Management of Acute Autonomic Dysreflexia (AD)

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Definition, assessment, and diagnostic considerations

A. Definition

1. Autonomic Dysreflexia (AD) is an acute elevation in blood pressure generated by unmodulated sympathetic output below the level of spinal cord injury (SCI). The mechanism of AD is an uncoordinated sympathetic response triggered by a visceral or cutaneous stimulus below the level of injury, which activates a massive sympathetic discharge and widespread arterial vasoconstriction. [1,2]

a. The risk of developing AD increases with a higher level of SCI. [1]
b. In SCI, descending sympathetic inhibitory signals are disrupted, which leads to persistent hypertension until the stimulus is removed and/or pharmacologic therapy is initiated. [3]
c. Baroreflex-mediated bradycardia frequently accompanies episodes of AD as a result of the increased vagal activity to the heart, although tachycardia can occur. [1] The medullary vasomotor center will send parasympathetic signals, but vasodilation will only occur at the levels above the lesion. [1]
d. AD requires immediate treatment to prevent hypertensive encephalopathy, stroke, cardiac arrest, seizure, retinal hemorrhages, pulmonary edema, renal insufficiency, and/or death. [4, 8] AD can increase the risk of stroke by 300%-400%. [1]
e. AD is more frequent in chronic SCI patients, particularly between 3 and 6 months after injury [5]. However, AD can occur in the acute phase. [6,7] It has been demonstrated that AD occurs in 5.7% of patients with acute SCI above T6, and it can happen as early as 4 days after a severe cervical SCI. [7] The risk of AD tends to decrease after one year. [5]
f. It is important to note that AD can occur up to 40 times daily in susceptible patients. [1]

B. Assessment

1. Signs and symptoms [4]

a. An episode of AD is defined as an increase in systolic blood pressure of at least 20 mm Hg above baseline in adults and 15 mm Hg in children with or without: [1,5]

1) Bradycardia; tachycardia is possible [1,5]
2) Pounding headache
3) Profuse sweating, vasodilation, or skin flushing of the face, neck, shoulders, and/or 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) Confusion/Altered Mental Status
11) Nausea and/or vomiting
12) Miosis
13) Cardiac Arrhythmias
14) Thermo dysregulation
15) Anxiety
16) Minimal to no symptoms despite elevated blood pressure
17) Sudden Death

2. Etiology [4]

a. Urinary system (most common; ~85% of cases) [1,]
   1) Bladder distention/urinary tract infection
   2) Kidney stones
   3) Obstructed urinary catheter
   4) Urological instrumentation of the genitourinary tract
      a) Catheterization of the urethra
      b) Cystoscopy

b. Gastrointestinal system (second most common) [1, 3]
   1) Constipation
   2) Fecal impaction
   3) Appendicitis
   4) Gallstones or cholecystitis
   5) Gastric ulcers or gastritis
   6) Gastrointestinal tract instrumentation
   7) Hemorrhoids
   8) Anal abscesses
   9) Anal Fissures

c. Integumentary system
   1) Pressure ulcers and other skin tissues
   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
10) Breastfeeding

e. Other

1) Performance-enhancing drugs (e.g., stimulants, PDE5 inhibitors)
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, but can be seen with tachycardia. [1]

2. Key populations:

i. Patients undergoing urological procedures

1. Procedures such as cystometry, cystoscopy, transurethral litholapaxy, extracorporeal shock wave lithotripsy, and penile vibrostimulation can trigger AD [9, 10]

ii. Pregnant women

1. Symptoms of AD can mimic pre-eclampsia. However, the rise in blood pressure from pre-eclampsia will not resolve despite removing potential triggers. [11]

iii. Patients with a history of AD

1. If a patient has a history of AD, the risk of recurrence increases.
2. Triggers can repeat.

II. **Acute Management**

A. Check blood pressure:

1. If blood pressure is not elevated, but signs and symptoms of AD are present, and the cause has not been identified, continue to look for a nociceptive cause. If necessary, refer to an appropriate consultant depending upon the symptoms and duration of increased blood pressure. Studies show that the MAP goal in patients with acute spinal cord injury within the first seven days after injury is between 85 and 90 mmHg. [12]

2. If blood pressure is elevated:

a. Immediately sit the patient up to take advantage of Orthostatic hypotension seen in spinal cord injury.

b. Loosen clothing or constrictive devices to decrease possible nociception from the clothing or devices.

c. Monitor blood pressure and heart rate every 2-5 minutes.

B. Survey the urinary system

1. If an indwelling urinary catheter is not in place, catheterize the individual. Before inserting the catheter, instill 2% Lidocaine jelly into the urethra and wait two minutes, if possible, to prevent additional nociceptive sensory input from inserting the catheter. If initial placement of a Foley is difficult, a Coude catheter or a guidewire and a council-tip catheter are recommended. [1]

2. If the individual has an indwelling catheter:

a. Check the system along the entire length of the catheter and tubing for kinks, folds, constrictions or obstructions.

b. Check for the correct placement of the indwelling catheter.

If a problem is found, correct it immediately and then recheck the blood pressure.

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. Before replacing the catheter, repeat 2% Lidocaine jelly. Install and wait two minutes. If there is initial difficulty inserting the Foley catheter, use a Coude catheter or a Council tip catheter. Note that repeated attempts at catheterization can increase the blood pressure. If attempts at catheterization are unsuccessful, consult urology. It is important to avoid manually compressing the bladder due to the risk of increasing blood pressure and worsening AD. [1]

ii. Monitor blood pressure during bladder drainage. If acute symptoms of AD persist, including a sustained elevated blood pressure, suspect fecal impaction.

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

1. Use an antihypertensive agent with a rapid onset and short duration. Starting with topical therapy is generally a safer option because of its immediate effects on the blood pressure without lasting effects.

a. Topical 2% nitroglycerin paste (1-2 inches applied to the skin above the level of SCI), or sublingual nitroglycerin 0.4 mg.[1, 8, 9, 13] May repeat every 5 minutes if needed. Note: Nitrates are contraindicated in patients using Sildenafil or other PDE5 inhibitors due to the risk of excessive hypotension. In patients taking PDE5 inhibitors, sublingual captopril (dose of 25 mg) or nifedipine (dose of 10 mg) can be administered instead of nitroglycerin.

b. Oral Agents: Nifedipine (10 mg PO or SL) [1, 10, 14], 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]

c. 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 the GI system

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

a. Using gloved hands, apply 2% lidocaine jelly into the rectum; wait 2 minutes to proceed if possible

b. Insert a lubricated and gloved finger into the rectum with fingertips toward the spine and not the bladder. Then, if the stool is able to be removed, manually remove the stool. If there is no stool to remove, then proceed to move the finger in a slow circular motion for 10 rotations in an attempt to stimulate the rectocolic reflex. If the bowels have not emptied by this time, then insert a suppository or administer an enema. [3]

c. Stop exam if blood pressure worsens. Instill more anesthetic, wait 20 minutes, and resume the exam.

E. After the above steps have been followed, and 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 if in a clinic setting. If the patient is already admitted, then continue regular blood pressure monitoring and continue to look for noxious causes or other medical causes such as infection.

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. Document the episode in the medical record and include an evaluation of treatment efficacy.
3. Once the patient is stabilized, review with the patient, family, and friends the causes of AD and preventative measures.
4. Schedule a detailed evaluation for patients with recurrent AD.

III. Chronic Management

Twelve months after injury, the incidence of AD in SCI at T6 or above is 48–90%. [15]

A. Blood pressure monitoring at home should be regularly performed to detect the development of potential AD in patients with a history of SCI. Ambulatory blood pressure monitoring is especially important in those scheduled to undergo urological procedures [16]. Additionally, regular monitoring of blood pressure is imperative in spinal cord injury patients due to the increased risk of developing cardiovascular disease.

B. Prophylactic administration of nifedipine or prazosin can prevent or mitigate AD in patients with a history of SCI undergoing urological procedures [17]. During urologic procedures, it is important to monitor blood pressure. If bladder irrigation is necessary, consider using warmed normal saline, as cold saline can worsen autonomic dysreflexia.

1. Nifedipine is a common agent used for prophylaxis for potential urologic procedures. Patients should be counseled that nifedipine can cause a prolonged drop in blood pressure for periods of up to 5 hours, which may result in dizziness, fatigue, and weakness that can persist after the procedure is completed. Due to these risks and the risk of continued orthostatic hypotension, recent studies show that clinicians are using nifedipine at a lower frequency [13]. For patients with recurrent AD, consider using terazosin (1 mg per night, gradually increasing to 5 mg) and prazosin (0.5-1 mg for 2-3 times a day) [14].

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 α-mediated pathways, prazosin only affects the α-mediated pathways. These α-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)

3. For patients with recurrent AD and urologic procedures demonstrating increased bladder pressure at the detrusor sphincter, these patients may benefit from detrusor or urethral sphincter injections. Botox injections can decrease detrusor pressure and increase bladder compliance, therefore reducing the risk of AD. [18,19]

C. It is important to manage AD in SCI aggressively because it is a life-threatening complication of SCI. [20] In a review of case reports in the literature, thirty-two cases of death or life-threatening complications of AD were reported. [21]

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 the development of AD, and should be considered if other treatment strategies are not successful. [22,23]

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.

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