

Autonomic dysreflexia: the forgotten medical emergency

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Autonomic dysreflexia (AD) is a potentially dangerous complication of spinal cord injury (SCI). In AD, an individual's blood pressure may rise to dangerous levels and, if not treated, can lead to stroke and possibly death. All medical personnel, especially those interacting with SCI patients, must have a good understanding of its aetiology, complications and emergency management.

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Autonomic dysreflexia (AD) is classically seen in spinal cord injury (SCI) patients, when the level of injury is above that of the splanchnic sympathetic outflow, i.e. T6. Rarely, this may be seen in patients with SCI below T6 and is usually milder in presentation (Moeller and Scheinberg, 1973). The clinical features are more defined in complete lesions of the cord than in incomplete lesions, as well as in cervical lesions compared with thoracic (Kewalramani, 1980; Trop and Bennett, 1991). Even though the incidence of AD is reported to be higher among female SCI patients, males are four times more likely to develop SCI, making AD a predominantly male phenomenon (Shergill et al, 2004).

CLINICAL FEATURES

AD is a medical emergency characterized by uncontrolled hypertension associated with bradycardia. The patient complains of bilateral pounding headache, facial flushing, excessive sweating above the level of the lesion, nasal congestion, malaise, nausea, anxiety, blurring of vision and a feeling of impending doom. There may be tightness felt in the chest and goose bumps above, or possibly below, the level of lesion. On examination there is flushed sweaty skin above the level of the lesion with congestion of the conjunctiva and other mucous membranes. Below the level of the lesion, the skin is cool and pale with pilomotor erection, increased spasticity, bladder and bowel contractions and occasionally penile erections and ejaculation.

The most dramatic clinical feature is the sudden uncontrolled hypertension, with elevation of both systolic and diastolic blood pressures. Even though systolic pressures as high as 250–300 mmHg and diastolic pressures of 200–220 mmHg have been reported (Karlsson,

1999), any pressure rise of 20–40 mmHg should be considered as a sign of AD in susceptible individuals. The condition is associated with bradycardia as a result of parasympathetic vagal inhibition of the heart. Importantly, the resting blood pressure in SCI patients is often around 90/60 mmHg and therefore recordings of 120/80 mmHg may even be considered as elevated.

Although symptoms may be present, AD can present without any symptoms, characterized by elevation in blood pressure alone, and has been termed silent AD (Lisenmeyer et al, 1996).

PATHOPHYSIOLOGY

In a neurologically intact individual, sensory inputs from the periphery ascend via the dorsal and lateral spinothalamic tracts. This is also associated with intraspinal spread of impulses leading to the activation of primary cutaneous vascular, viscerovascular and somatovascular reflexes resulting in arteriolar vasoconstriction, pelvic visceral contraction and pilomotor spasms. The sensory afferents from the bladder are mediated via the capsaicin sensitive C-fibres, and intravesical treatment with capsaicin have been shown to sup-

Box 1. Case example of autonomic dysreflexia

A 36-year-old man presented to the accident and emergency department complaining of anxiety and 'goose-flesh' with a bilateral pounding headache. His symptoms had developed since his suprapubic catheter (SPC) had stopped draining urine. He had suffered a cervical spinal cord injury (SCI) 18 months before, which had left him with quadriplegia. He was found to be severely hypertensive (blood pressure = 210/115 mmHg) and bradycardic. He was sat upright and a single, unsuccessful attempt was made to flush the SPC. Immediately, a urethral catheter was inserted, and over the next few minutes 750 ml of urine drained and the patients symptoms and signs completely resolved. Following transfer to a SCI specialist centre, and in-patient observation, the SPC was changed and the urethral catheter removed and the patient has been well since.

press the bladder vascular reflexes (Geirsson et al, 1995). In a normal individual, the brain modifies these reflexes, thus preventing hypertension. In SCI, these messages from the higher centres are interrupted at the level of the lesion, leading to an uninterrupted sympathetic outflow below the level of the lesion. This can cause considerable rise in blood pressure, especially when the splanchnic sympathetic outflow is involved, i.e. lesions above T6, which provide the critical mass of blood vessels. This spike in blood pressure leads to the activation of parasympathetic responses via the baroreceptor mechanisms in the carotid sinus and aortic arch, which is limited above the level of lesion. The parasympathetic inhibition of the heart via the vagus nerve, resulting in bradycardia, is unable to counter the elevated blood pressure, as explained by the Poiseuille formula, where pressure in a tube is affected to the fourth power by change in radius (vasoconstriction) and only linearly by the flow through it (bradycardia).

The continual parasympathetic output above the level of lesion, results in vasodilation of cerebral and head and neck vasculature and pupillary dilatation. In high lesions there may be a transient rise in heart rate, presumably resulting from the sympathetic stimulation of heart, which is reversed once the parasympathetic response sets in.

PRECIPITANTS

AD can be triggered by any noxious stimuli below the level of the cord lesion (*Table 1*). The lower urinary tract accounts for the majority of these (75–85%), followed by the gastrointestinal tract, in 13–19% of cases (Lindan et al, 1980).

Bladder distension is by far the most common cause, which can result from a de-novo urinary retention or blocked, and kinked urinary catheters, or from infrequent intermittent catheterization. Other common urinary tract triggers include infection, stones or instrumentation. Common gastrointestinal stimuli include faecal impaction, manual evacuation of stools, instrumentation, haemorrhoids and anal fissures.

Labour and delivery in SCI patients is especially challenging with a reported incidence of AD in 85–90% (Plotz and von Hugo, 1996). Medications like nasal decongestants and misoprostol can also potentially trigger an episode of AD.

DIAGNOSIS

The presence of persistent high blood pressure, associated with bradycardia, in any patient with SCI is diagnostic of AD. Some patients may show the typical symptoms while others may

TABLE 1.
Aetiology of autonomic dysreflexia

Site	Stimulus
Genitourinary tract	<ul style="list-style-type: none"> • Bladder distension • Urethral distension • Procedures <ul style="list-style-type: none"> – Cystoscopy – Urodynamics – Catheterization – Extracorporeal shock wave lithotripsy – Percutaneous nephrolithotomy • Infection • Stones <ul style="list-style-type: none"> – Renal – Bladder – Ureter • Testicular torsion • Epididymitis • Scrotal trauma or compression • Sexual intercourse • Ejaculation
Gastrointestinal tract	<ul style="list-style-type: none"> • Faecal impaction • Bowel distension • Haemorrhoids • Anal fissures • Gastritis • Gastric ulcers • Gall stones • Appendicitis • Abdominal trauma
Cutaneous	<ul style="list-style-type: none"> • Pressure sores • Ulcers • Ingrowing toe nail • Burns, blisters, insect bites • Constrictive clothes, shoes or appliances
Orthopaedic	<ul style="list-style-type: none"> • Fractures • Dislocations • Heterotrophic ossification
Gynaecology	<ul style="list-style-type: none"> • Menstruation • Pregnancy • Labour • Delivery • Vaginitis
Medical therapy	<ul style="list-style-type: none"> • Decongestants • Misoprostol • Sympathomimetics
Others	<ul style="list-style-type: none"> • Deep vein thrombosis and pulmonary embolism • Syringomyelia

have few or no symptoms at all. Any sudden rise in blood pressure should be viewed with suspicion in susceptible populations. Prompt control of blood pressure on removing any obvious precipitating factor confirms the diagnosis. A typical case of AD is highlighted in *Box 1*. The diagnosis can also be established in a controlled setting by monitoring the vital signs during urodynamic study while filling the bladder and

TABLE 2.
Differential diagnosis of autonomic dysreflexia

Phaeochromocytoma
Cluster headaches
Migraine
Posterior fossa tumours
Toxaemia of pregnancy

importantly helps the patients to understand and recognize the early symptoms of AD (Lindan et al, 1980). The differential diagnosis of AD is listed in *Table 2*.

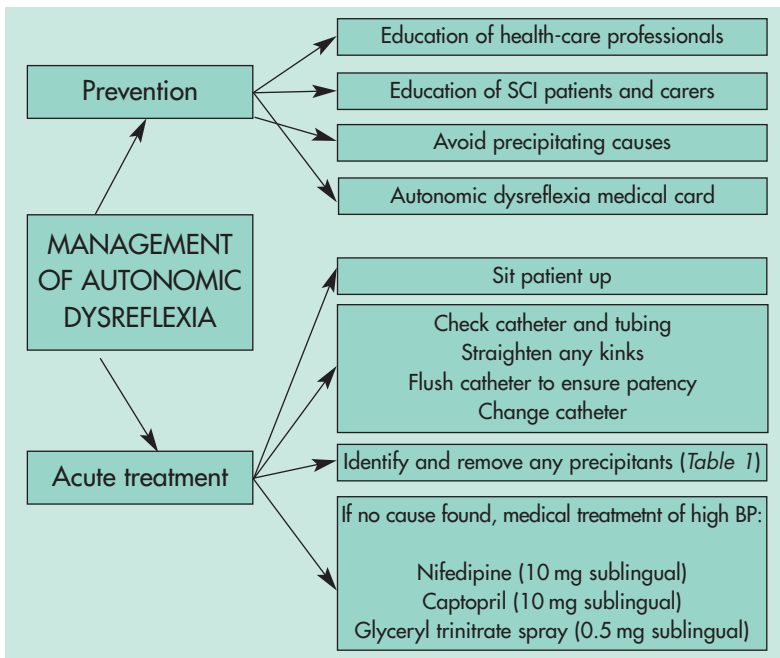
COMPLICATIONS

The majority of AD resolves without any significant consequence. Even so, if not treated adequately or in time, AD can have serious complications, mostly secondary to the very high blood pressure (*Table 3*)

MANAGEMENT

The management of AD can be broadly divided into prevention and the management of acute problems. An algorithm is presented in *Figure 1*. If an obvious cause for the acute episode cannot be found, the patient should be referred to the nearest specialist centre to investigate and identify any of the rare causes of AD. In severe and recurring

Figure 1. Algorithm for management of autonomic dysreflexia. SCI = spinal cord injury.



KEY POINTS

- Autonomic dysreflexia (AD) is a potentially dangerous complication of spinal cord injury (SCI).
- In AD, an individual's blood pressure may rise to dangerous levels.
- AD is a clinical emergency that can have serious consequences if not identified and corrected in time.
- Any personnel involved in the care of SCI patients should be fully educated regarding AD so that complications can be minimized.
- The condition can be easily and successfully managed in the vast majority of cases, provided it is recognized promptly.

TABLE 3.
Complications of autonomic dysreflexia

Convulsions
Subarachnoid haemorrhage
Intracerebral bleed
Hypertensive encephalopathy
Cardiac arrhythmias
Neurogenic pulmonary oedema
Retinal haemorrhage
Death

TABLE 4.
Neuroablative techniques available for resistant cases.

Subarachnoid block with alcohol or phenol
Sympathectomy
Sacral neurectomy
Rhizotomy
Corpectomy
Dorsal root ganglionectomy

cases, where pharmacological therapy fails or cannot be tolerated, a variety of neurological ablative procedures can be considered (*Table 4*).

CONCLUSION

AD is a clinical emergency that can have serious consequences if not identified and corrected in time. Any personnel involved in the care of SCI patients should be fully educated regarding AD so that complications can be minimized. This is of special interest to the urologist as the vast majority of cases are precipitated by stimuli from the urinary tract. The condition can be easily and successfully managed in the vast majority of cases, provided it is recognized promptly. **HM**

Conflict of interest: none

- Geirsson G, Fall M, Sullivan L (1995) Clinical and urodynamic effects of intravesical capsaicin treatment in patients with chronic traumatic spinal detrusor hyperreflexia. *J Urol* **154**: 1825–9
- Karlsson AK (1999) Autonomic dysreflexia. *Spinal cord* **37**: 383–91
- Kewalramani LS (1980) Autonomic dysreflexia in traumatic myelopathy. *Am J Phys Med* **59**: 1–21
- Lindan R, Joiner E, Freehafer A et al (1980) Incidence and clinical features of autonomic dysreflexia in patients with spinal cord injury. *Paraplegia* **18**: 285–92
- Lisenmeyer TA, campagnolo DI, Chou IH (1996) Silent autonomic dysreflexia during voiding in men with spinal cord injuries. *J Urol* **155**(2): 519–22
- Moeller BA, Scheinberg D (1973) Autonomic dysreflexia in injuries below the sixth thoracic segment (letter). *JAMA* **224**: 1295
- Plotz J, von Hugo R (1996) Autonomic hyperreflexia, pregnancy and delivery in para-tetraplegia. The obstetric anesthesiologic viewpoint on a case. *Anaesthesist* **45**: 1179–83
- Shergill IS, Arya M, Hamid R et al (2004) The importance of autonomic dysreflexia to the urologist. *BJU Int* **93**(7): 923–6
- Trop CS, Bennett CJ (1991) Autonomic dysreflexia and its urological implications: a review. *J Urol* **146**: 1461–9