

Case Report

Malignant autonomic dysreflexia in spinal cord injured men

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Study design: Case reports.

Objectives: To present a series of cases of protracted and severe autonomic dysreflexia (AD) in men with spinal cord injury (SCI), who sustained damage to their descending autonomic pathways.

Settings: GF Strong Rehabilitation Centre, Sexual Health Rehabilitation Service, Vancouver Sperm Retrieval Clinic, Vancouver Coastal Health Authority, Vancouver, BC, Canada.

Case report: AD is a serious complication of SCI triggered by a variety of noxious or non-noxious stimuli below the level of injury. However, we are presenting three cases of protracted, severe AD we have termed ‘malignant’, owing to the tendency of progressive worsening not usually seen with AD once the alleviating factor is removed. In all three individuals, AD was initially triggered by ejaculation and continued for a period of more than 1 week. Systolic blood pressure in these individuals increased above 220 mmHg and required either acute hospitalization or hospital assessment. Two of the individuals with malignant AD had American Spinal Injury Association (ASIA) B and C high cervical injury, respectively, with the third having a high thoracic ASIA A injury. In addition to detailed history and neurological examination, electrophysiological assessment of sympathetic skin responses (SSR) demonstrated a significant disruption of the descending autonomic pathways in these individuals.

Conclusions: Our findings suggest that in addition to the severe injury of the motor and sensory pathways (assessed by ASIA score), these individuals sustained severe injury to the supraspinal autonomic control. A combination of strong triggers such as ejaculation and bladder or colono-rectal irritation with total loss of descending autonomic control to the spinal sympathetic circuits could therefore contribute to the unusual manifestation of AD.

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Introduction

Autonomic dysreflexia (AD) is a clinical emergency that commonly occurs in individuals with spinal cord injury (SCI) at level T6 and above.^{1,2} An episode of AD is characterized by acute elevation of arterial blood pressure (BP) and bradycardia, although tachycardia may also occur. Symptomatically, patients can experience severe headache, profuse sweating and/or flushing and piloerection above the injury.³ Objectively, an increase in systolic BP greater than 20–30 mmHg is considered a dysreflexic episode.² Commonly, episodes

of AD could be triggered by urinary bladder or colon irritation.³ However, it is not unusual in sperm retrieval and urology clinics to see 100% increases in systolic and diastolic BP, respectively, during ejaculation or urology procedures.^{4,5} AD is caused by massive sympathetic discharge triggered by either noxious or non-noxious stimuli below the level of the SCI.^{3,6} Numerous reports of AD cases are cited in the literature: they are usually short-lived due to being treated or being self-limiting *per se*. However, there are a few reports of AD triggered by a specific stimulus, which then continued to be present for a period of days to weeks.⁷ The purpose of this paper is to describe the occurrence, clinical pathway, pathophysiology and sexual implications of prolonged AD triggered by ejaculation in three men with SCI.

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We present three cases of protracted, severe AD we have termed 'malignant', owing to the tendency of progressive worsening not usually seen with AD once the alleviating factor is removed. In each case, systolic arterial BP was not only increased to greater than 220 mmHg, but became protracted over days or weeks. All three individuals were patients who underwent rehabilitation at the GF Strong Rehabilitation Centre in Vancouver, BC and were clients of the Sexual Health Rehabilitation Service (SHRS). The unusual presentations of AD were described during routine history taking prior to sperm retrieval procedures at the Vancouver Sperm Retrieval Clinic (VSRC). In all three individuals, the malignant AD initially triggered was by ejaculation and continued for a period of at least 1 week.

In addition to demographic and medical data collection, neurological assessment using the American Spinal Injury Association (ASIA) score was conducted in all three individuals.⁸ The ASIA score is widely utilized for assessments of the completeness and the severity of SCI and correlates well with the electrophysiological and histopathological findings of integrity of the motor and sensory spinal pathways in individuals with SCI.⁹⁻¹¹ However, ASIA assessment does not evaluate the integrity of the spinal autonomic circuits.¹² To assess the latter, electrophysiological examination of sympathetic skin responses (SSR) were conducted on each individual in order to evaluate the preservation of the descending spinal autonomic pathways.^{13,14}

Case 1

This 30-year-old man with incomplete quadriplegia (C5 ASIA C) presented to SHRS following a motor vehicle accident 10 years earlier. At 3 years postinjury, he had presented to the VSRC for fertility assessment but had failed to ejaculate using vibrostimulation (VS). The resting and maximum BP at that visit were recorded as 121/65 and 148/83 mmHg, respectively. Under the direction of the VSRC, he continued the use of VS at home in order to achieve ejaculation. Approximately 1 year later, with the use of a sympathomimetic medication (Sudafed[®]), he was able to successfully ejaculate and experience orgasm, and denied experiencing any symptoms of AD. He then began to ejaculate during sexual intercourse and continued with this form of sexual activity without incidence of AD. He denied any history of headaches or migraines prior to the episode of malignant AD. The patient had also preserved sensation of bladder fullness, therefore, he voided on his own several times per day and only catheterized once at night.

However, approximately 5 years postinjury, following an ejaculation during sexual intercourse, this individual developed an unusually severe and prolonged episode of AD. Immediately following the ejaculation, he experienced symptoms of AD not typical for him: a severe pounding headache starting from the back of the neck, increased head pressure and sweating. These symptoms did not subside but became protracted. Furthermore, he began to experience severe episodes of AD with each

void, confining him to sitting or lying quietly for up to 30 min several times per day until the symptoms subsided. This unusually severe AD around voiding eventually resolved within the following 5 days. He denied having a urinary tract infection or any other obvious causes that could have exacerbated the episodes of AD at that time. The patient avoided sexual activities for a brief period following this episode for fear of AD recurrence.

The patient did not seek medical attention until several months later after he had resumed sexual intercourse and ejaculation without occurrence of AD. Imaging studies conducted at this time did not show any new changes within the brain or the spinal cord. Ultrasound showed normal upper urinary tracts. Multi-channel urodynamic studies demonstrated early first sensation at 44 cc and decreased bladder capacity of 85 cc, with development of a 54 cm H₂O bladder pressure and a high urethral pressure of 100 cm H₂O. Cystoscopy demonstrated a meatal stenosis, which was then dilated. It was speculated that sphincter spasm had triggered his earlier malignant episodes of AD.

Currently, this man experiences mild episodes of AD of variable duration approximately every third ejaculation. A recent ejaculation trial at the VSRC demonstrated a significant increase of BP to 214/136 mmHg at the time of ejaculation (resting BP 97/64 mmHg) accompanied by a moderate headache and sweating. The patient is not able to identify any predicting factors for the occurrence of AD with ejaculation in a private situation. However, he is no longer experiencing any symptoms of AD with voiding despite continued regular ejaculation.

Case 2

This 29-year-old with incomplete quadriplegia (C5 ASIA B) experienced his first episode of AD during his early rehabilitation period. Hospital chart records indicated increase in BP to 220/120 mmHg resulting from blockage of an indwelling Foley catheter. This episode of AD was resolved by bladder drainage when BP appropriately reduced to approximately 90/60 mmHg within 10 min. After discharge, he began a sexual relationship with a woman but he did not experience ejaculation or AD during their 3 years of sexual activity. However, he acknowledged frequent episodes of sweating and muscle tightening with voiding (bladder management was condom drainage) during this period.

This individual presented to the VSRC 4 years after injury with a new partner, for fertility assessment. His resting BP was 108/68 mmHg. At ejaculation using VS, his BP increased to 205/115 mmHg, and he experienced a moderate headache and muscle spasm. For the following 18 months, despite continued sexual activity with this partner, he was not able to ejaculate. He then experienced his first unexpected ejaculation with her, which was accompanied by 'unbearable' symptoms of AD. The AD gradually subsided over the following 30 min; however, any void in the following hours would precipitate severe symptomatic episodes of AD. Persis-

tence of these symptoms through the night resulted in admission to the emergency department the next morning. Emergency room examination did not reveal any changes in neurological status, and CT of the head was negative. Urology was consulted and antibiotic therapy was initiated for a suspected urinary tract infection. Despite ongoing antibiotic treatment and numerous admissions to the emergency department, the symptoms of AD persisted for more than a week before they subsided. After a period of abstinence during which he had feared recurrence of AD, the patient resumed sexual activity. Continued activity over the following months did not result in ejaculation, but did provoke AD symptoms. A few months later, cystoscopy examination revealed a high residual urine volume of 400 cc, as well as a grossly trabeculated inflamed bladder with numerous stones. The smaller stones were flushed and lithotripsy was executed on the remaining ones. The urology report noted a normal but spastic sphincter, a small prostate and the presence of small false prostatic urethral passages possibly related to previous stones.

At 6 years postinjury, the patient is now able to occasionally ejaculate and experience some orgasmic sensations with his partner during sexual activity, with only minimal AD symptoms. He attributes this diminished AD to improve genital sensation, conscious relaxation and an emotional connection to his partner. He has also returned to the VSRC for two VS trials. However, at ejaculation, while his subjective AD symptoms were minimal (muscle spasm with mild or no headache), his BP increased from a resting 93/54 and 82/47 mmHg to a high of 177/108 and 238/167 mmHg, respectively.

Case 3

This 23-year-old man sustained incomplete quadriplegia (T3 ASIA A) following a motor vehicle accident. During his early period of rehabilitation, mild AD accompanied only by sweating was noted with either voiding or difficult catheterization. Cystoscopy demonstrated a spastic sphincter and some inflammatory changes along the prostatic urethra. After discharge, he continued to experience erectile dysfunction and anejaculation during sexual activity, and denied any symptoms of AD. His bladder was managed with condom drainage.

By 2 years postinjury, his sensation and erection had improved and he ejaculated for the first time with self-stimulation. However, immediately following ejaculation, he developed a prolonged (1 h) severe, bilateral, throbbing headache described as 'unbearable'. Prior to this episode of headache, he denied any previous history of significant headaches or migraine. Despite this headache, he successfully ejaculated again within 12 h and again experienced another episode of severe headache that did not subside and required medical attention. He was examined at the emergency department and urinary tract infection and intracranial pathology was ruled out. During the following few days, he continued

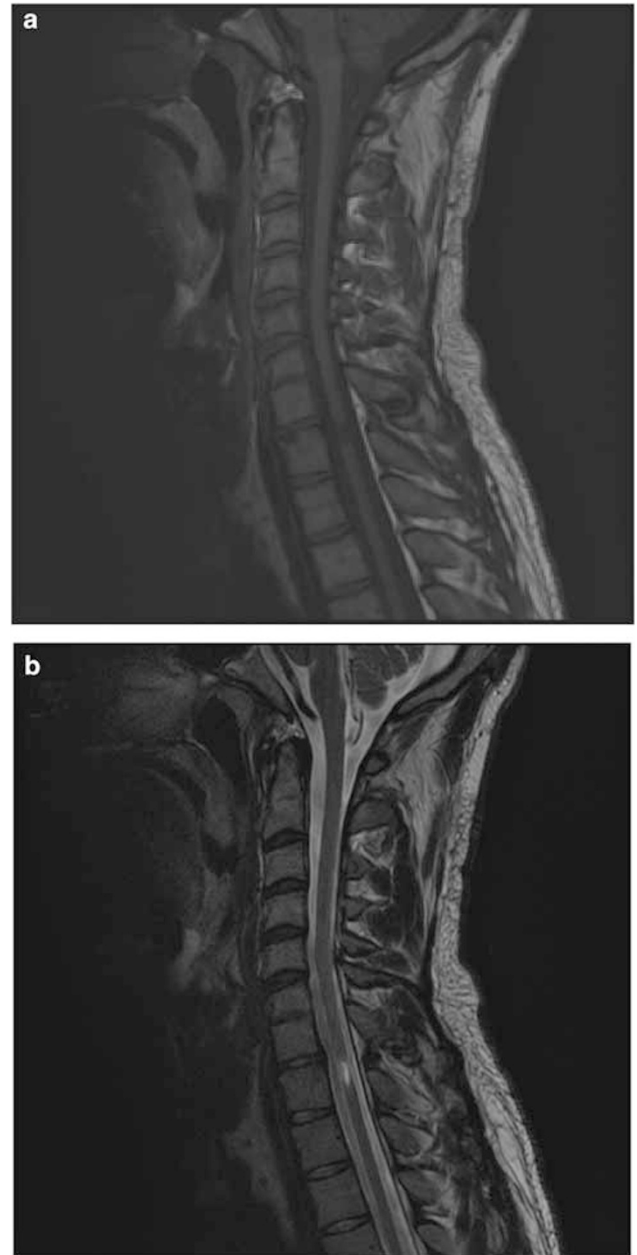


Figure 1 Magnetic resonance images (MRI) of the cervical spine of a 23-year-old male (Case 3, T3 ASIA A) T1 (a) and T2 (b) weighted sagittal views. A signal change within spinal cord is present in upper thoracic region

to experience a debilitating headache with every void or bowel movement. As a result of this condition, he became housebound. An indwelling catheter alleviated the symptoms, but over 2 weeks, attempts to return to condom drainage would result in the return of severe AD. During this 2-week period he was hospitalized four times in order to manage this unusually severe form of AD. MRI of the spine (Figure 1) conducted during one of these admissions did not reveal any changes within the spinal cord. On admission to the hospital, cystoscopy

examination revealed a spastic sphincter, inflammatory changes along the prostatic urethra and a normal bladder except for some trabeculation.

Currently, the patient continues to experience sweating during voiding and has more pronounced symptoms of AD during sexual intercourse with a partner. Although very sexually active, he has not yet experienced ejaculation with a partner. He continues to be able to ejaculate with masturbation and still experiences the symptoms of AD with ejaculation. During ejaculation he experiences tight spasm, a hot flush, and the 'wind being taken out of him', with loss of spasm for 15–30 min postejaculation. However, the prior use of Propranolol[®] (10 mg) and an upright position immediately following ejaculation have helped to reduce the severe headache. Despite continued self-stimulation to ejaculation, he has not experienced further episodes of malignant AD related to bladder or bowel function.

ASIA score and SSR

In all three individuals, SSRs were obtained by electrical stimulation of the median nerve and posterior tibial nerve. Recording electrodes were applied to the palmar and dorsal cervices of the hands and plantar and dorsal cervices of the foot bilaterally (Figure 2). SSRs were recorded simultaneously from both hands and feet over 8 s and sampled at a band pass of 3 Hz to 3 kHz. At least 10 recordings with stimulations (0.2 ms duration, 10–20 mA intensity) at each site were obtained. Finally, the latency and amplitude of SSRs were measured and compared in each case. The subjects were positioned in a temperature-controlled room and rested supine for at least 10 min before the beginning of the examination using a Powerlab 16 SP electrophysiological system (AD Instruments Inc., Colorado Springs, USA).

No SSRs were obtained in two individuals with cervical SCI (Cases 1 and 2) below the level of injury (Figure 2 and Table 1). Only inconsistent responses in the left arm (two out of 10 stimulation, see Table 1) were present in the individual with complete thoracic SCI (Case 3). These observations suggest a total destruction of the spinal autonomic pathways involved in generation of SSR in all these individuals. The ASIA neurological examination revealed that in two cases (Cases 1 and 2) there was incomplete injury to motor and sensory pathways but complete interruption of autonomic pathways involved in the generation of SSR. However, in Case 3 there was complete injury to motor, sensory and autonomic pathways.

Table 1 Frequency of elicited SSRs for stimulation of the median (Med. N.) and posterior tibial (Tib. Post. N.) nerves in individuals with SCI

	ASIA	Case 1		Case 2		Case 3	
		C5	ASIA C	C5	ASIA B	T3	ASIA A
L. Med. N	R arm	0/10		0/10		0/10	
	L arm	0/10		0/10		2/10	
	R foot	0/10		0/10		0/10	
	L foot	0/10		0/10		0/10	
L. Tib. Post. N	R arm	0/10		0/10		0/10	
	L arm	0/10		0/10		0/10	
	R foot	0/10		0/10		0/10	
	L foot	0/10		0/10		0/10	

SSR were recorded simultaneously from all four extremities. Ten stimuli were applied to each nerve in random fashion in order to eliminate habituation (dominator). Number of positive SSR presented as a numerator

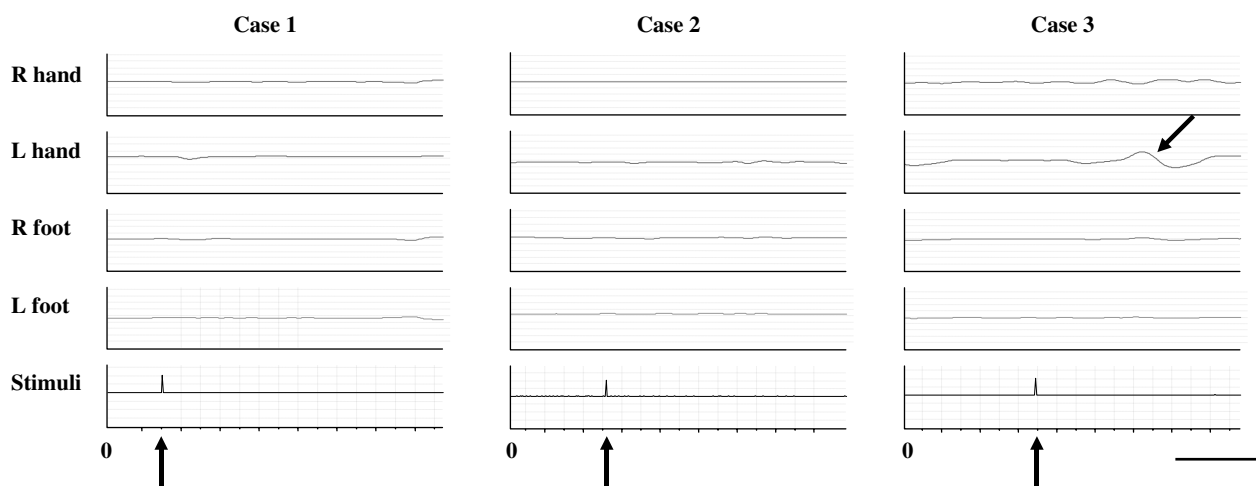


Figure 2 Sympathetic skin responses (SSRs) recorded in three individuals in response of stimulation of the median nerve. No SSR were obtained in two individuals below the level of injury (Cases 1 and 2). Inconsistent responses were present in individual with thoracic SCI in left arm (arrow in Case 3, see Table 1). These findings suggest a total destruction of the spinal autonomic pathways involved in generation of the SSR in these individuals. Arrows indicate time of stimulation. Calibrations: time is 2 s, amplitude is 5 μ V

Discussion

Abnormal cardiovascular control following SCI

SCI disrupts connections between neurons in the brain and spinal cord leading to lower resting arterial BP in the acute stage of SCI. With time, volatile changes in arterial pressure, known as AD can develop in persons with SCI above the neurological level T6.^{2,3,15} In this condition, normally mild stimuli such as distension of the urinary bladder or bowel results in dramatic elevation in BP that may lead to intracranial hemorrhage and stroke.¹⁶ These cardiovascular abnormalities are attributed to autonomic instability caused by the combination of changes occurring within the spinal cord after SCI, including loss of descending autonomic control, plastic changes within spinal circuits and peripheral receptor hypersensitivity.^{3,17-19}

It is important to acknowledge that severity of cardiovascular dysfunctions after SCI varies between individuals. For example, AD, even in quadriplegics, is not always severe: it may be characterized by only sweating or piloerection, or may even be asymptomatic.¹⁵ There is clinical evidence that severity of AD correlates with completeness of spinal injury as assessed by ASIA score: only 27% of incomplete quadriplegics present signs of AD in comparison with 91% of complete quadriplegics.²⁰ One report noted that while patients with motor-complete SCI (ASIA A or ASIA B) had a higher percentage of AD during their initial hospitalization than did patients with motor-incomplete SCI, the latter group were also at risk for developing AD between 1 and 6 months after injury.²¹

Assessment of spinal autonomic circuits

Two individuals in this report had complete motor SCI (ASIA A and B) and one individual had incomplete motor SCI (ASIA C). However, ASIA assessment allows only for the examination of motor and sensory spinal pathways.⁸ Examination of the spinal autonomic pathways that are responsible for cardiovascular control is not a part of the standard evaluation of the individuals with SCI at the present time. Therefore, in addition to the standard motor and sensory examination (ASIA score), we also examined integrity of the spinal autonomic pathways in these individuals using SSR. Historically the SSRs are among the most acceptable and reliable techniques for the evaluation of the descending sympathetic tracts in individuals with SCI.^{22,23} This method allows the integrity of the sympathetic spinal circuits to be evaluated, as no other conventional electrophysiological procedures are available to assess the autonomic nervous system adequately.

Palmar SSRs are generated by activation of the sympathetic circuits within the upper thoracic region (T1–T4) and pedal SSRs by activation of lower thoracic (T10–T12) segments (the last segments of the spinal cord contained autonomic preganglionic neurons).^{13,14,24} With injury below T3 segment, autonomic innervation of the palms is expected to be preserved.¹³ The level of

injury in all these individuals was established using ASIA score, that is, evaluation of the motor and sensory circuits of the spinal cord. Since different spinal tracts can be affected to different degrees by SCI in humans, autonomic pathways localized superficially within dorsal columns or spinal autonomic neurons localized centrally (around the central canal and within the lateral horn) could be injured to a different degree than the motor and sensory structures.⁹ Our findings are similar to the British investigators, who also reported that in some individuals, even with T4 and T5 ASIA A, palmar SSRs were absent when the peripheral nerve either above or below the level of lesion was stimulated.¹⁴ Similarly, our patient (Case 3) with T3 ASIA A had only partial preservation of palmar SSR.

It has been previously shown that tetraplegic and paraplegic patients with a lesion above T6 who presented with bladder dysfunction and associated AD either had abnormal or no SSR in their hands or feet.^{20,22} Furthermore, it is suggested that the absence of SSRs after SCI suggests complete interruption of autonomic supraspinal pathways.¹⁴ More recently SSRs were utilized for the assessment of the individuals with erectile dysfunction.²⁵ In the present study, all three individuals with malignant AD had a total loss of plantar SSRs and only in one case, partial preservation of palmar SSRs, suggesting severe injury of the descending autonomic pathways.

Trigger factors of AD in SCI

It is well documented that noxious or non-noxious stimuli below the level of injury (ie over-distended bladder, impacted bowel, ingrown toenail) in persons with SCI could trigger distressing symptomatic episodes of AD.^{3,26} Ejaculation is also a well-known trigger for these conditions.^{27,28} However, after ejaculation, episodes of AD are usually self-limiting in time to min, although an unwell feeling may persist for 30–60 min and certainly spasm may be reduced.^{29,30}

It has been our clinical experience that consistent ejaculation over time (months to years) has, in general, resulted in a diminution of symptomatic AD in some individuals who may have experienced severe AD at the onset of their ejaculatory experience. A resurgence of the severity of AD would often provoke investigation for urinary bladder blockage, urinary tract infection, decubitus ulcer or other health issues.³¹

The subjects under discussion in this paper have atypical, protracted AD with increased sensitivity to voiding and continued sexual activity that caused symptomatic and debilitating AD that required medical attention. Except for the one subject who still uses Propranolol[®], the other two individuals had a decline in their malignant AD over time without further medication. This prolonged and severe 'malignant' dysreflexia may have been relieved by urinary sphincter relaxation, removal of potential obstruction (such as dislodged bladder stone), reduced detrusor-sphincter dyssynergia or may simply have resolved with time. Our experience

at the VSRC suggests that, for some individuals, the symptoms of AD with repeated ejaculations can significantly reduce over time. This is in contrast to what would be expected with usual AD triggers such as urinary tract infections, colon impaction, pressure sores, etc. that would trigger a predictable AD episode with each occurrence.³ Although all the individuals in these case reports, once past their malignant AD, acknowledged decreased symptoms of AD with ejaculation as time progressed, their BP continued to be significantly elevated in sperm retrieval procedures. This discrepancy in symptoms and objective parameters of AD (silent AD) can sometimes give false assurance to both patients and clinicians working in this area.³²

The mechanism behind these case examples of malignant AD may involve an ongoing hyperexcitation of spinal circuits that are severed from the descending control (revealed by the absence of SSR in these cases) that do not deactivate once the initial triggering stimulus is removed. The underlying mechanism of this sustained, malignant AD as compared to the self-limiting AD described above is not fully understood. It can be speculated that a prior injury or inflammation to the internal pathway of semen propulsion needs time to heal and that these circuits remain activated until healing is complete. If this is the case, and if malignant AD is apparent and debilitating, the introduction of an indwelling Foley catheter may be prudent for a minimum of one month, along with bladder medications to reduce any sphincter spasm. This speculation needs further clinical investigation.

Conclusion

Although ejaculation was the initial trigger of 'malignant' AD in all three individuals, the combination of severe injury to the descending autonomic pathways and the presence of other triggers such as bladder irritations could be responsible for this condition. The importance of monitored ejaculation trials after SCI to assess the severity of subjective AD symptoms, the objective BP regardless of the symptoms, and the extrapolated safety of sexual activity cannot be emphasized enough (note Case 2 had severe BP rise with minimal symptoms during further ejaculation trials). Furthermore, we propose that SSR may be an effective tool to predict individuals who may be susceptible to more severe, prolonged malignant AD, as complete destruction of the descending autonomic pathways may be an underlying mechanism in this regard. SSR may be a more useful, important predictive factor in the severity and/or protraction of AD over the level and completeness of injury assessed by ASIA scores. Malignant AD in all three cases demanded medical intervention, interfered with the ability to work and caused a significant interruption in the social and sexual life of these individuals. Prompt medical attention, including urological investigation, to the underlying causes of dysreflexia and discouragement of further ejaculation until AD has been managed is recommended.

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