TELE-REHABILITATION GUIDELINE

Management of Acute Autonomic Dysreflexia (AD)

Author(s):	Thomas Kiser, Rani Lindberg, Saint Adeogba	Peer Reviewed:	Thomas Kiser, Lucas Bider	Finalized:	February 2014
Drafted:	February 2014	Date:	February 2018	Published:	July 2018

I. Definition, assessment, and diagnostic considerations

A. Definition

1. Autonomic Dysreflexia (AD) is acute hypertension generated by unmodulated sympathetic reflexes below the level of spinal cord injury (SCI). AD is usually triggered by noxious stimulation below the level of injury which activates a massive sympathetic discharge and widespread vasoconstriction. [1]
   a. SCI is typically at or above the sixth thoracic spinal cord segment (T6). [1] Cases involving the T8 neurologic level have been reported. [2]
   b. Descending sympathetic inhibitory signals that originate above T6 are unable to traverse the zone of the spinal cord injury to mitigate the hyperactivity. [3]
   c. Due to loss of descending modulation hypertension persists until noxious stimulus is withdrawn [1]
   d. Typically accompanied by baroreceptor-mediated bradycardia although tachycardia can occur [1,5]
   e. Requires immediate treatment to prevent hypertensive encephalopathy, stroke, cardiac arrest, seizure, and/or death [1]

B. Assessment

1. Signs and symptoms [4]
   a. Hypertension (blood pressure 20-40mmHg above baseline) with or without:
      1) Bradycardia; tachycardia is possible [1,5]
      2) Pounding headache
      3) Profuse sweating, vasodilation or skin flushing of face, neck, shoulders, and trunk above the level of SCI.
      4) Vasoconstriction below the level of the SCI
      5) Piloerection or goose bumps above the level of SCI
      6) Blurred vision
      7) Spots in the visual fields
      8) Nasal congestion
      9) Apprehension or anxiety
      10) Minimal to no symptoms despite elevated blood pressure
      11) Sudden Death [12]
2. Etiology [4]
   a. Urinary system (most common; 75-90% of cases) [1, 3, 5, 6]
      1) Bladder distention/urinary tract infection
      2) Kidney stones
      3) Obstructed urinary catheter
      4) Urological instrumentation of genitourinary tract
         a) Catheterization of urethra
         b) Cystoscopy
   b. Gastrointestinal system (second most common) [1, 3, 5]
      1) Bowel distention
      2) Bowel impaction
      3) Appendicitis

4) Gallstones or cholecystitis
5) Gastric ulcers or gastritis
6) Gastrointestinal tract instrumentation
7) Hemorrhoids

c. Integumentary system
1) Pressure ulcers
2) Constrictive clothing, shoes or appliances
3) Contact with hard or sharp objects
4) Blisters
5) Burn, sunburn or frostbite
6) Ingrown toenail
7) Insect bite

d. Reproductive system
1) Intercourse
2) Sexually transmitted diseases
3) Ejaculation
4) Epididymitis
5) Scrotal compression
6) Vibratory stimulation
7) Menstruation
8) Pregnancy
9) Vaginitis

e. Other
1) Performance-enhancing drugs (e.g. stimulants)
2) Deep vein thrombosis
3) Excessive alcohol
4) Excessive caffeine or other diuretic intake
5) Fractures or trauma
6) Heterotopic ossification
7) Pulmonary emboli
8) Substance abuse
9) Surgical or invasive diagnostic procedures

C. Diagnostic Considerations
1. Suspect in patients with SCI at T6 or above who develop acute hypertension likely with associated bradycardia.
2. Key populations:
i. Pregnant women
1. AD in pregnant women can be mistaken for preeclampsia [4]
ii. Patients undergoing urological procedures
1. Procedures such as cystometry, cystoscopy, transurethral litholapaxy, extracorporeal shock wave lithotripsy, and penile vibrostimulation can trigger AD [6, 7]
2. Urodynamic study risk factors that have been determined to increase the risk of AD are: patient's age, SCI completeness, traumatic etiology, indwelling catheter, presence of chills or sweating, anticholinergic treatment, maximum detrusor voiding pressure, detrusor pressure at maximum flow rate, detrusor external sphincter dyssynergia, and bladder outlet obstruction. The two major risk factors were. patient's age equal to or above 45 years of age (OR=10.995) and maximum

detrusor voiding pressure equal to or above 31 cm H2O (OR=3.879). [13]

II. **Acute Management [4]**

A. Check blood pressure

1. If blood pressure is not elevated but signs and symptoms are present and the cause has not been identified, continue to look for nociceptive cause and if necessary refer to an appropriate consultant depending upon the symptoms.
2. If blood pressure is elevated
   a. Immediately sit the person up if the individual is supine
   b. Loosen clothing or constrictive devices
   c. Monitor blood pressure and heart rate frequently (every 2-5 minutes)

B. Survey urinary system

1. If an indwelling urinary catheter is not in place, catheterize the individual. Prior to inserting the catheter, instill 2% Lidocaine jelly into urethra and wait two minutes, if possible (in order to prevent additional nociceptive sensory input).
2. If the individual has an indwelling catheter
   a. Check the system along entire length for kinks, folds, constrictions or obstructions.
   b. Check for correct placement of the indwelling catheter.
   c. If a problem is found, correct it immediately.
      1) If the catheter appears blocked, gently irrigate with 10-15 mL of normal saline (in children <2, use 5-10 mL) warmed to body temperature.
      2) If the catheter is draining and the blood pressure remains elevated, suspect fecal impaction.
      3) If the catheter is *not* draining and the blood pressure remains elevated, remove and replace the catheter.
         i. Prior to replacing the catheter, repeat 2% Lidocaine jelly installation and wait two minutes. If difficulties arise in replacing the catheter, consider attempting to pass a coudé catheter or consult urology.
         ii. Monitor blood pressure during bladder drainage.
         iii. If acute symptoms of AD persist, including a sustained elevated blood pressure, suspect fecal impaction.

C. Evaluate systolic blood pressure (SBP): if SBP $\geq$ 150 mmHg, consider pharmacologic management as outlined below. If SBP < 150 mmHg proceed to step D.

1. Use an antihypertensive agent with rapid onset and short duration
   a. Topical 2% nitroglycerin paste (1-2 inches applied to the skin above the level of SCI) [1, 8, 9]
      1) Nitrates are contraindicated in patients using Sildenafil or other PDE5 inhibitor
   b. Nifedipine (10 mg PO or SL) [1, 10]
   c. Labetalol (100 mg PO or 20 mg IV), prazosin (1-2 mg PO), hydralazine(10-25 mg PO or 10-20 mg IM/IV) or IV sodium nitroprusside (0.3-10 mcg/kg/min) for severe, refractory cases [1, 7, 11,14]
1. Monitor for symptomatic hypotension and if low elevate the foot of the bed and add external compression garments, such as an abdominal binder or TED hose.

D. Survey GI system, beginning with evaluation for fecal impaction.

1. Evaluate for fecal impaction by checking the rectum for stool.

a. Using gloved hands apply 2% lidocaine jelly into rectum; wait 2 minutes to proceed if possible
b. Insert lubricated finger into rectum and check for stool; remove if present
c. Stop exam if blood pressure worsens. Instill more anesthetic, wait 20 minutes and resume exam.

E. If the precipitating cause has not been determined, check for less frequent causes of noxious stimulation delineated above that can cause AD. The individual may need to be admitted to the hospital for monitoring and pharmacological control of blood pressure.

F. Follow up:

1. Following an episode of AD, instruct the individual to monitor symptoms and blood pressure for at least 2 hours after resolution.
2. Consider admission for monitoring to maintain pharmacologic control of the blood pressure and to investigate other causes.
3. Document the episode in the medical record and include evaluation of treatment efficacy.
4. Once the patient is stabilized, review with the patient, family and friends the causes of AD and preventative measures.
5. Schedule a detailed evaluation for patients with recurrent AD

III. Chronic Management

A. Blood pressure monitoring should be performed to detect development of AD in patients with history of SCI undergoing urological procedures [6]

B. Prophylactic administration of nifedipine or prazosin can prevent or mitigate AD in patients with history of SCI undergoing urological procedures [6, 7, 14]

1. Nifedpine can cause a prolonged drop in blood pressure for periods of up to 5 hours, which may result in dizziness, fatigue, and weakness.
2. Prazosin is a selective adrenergic blocker with a slower and less abrupt suppressive effect on blood pressure. Unlike nifedipine, which blocks both the renin-angiotensin (RAS) and a-mediated pathways, prazosin only affects the a-mediated pathways. These a-mediated pathways contribute greatly to the severity of AD. Retaining the renin-angiotensin pathways allows for maintained resting seated BP (which is more RAS-dependent after SCI)

C. It is important to manage AD in SCI aggressively because it is a life threatening complication of SCI. In a review of case reports in the literature, thirty-two cases of death or life-threatening complications of AD were reported. [15]

1. Twenty-three (72%) cases were CNS-related,
2. Seven (22%) cases were CV-related, and
3. Two (6%) cases were pulmonary-related.
4. In total, seven (22%) deaths were noted as a direct result of complications following an AD attack.

D. Aggressive spasticity management with an intrathecal baclofen pump can reduce the symptomatic episodes of AD even in those with other risk factors for development of AD, and should be considered it other treatment strategies are not successful. [16]

This guideline was developed to improve health care access in Arkansas and to aid health care providers in making decisions about appropriate patient care. The needs of the individual patient, resources available, and limitations unique to the institution or type of practice may warrant variations.

Guideline Developers

Guideline developed by Saint Adeogba, MD, in collaboration with the TRIUMPH team led by Thomas Kiser, MD, and Rani Lindberg, MD. 2014

Revised by Thomas Kiser, MD and Lucas Bider, MD 2018

Selected References

1. Eldahan, K.C. and A.G. Rabchevsky, *Autonomic dysreflexia after spinal cord injury: Systemic pathophysiology and methods of management.* Auton Neurosci, 2018. **209**: p. 59-70.
2. Erickson, R.P., *Autonomic hyperreflexia: pathophysiology and medical management.* Archives of physical medicine and rehabilitation, 1980. **61**(10): p. 431-440.
3. Teasell, R.W., et al., *Cardiovascular consequences of loss of supraspinal control of the sympathetic nervous system after spinal cord injury.* Arch Phys Med Rehabil, 2000. **81**(4): p. 506-16.
4. *Consortium for Spinal Cord Medicine: Acute management of autonomic dysreflexia: Individuals with spinal cord injury presenting to health-care facilities.* Journal of Spinal Cord Medicine, 2002. **25(Suppl. 1)**: p. 67-88.
5. Lindan, R., et al., *Incidence and clinical features of autonomic dysreflexia in patients with spinal cord injury.* Paraplegia, 1980. **18**(5): p. 285-92.
6. Liu, N., et al., *Iatrogenic urological triggers of autonomic dysreflexia: a systematic review.* Spinal Cord, 2015. **53**(7): p. 500-9.
7. Zheng, M.M., et al., *Prazosin: a potential new management tool for iatrogenic autonomic dysreflexia in individuals with spinal cord injury?* Neural Regen Res, 2015. **10**(4): p. 557-8.
8. Braddom, R.L. and J. Rocco, *Autonomic dysreflexia. A survey of current treatment.* American journal of physical medicine & rehabilitation, 1991. **70**(5): p. 234-241.
9. Solinsky, R., et al., *Pharmacodynamics and effectiveness of topical nitroglycerin at lowering blood pressure during autonomic dysreflexia.* Spinal Cord, 2017. **55**(10): p. 911-914.
10. Lindan, R., E.J. Leffler, and K.R. Kedia, *A comparison of the efficacy of an alpha-I-adrenergic blocker in the slow calcium channel blocker in the control of autonomic dysreflexia.* Paraplegia, 1985. **23**(1): p. 34-8.
11. *Valles, M., et al., Cerebral hemorrhage due to autonomic dysreflexia in a spinal cord injury patient. Sp*inal Cord, 2005. **43**(12): p. 738-740.
12. Dolinak, D, and Balraj, E, Autonomic Dysreflexia and Sudden Death in People with Traumatic Spinal Cord Injury Am J Forensic Med Pathol 2007;28: 95–98
13. Vırseda-Chamorro, M. et al, *Risk Factors to Develop Autonomic Dysreflexia during Urodynamic Examinations in Patients with Spinal Cord Injury.* Neurourol. Urodynam. 36:171–175, 2017.
14. Phillips, A.A., Elliott, S.L., Zheng, M.M., and Krassioukov, A.V. *Selective Alpha Adrenergic Antagonist Reduces Severity of Transient Hypertension during Sexual Stimulation after Spinal Cord Injury.* JOURNAL OF NEUROTRAUMA 32:392–396 (March 15, 2015)
15. Wan D., Krassioukov, A.V. *Life-threatening outcomes associated with autonomic dysreflexia: A clinical review.* The Journal of Spinal Cord Medicine 37 (1):1-10, 2014
16. Del Fabro, A.S., Mejia, M., Neumanaitis G. *An investigation of the relationship between autonomic dysreflexia and intrathecal baclofen in patients with spinal cord injury.* The Journal of Spinal Cord Medicine 41 (1): 102-105.