dysreflexia after spinal cord injury. *Arch Phys Med Rehabil* 2009;90(4):682–95.
- 15 Karlsson AK. Autonomic dysreflexia. *Spinal Cord* 1999;37(6):383–91.
- 16 Curt A, Nitsche B, Rodic B, Schurch B, Dietz V. Assessment of autonomic dysreflexia in patients with spinal cord injury. *J Neurol Neurosurg Psychiatry* 1997;62(5):473–7.
- 17 Silver JR. Early autonomic dysreflexia. *Spinal Cord* 2000;38(4):229–33.
- 18 Linsenmeyer TA, Campagnolo DI, Chou IH. Silent autonomic dysreflexia during voiding in men with spinal cord injuries. *J Urol* 1996;155(5):519–22.
- 19 Giannantoni A, Di Stasi SM, Scivoletto G, Mollo A, Silecchia A, Fuoco U, *et al.* Autonomic dysreflexia during urodynamics. *Spinal Cord* 1998;36(11):756–60.
- 20 Gunduz H, Binak DF. Autonomic dysreflexia: an important cardiovascular complication in spinal cord injury patients. *Cardiol J* 2012;19(2):215–9.
- 21 Consortium for Spinal Cord Medicine. Acute management of autonomic dysreflexia: individuals with spinal cord injury presenting to health-care facilities. *J Spinal Cord Med* 2002;25(1):67–88.
- 22 Colachis SC. Autonomic hyperreflexia with spinal cord injury. *J Am Paraplegia Soc* 1992;15(3):171–86.
- 23 Krassioukov AV, Furlan JC, Fehlings MG. Autonomic dysreflexia in acute spinal cord injury: an under-recognized clinical entity. *J Neurotrauma* 2003;20(8):707–16.
- 24 Krassioukov A, Biering-Sørensen CF, Donovan W, Kennelly M, Kirshblum S, Krogh K, *et al.* International Standards to document remaining Autonomic Function after Spinal Cord Injury (ISAFSCI), First Edition 2012. *Top Spinal Cord Inj Rehabil* 2012;18(3):282–96.
- 25 Krum H, Louis WJ, Brown DJ, Clarke SJ, Fleming JA, Howes LG. Cardiovascular and vasoactive hormone responses to bladder distension in spinal and normal man. *Paraplegia* 1992;30(5):348–54.
- 26 Wurster RD, Randall WC. Cardiovascular responses to bladder distension in patients with spinal transection. *Am J Physiol* 1975;228(4):1288–92.
- 27 Shergill IS, Arya M, Hamid R, Khastgir J, Patel HR, Shah PJ. The importance of autonomic dysreflexia to the urologist. *BJU Int* 2004;93(7):923–6.