Management of Autonomic Dysreflexia

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Autonomic dysreflexia (AD) is a life-threatening complication of spinal cord injury at T6 or above that results in an uncontrolled sympathetic response secondary to a precipitant. If not recognized as a medical emergency and promptly treated, acute AD may result in devastating complications. Health care providers should be aware of the potential causes of AD and how to treat it when it occurs. This article reviews the acute management of AD, with special consideration to anesthesia and pregnant patients. The key to successful management of recurrent AD is prevention through education, identification, and avoidance of noxious stimuli.

Introduction

Autonomic dysreflexia (AD) is a life-threatening complication of spinal cord injury (SCI) at T6 or above that results in an uncontrolled sympathetic response secondary to a precipitant. Acute AD is considered a medical emergency; health care providers should be familiar with its signs, symptoms, and treatment in patients with SCI. The incidence of AD for SCIs above T6 is between 50% and 70% [1–3]. However, the signs and symptoms of AD may be minimal or absent, despite elevated blood pressure. Cognitive and verbal communication impairment may also make it difficult for SCI patients to relate symptoms. The key to successful management is prevention through patient and family education; proper bladder, bowel, and skin care; and identification and avoidance of noxious stimuli. Health care providers should be aware of the potential causes of AD and how to treat it when it occurs.

Diagnosis

Early recognition of signs and symptoms of AD is a major key to immediate and appropriate treatment of this urgent condition. Late recognition or inappropriate management may result in severe hypertension and complications such as cerebral or subarachnoid hemorrhage, seizures, arrhythmia, coma, and even death [4–8]. Suspicion of AD should always be high in patients with SCI at T6 or above. Normally, patients with SCI at T6 or above have normal systolic blood pressure of 90 to 110 mm Hg. A sudden 20- to 40 mm Hg increase of systolic and diastolic blood pressures over baseline that is frequently associated with bradycardia may indicate AD. An elevation of systolic blood pressure of 15 to 20 mm Hg in adolescents or higher than 15 mm Hg above baseline in children is significant and may suggest AD [9,10]. Other classic signs and symptoms of AD include pounding headache, profuse sweating above the level of the lesion, piloerection or goose bumps, flushing of the skin, blurred vision, spots in the visual field, nasal congestion, and anxiety. Besides bradycardia, other cardiac abnormalities may be encountered, such as cardiac arrhythmias (atrial fibrillation, premature ventricular contraction, and atrioventricular conduction anomalies). AD patients may have one or more of these symptoms, which may be minimal. If AD signs and symptoms are present but blood pressure is normal, patients should be referred to an appropriate consultant, depending on symptoms [10].

Acute Treatment

Life-saving measures

The acute management of AD requires several steps to be effective (Table 1). First, when blood pressure is elevated and typical signs and symptoms suggestive of AD are noted, patients should be placed upright in the seated position with legs down. This maneuver provokes an orthostatic drop in blood pressure by allowing pooling in the lower extremities. Tight clothing or any constrictive devices should also be loosened or removed to allow blood to pool in the abdomen and lower extremities [10].

Patients with SCI usually have impaired autonomic regulation. Their blood pressure has the potential to fluctuate rapidly and significantly during an AD episode; therefore, blood pressure should be monitored every 2 to 5 minutes until the patient has stabilized [9,10,11,12].

Eliminating the cause of autonomic dysreflexia

The next step is eliminating the triggering factors. The most common cause of AD is bladder distention [3]. An indwelling urethral catheter must be placed in the bladder if the patient does not already have one. Because catheter-

Table 1. Acute management of autonomic dysreflexia

Intervention	Rationale
1. Seat the patient upright and lower the legs	May allow pooling of blood in the lower extremities
2. Loosen clothing or constrictive devices	May allow pooling of blood in the abdomen and lower extremities
3. Monitor blood pressure every 2–5 min	Blood pressures may fluctuate rapidly during an AD episode
4. If no indwelling urinary catheter is present, catheterize the patient	Bladder distension is the most common cause of AD
5. If the patient has an indwelling urinary catheter, check it for obstruction and irrigate the catheter (which may need to be changed)	Bladder distension is the most common cause of AD
6. If systolic blood pressure is elevated to 150 mm Hg or above, consider pharmacologic management	The risk of adverse sequelae increases when systolic blood pressure exceeds 150 mm Hg
7. If symptoms persist, suspect fecal impaction	Fecal impaction is the second most common cause of AD
8. If symptoms persist, check for other less frequent causes	Many other precipitating factors may be the trigger, and appropriate treatment should resolve the AD episode
9. Consider admission or referral if symptoms persist or no precipitants are found	The patient is at risk of further episodes if no precipitant is found, and appropriate evaluation and treatment should be undertaken
10. Monitor symptoms and blood pressure for at least 2 hours after AD resolution	AD may resolve because of medication and not because of resolution of the underlying cause

AD—autonomic dysreflexia.

ization can exacerbate AD, apply intraurethral lidocaine jelly 2% and wait for 2 minutes. Intraurethral lidocaine jelly may decrease sensory input and relax the sphincter [10••]. If the patient already has an indwelling urethral catheter, check for obstruction or kinks on the catheter and tubing system. Irrigation of the catheter with 10 to 15 mL of warm saline may help if obstruction of the catheter is suspected (the use of a large volume or of a cold solution can exacerbate AD). Irrigation should also be limited to 5 to 10 mL for children under 2 years of age and to 10 to 15 mL in older children [10]. Lidocaine solution for irrigation may help decrease sensory input from the bladder. Avoid bladder distention and suprapubic percussion because it can worsen the condition. Catheter change with lidocaine jelly 2% might be necessary if the previous attempt to relieve the obstructed catheter has failed to decompress the bladder. A catheter with a coudé tip may be considered if catheterization is difficult or associated with bladder neck obstruction. During bladder decompression, monitor the patient's blood pressure because sudden decompression will normalize it. However, hypotension might occur after resolution of the trigger, especially if the patient has been given antihypertensive medication [10].

Pharmacotherapy to lower blood pressure

Persistent elevated blood pressure may be caused by fecal impaction, the second most common cause of AD. Appropriate precautions should be taken before disimpaction because additional stimulation may further aggravate AD. Use intrarectal lidocaine jelly 2% and wait 2 minutes before checking for a stool. If present, gently disimpact it. If AD worsens, stop manual evacuation and recheck after 20 minutes [10].

Pharmacologic management of high blood pressure should be considered if blood pressure is above 150 mm Hg in adults, 140 mm Hg in adolescents, 130 mm Hg in children 6 to 12 years old, and 120 mm Hg in children 5 years old and under [10]. Appropriate-sized blood pressure cuffs are necessary when measuring blood pressure in children and adolescents. Antihypertensive medication should preferably have a rapid onset and short duration of action. Nifedipine or nitrates are the most commonly used medications during acute attack. Nifedipine, 10 mg, bitten and swallowed (not taken sublingually [SL]) in the immediate-release form, is the preferred method of administration. The treatment can be repeated in 30 minutes. Extreme caution should be exercised in the elderly or in patients with coronary artery disease; it has been reported that nifedipine can cause hypotension and reflex tachycardia in individuals without SCI [10,11–14]. Nitropaste (nitroglycerin ointment) 2% as a 2.5-cm application on the skin above the level of the lesion is useful, as the paste can easily be removed when the hypertension subsides, and risk of subsequent hypotension may be reduced. Intravenous sodium nitroprusside drip may be required if the hypertension is refractory to the initial medical treatment [10]. Phenoxybenzamine, 10 mg, an α-receptor blocker, can be orally administered to treat acute AD; it relaxes the internal sphincter and controls the symptoms. An α-receptor blocker such as tamsulosin is not recommended in the acute treatment of AD [15]. Glyc-

eryl trinitrate, 300 to 600 μg SL, can be administered in an appropriately monitored setting for rapid blood pressure control. It can be repeated after 10 minutes.

Sildenafil is increasingly used to treat erectile dysfunction in SCI patients. Nitrates are contraindicated in patients taking sildenafil. The resulting blood-pressure decrease may be significant and dangerous. If sildenafil was used in the previous 24 hours, another short-acting antihypertensive medication should be given. However, if the patient receives nitrates, he should be informed not to take sildenafil for at least 24 hours.

Prazosin is an alternative for the management of hypertensive emergencies in AD. A pilot study has shown that captopril 25 mg SL is safe and effective to treat patients with hypertension caused by AD. Prazosin and captopril act within 30 minutes, achieve therapeutic levels within 1 to 3 hours, and have a half-life of 2 to 4 hours.

Hydralazine, 5 mg, intravenous (IV) or 5 to 20 mg intramuscular (IM) is another alternative that may be administered and repeated every 5 minutes, as necessary, to maintain proper blood pressure [10,15–19].

Managing secondary hypotension

Blood pressure should be monitored for possible hypotension. The patient should be placed lying down with legs elevated. Intravenous fluids and an adrenergic agonist should also be considered if symptomatic hypotension persists. If the precipitating cause has still not been determined, check for the least frequent cause. After an episode of AD, patients should be instructed to monitor symptoms and blood pressure for 2 hours after resolution of the episode to identify recurrences [10].

Prevention and Prophylactic Treatment

Prevention is the most important aspect of AD management. Patients should be offered early, structured education and support, and appropriate follow-up. Special consideration of the genitourinary system is required to avoid urinary tract complications, such as acute urinary retention, urinary tract infection, or bladder calculi. Education should also be emphasized to reduce AD triggers, including gastrointestinal complications such as constipation, pressure ulcers, and ingrown toenails. Documentation of previous AD episodes is important and should include presenting signs and symptoms, triggers implicated, treatment implemented with blood-pressure documentation, and response to treatment [10].

In patients with recurrent attacks, an α-adrenoceptor blocker may result in some suppression of dysreflexic symptoms; a nightly dose of terazosin, 5 mg, or tamsulosin, 0.8 mg, may reduce the frequency, severity, and bother of AD [15,19]. Acute AD may be precipitated by surgical, cystoscopic, urodynamic, and radiologic procedures. Prophylactic nifedipine, 10 mg, or nitropaste 2% could therefore be given shortly before the procedure, especially if the patient is known to have recurrent acute AD episodes. Prophylactic treatment of chronic patients with an α-adrenoceptor blocker or premedication before a procedure does not eliminate the need for careful monitoring during provocative procedures [7,10–12,15,19].

Anesthetic Considerations

Urologic manipulations or surgery account for a significant proportion of AD triggers. Multidisciplinary preoperative evaluation should include anesthesia assessment and determination of injury level with the history of dysreflexic episodes. Anesthetic techniques for controlling AD include topical application for cystoscopy, general anesthesia, and spinal and epidural anesthesia. Lidocaine jelly may decrease the sensation and relax the sphincter during cystoscopy, but bladder distention may trigger an AD episode despite local anesthesia. During procedures done under general anesthesia, nifedipine may be given prophylactically 30 to 60 minutes before surgery [20]. Halothane, isoflurane, and enflurane anesthesia have been found to effectively treat AD. If AD is encountered during the procedure, the first step should be to increase anesthetic depth [20]. Intraoperative and postoperative cardiac monitoring is warranted because AD may occur shortly after surgery.

Spinal anesthesia has been reported to provide excellent control, and has been recommended in acute AD refractory to medical management [20–29].

Treatment During Pregnancy and Labor

Autonomic dysreflexia is a common complication of pregnancy in SCI. Prevention remains the most important factor in AD management to avoid morbidity and mortality in patients or their fetuses.

During labor and delivery, risk of AD in patients with lesions at or above T6 is 85% to 90% [21]. SCI patients should be monitored for urinary tract infections, fecal impaction, and blood pressure during gestation and labor. AD during pregnancy has the same pathophysiology and requires the same type of management. AD signs and symptoms may be the only clue to the onset of labor. Unlike preeclampsia, elevated blood pressure and other classic symptoms of AD occur during uterine contraction and resolve with relaxation of the uterus [21]. In addition, before vaginal examination, urinary catheterization, or rectal manipulation, an anesthetic jelly should be used to reduce stimulation. Bladder catheter drainage should be initiated and frequently monitored to avoid obstruction during labor. It has also been proposed that monitoring during labor and delivery should ideally include an intra-arterial catheter for continuous blood-pressure reading, telemetry for continuous cardiac rhythm monitoring, and constant electronic fetal monitoring to identify fetal distress [22,23].

Complete anesthesia consultation should be undertaken before labor. AD control in labor without epidural block is unsatisfactory [24]. Epidural anesthesia interrupts the reflex arc from the uterus to the cardiovascular system via the spinal cord and is thought to prevent AD triggering. Prophylactic placement of an epidural catheter at 37 weeks of gestation has been proposed. Early placement of an epidural catheter is usually done at the first sign of labor [25]. Combination of bupivacaine and fentanyl has been reported in cases of successful deliveries in women with spinal cord lesions [25]. Because AD may occur up to 48 hours after delivery, maintaining epidural anesthesia for that period is usually recommended [27]. Oral nifedipine, intravenous hydralazine, or trimethaphan has been suggested to control extremely high blood pressure in this population during labor. Intravenous nitroprusside is not recommended because of elevated fetal cyanide levels. Ganglionic-blocking agents with a short duration of action, such as a 0.1% solution of trimethaphan in 5% dextrose by IV drip, can be administered in refractory AD cases during labor that is not adequately controlled by regional anesthesia [29].

Conclusions

AD is an emergency often secondary to urologic, gastrointestinal, or gynecologic problems or manipulations. Its management starts primarily with its prevention. Easy measures can avoid this highly risky event. Facing such events, physicians must be aware of simple procedures and the possible treatment cascade that could be undertaken. Pregnancy and anesthesia have to be considered as precipitating factors supporting preventive and aggressive management.

Disclosures

No potential conflicts of interest relevant to this article have been reported.

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