

Recurrent Autonomic Dysreflexia; A Case Report and Review of the Literature

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Abstract

Autonomic dysreflexia is induced by spinal reflex mechanisms which continue to be intact in spite of the patient's injury. Any proprioceptive or noxious stimuli below the injury level generates an afferent impulse. This initiates a generalized sympathetic response. In response, it results in vasoconstriction below the neurologic lesion. We present a case of a male patient with C6 ASIA-A tetraplegia who developed recurrent dysreflexic episodes which were relieved by replacing the clean intermittent catheterization by indwelling Foley catheterization.

Keywords: Autonomic dysreflexia, tetraplegia, rehabilitation

INTRODUCTION

Autonomic dysreflexia (AD) is an emergency syndrome characterized by excessive, uncontrolled sympathetic output seen in patients with spinal cord injury (SCI) at or above the sixth thoracic neurologic level.¹ An acute rise in blood pressure (20-40 mmHg increase in systolic blood pressure), reflex bradycardia, anxiety, visual changes, nasal congestion, headache, flushing and sweating above the level of injury usually occur. Any proprioceptive or noxious stimuli below the level of the injury such as bladder distension or blocked urinary catheter, fecal impaction, pressure ulcers, ingrown toenails, urinary tract infection, or bladder stones can trigger the potentially life-threatening complication of SCI.²⁻⁴ Bladder distension is the most frequent stimulus which is seen in 75% to 85% of cases.¹ A sudden rise in arterial blood pressure may cause seizures, intracerebral hemorrhage, or even death.²⁻⁵ AD is more common and the reaction seems to be more severe in patients with complete lesions.^{1,6}

In this report, we present a male patient with C6 ASIA-A tetraplegia who developed recurrent dysreflexic episodes which were relieved by replacing the clean intermittent catheterization (IC) by indwelling Foley catheterization.

CASE PRESENTATION

A 52-year-old man with C6 tetraplegia ASIA-A due to traumatic C5-6 dislocation 3 months prior had urinary and fecal incontinence. He was hospitalized in the pulmonary medicine inpatient service for the treatment of pulmonary embolism and was using warfarin. Clean IC was being administered to manage neuropathic bladder. On the 44th day of hospitalization, his blood pressure, which was usually between 80/50 mmHg and 100/60 mmHg, increased to 180/75 mmHg without any complaints and it decreased to 95/55 mmHg after placing the patient in an upright position. Three hours later, his blood pressure again increased to 190/90 mmHg and decreased to 85/50 mmHg by again placing the patient in an upright position. Two hours later, he

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developed another hypertensive attack characterized by hypertension (190/80 mmHg), pounding headache, cutis anserina, and flushed, sweaty skin at the neck. His heart rate was 66 beats per minute, his respiratory rate was 32 and his temperature was 37 °C. His blood pressure did not decrease after being placed in an upright position for 3 minutes. Suspecting a possible urinary distension, his bladder was emptied via clean IC. Following that, his blood pressure decreased to 120/70 mmHg, and his headache and other symptoms disappeared immediately. Two days later, he had another dysreflexic attack (170/75 mmHg) with a less severe headache which was resolved after bladder emptying. He also had frequent urine leakages between clean ICs. His urinalysis and urinary cultures were normal. Clean IC was stopped and an indwelling Foley catheter was inserted, which prevented the occurrence of further autonomic dysreflexic attacks. Informed consent was obtained.

DISCUSSION

AD is induced by spinal reflex mechanisms which continue to be intact in spite of the patient's injury. Any proprioceptive or noxious stimulus below the injury level generates an afferent impulse. This initiates a generalized sympathetic response. In patients with SCI, descending central inhibitory pathways are blocked and cannot modulate the sympathetic response. The loss of inhibitory and excitatory supraspinal input to the sympathetic preganglionic neurons is responsible for unstable blood pressure. It results in vasoconstriction below the neurologic lesion. As a result, peripheral and splanchnic vasoconstriction leads to hypertension. At or above the T6 lesion level, blood pressure increases because of the splanchnic vascular bed's critical vessel mass.^{1,7} Hypertension stimulates baroreceptors and a baroreceptor-mediated increase in vagal activity causes vasodilatation above the lesion level to oppose hypertension centrally. As a result of vagal activity, reflex bradycardia is often seen.^{1,6}

Above the level of the lesion, peripheral vasodilatation induced by excessive parasympathetic output and a lack of sympathetic tone causes headache, nasal congestion, flushing and sweating in the head and neck region.^{1,6}

In bladder management, incidences of symptomatic AD have been reported to be higher in those patients with reflex voiding and indwelling supra-pubic catheterization, even though the indwelling supra-pubic catheter is supposed to prevent AD reactions as opposed to inducing them. The aim of the treatment is to have a balanced bladder, which is completely emptied at regular intervals, thus obtaining low intravesical pressure and being free of urinary tract infections. In patients with SCI, the latest urologic information suggests clean IC as the gold standard for the drainage of the bladder and it supports the discontinuation of indwelling urinary catheter usage.^{2,6} A degree of independence is possible with ICs, and also, they negate the disadvantages of permanent urinary catheterization.⁸ Urinary tract infections and bacteriuria are more common among patients using ICs.⁶ Symptomatic AD risk with ICs was reported to be low. However, bladder distension between ICs was the precipitating factor in our case and the insertion of an indwelling Foley catheter prevented the occurrence of dysreflexic episodes in our patient. For those patients suffering from recurrent episodes of AD, terazosin is effective in the complete improvement of dysreflexic symptoms and the prevention of serious damage caused by AD.^{4,8} The best approach would have been the continuation of IC along with terazosin treatment in our patient. However, our patient was receiving warfarin treatment and minor and major bleeding complications

may be seen during anticoagulation therapy.⁹ IC carries a small risk of localized trauma and urethral perforation.¹⁰ Therefore, we decided to continue the application of the indwelling Foley catheterization and to reassess the patient after the discontinuation of the warfarin treatment. Episodes were immediately resolved after bladder emptying, so bladder distension was thought to be the precipitating factor.

In some SCI patients, recurrent episodes of autonomic dysreflexia may develop due to an underlying reason such as dyssynergic voiding or an indwelling urinary catheter.⁴ Bladder distension between ICs was the cause of the recurrent attacks in our patient and indwelling catheterization resolved these attacks in our case.

For the acute management of AD episodes, the aim is to achieve an orthostatic drop in blood pressure, for which the first step is to place the patient in an upright position. The next step is to loosen any tight or restrictive clothing or constrictive devices. Immediately afterwards, possible triggering factors such as an obstruction of the urinary outlet or any fecal mass should be dealt with. Blood pressure should be monitored every 2 to 5 minutes. If systolic blood pressure continues to be 150 mmHg or higher, the use of antihypertensive drugs is necessary.^{1,6,7,11} The best and the most commonly used antihypertensive medications to manage AD are nitrates and, especially, nifedipine (immediate release form), which have a rapid onset and short duration of action.¹ Fortunately, the dysreflexic attacks in our patient were resolved without the need for drug treatment.

CONCLUSION

Autonomic dysreflexic attacks due to bladder distension between ICs were totally prevented by the insertion of an indwelling Foley catheter in our patient. Although the avoidance of indwelling urinary catheters is highly recommended and clean IC is the gold standard for drainage of the bladder in SCI patients as is known, it is sometimes not applicable.

MAIN POINTS

- Autonomic dysreflexia is a clinical emergency syndrome which causes an acute rise in blood pressure.
- In patients with spinal cord injury, clean intermittent catheterization is the gold standard for the drainage of the bladder.
- Bladder distension between intermittent catheterizations can precipitate autonomic dysreflexia and indwelling Foley catheters can be used to prevent the occurrence of dysreflexic episodes in these patients.

ETHICS

Informed Consent: It was obtained.

Peer-review: Externally peer-reviewed.

Authorship Contributions

Concept: P.H.Y., A.E., Design: P.H.Y., A.E., Supervision: P.H.Y., A.E., Materials: P.H.Y., A.E., Data Collection and/or Processing: P.H.Y., A.E., L.M.G., F.H., F.R., Analysis and/or Interpretation: P.H.Y., A.E., L.M.G., F.H., F.R., Literature Search: P.H.Y., A.E., Writing: P.H.Y., Critical Review: A.E., L.M.G., F.H., F.R.

DISCLOSURES

Conflict of Interest: The authors have no conflicts of interest to declare.

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