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The Tip of the Iceberg: A Thorough Workup of Autonomic Dysreflexia

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Clinical Vignette

N.U. is a 45-year-old man with a history of a chronic traumatic C4 AIS C spinal cord injury (SCI) and spasticity managed with an intrathecal baclofen (ITB) pump who presented to the UPMC outpatient SCI clinic with an increase in global spasticity despite stable long-term dosing. He also was experiencing diaphoresis, headaches, and increased systolic blood pressure. The patient reported an episode of jaw shaking and unresponsiveness. The symptoms were felt to be consistent with autonomic dysreflexia (AD), which the patient first noted during his initial baclofen pump surgical placement. At the clinic visit, left knee swelling was observed, and he mentioned that he had a cold a few weeks ago that had resolved. His active medical history included a chronic left stage IV ischial tuberosity pressure injury that was improving per caregiver report because it seemed like the opening of the wound was getting smaller. N.U. wanted help to get these symptoms under control.

Background: Autonomic Dysreflexia

There are over 18,000 new traumatic spinal cord injuries per year in the United States with incomplete tetraplegia being the most common level of neurologic injury.[1,2] Autonomic dysreflexia is a life-threatening emergency that can occur in people with SCI, primarily those with a neurologic level of injury at T6 and above, though it can less commonly happen below T6.[3] Approximately 50-90% of patients with lesions at T6 or above can get AD.[1-3, 3-5] AD may also be frequent and asymptomatic. When in AD, the patient's systolic blood pressure (SBP) rises at least 20 mmHg from their baseline. It should be noted that the baseline SBP in people with SCI is often lower than pre-injury vital signs. If left untreated,

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Disclosures

Dr. Vail reports no relationships with proprietary entities producing health care goods and services.

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AD can progress or contribute to myocardial infarction, cardiac arrhythmias, seizures, strokes, and death.[6-8] In some cases, there has even been retinal detachment and blindness. [4,5] [9,10] Implications from AD may also include global or cumulative cognitive decline or immune system depression.[11-12]

Pathophysiology

One of the major sources of vascular reserve is the splanchnic system which is located below the T6 vertebral level. When an afferent stimulus below the level of injury occurs, a massive, unchecked sympathetic reflex initiates, which is why blood pressure rises suddenly. There is a mismatch between the sympathetic and parasympathetic nervous systems resulting in an unopposed sympathetic response. This manifests typically as elevated SBP, vasodilation above the injury with vasoconstriction below, and bradycardia or tachycardia.[4,13,14] Common AD symptoms/presentations can be found in Table 1.

Table 1. Common dysreflexia presentations [2,6-9]

Elevated systolic BP by 20 mmHg
Headache • Pulsating, pounding
Diaphoresis • Above the level of the injury
Bradycardia • Tachycardia less commonly
Facial flushing or darkening
Pallor below level of injury
Anxiety
Nasal congestion
Piloerection
Blurred vision

Of note, symptoms like anxiety may occur in AD that can mistakenly lead physicians away from this diagnosis.[6]

AD is prompted by afferent noxious stimuli below the neurologic level of injury, for example by bladder distension. Common causes or stimuli of AD can be found in Table 2.

Table 2. Common causes of autonomic dysreflexia[10,11]

Bladder • Urinary retention • Urinary tract infection • Bladder/renal stones • Obstruction • Catheter kinking
Bowel • Unregulated bowel program • Obstruction • Bowel program itself
Skin • Ingrown toenail(s) • Tight clothing

Beyond these more frequent sources, any noxious stimulus below the level of injury can cause AD. Visceral reasons may require a broader workup and should be investigated if routine causes of AD are not found. A more extensive, but certainly not exhaustive list, can be found in Table 3.

Table 3. Less common causes of AD [6,9,12-16]

Myocardial infarction
DVT/PE
Visceral causes • Appendicitis • Hepatobiliary — Gallstones • Peritonitis • Neoplasm
Infection • Abscess (such as perirectal) • Sepsis
Wounds • Pressure injuries • Deep tissue injuries • Complications including osteomyelitis
Bone/Trauma • Compression/insufficiency fracture • Other fracture • Arthritis/joint pain
Menstruation
Pregnancy; Labor & Delivery
Sexual activity
Medical/surgical procedures

Autonomic Dysreflexia Treatment

Recognition is the first step to treatment. After identifying that the person is in AD, SBP should be checked every 2-10 minutes while trying to locate the trigger.[11,17] It is common and best practice to sit the patient up if they are not already, remove or loosen tight clothing, and check if the bladder needs to be emptied or if there is a catheter blockage. Sometimes the Foley or suprapubic catheters should be replaced. If a bowel movement is overdue depending on the person's bowel program, a quick-acting bowel program such as a polyethylene glycol-based bisacodyl "Magic Bullet™" suppository could be used.[18] A longer acting, oil-based bisacodyl suppository, other suppositories, enemas, or a specific regimen including senna, polyethylene glycol (MiraLAX) or others could help. If the trigger has not been found, it is common practice to use 1 inch of topical nitroglycerin (nitropaste) on the patient's chest or forehead. While there is no consensus on a "dangerous" blood pressure (it is thought to be the significant and frequent fluctuations in blood pressure over time that contribute to stroke/MI risk), it is widely accepted to initiate pharmacologic treatment at SBP greater than or equal to 150.[9]

It is important to apply topical nitroglycerin to an area that is both above the neurologic level of injury but also in a place that is visible, because once the trigger is found, the SBP will decrease back to baseline. If the topical nitroglycerin is not removed, it can cause the person's blood pressure to plummet into hypotension, and their brain could be at risk of hypoperfusion.[15] For alternatives to nitrates, which are contraindicated with phosphodiesterase type 5 (PDE5) inhibitors, please see the Consortium for Spinal Cord Medicine's Clinical Practice Guidelines Acute Management of Autonomic Dysreflexia. The guidelines acknowledge that nitropaste may not be available in at least Canada and Latin America, so other short-duration but fast-acting medications such as nifedipine, hydralazine, or sublingual clonidine are acceptable alternatives.[19,20]

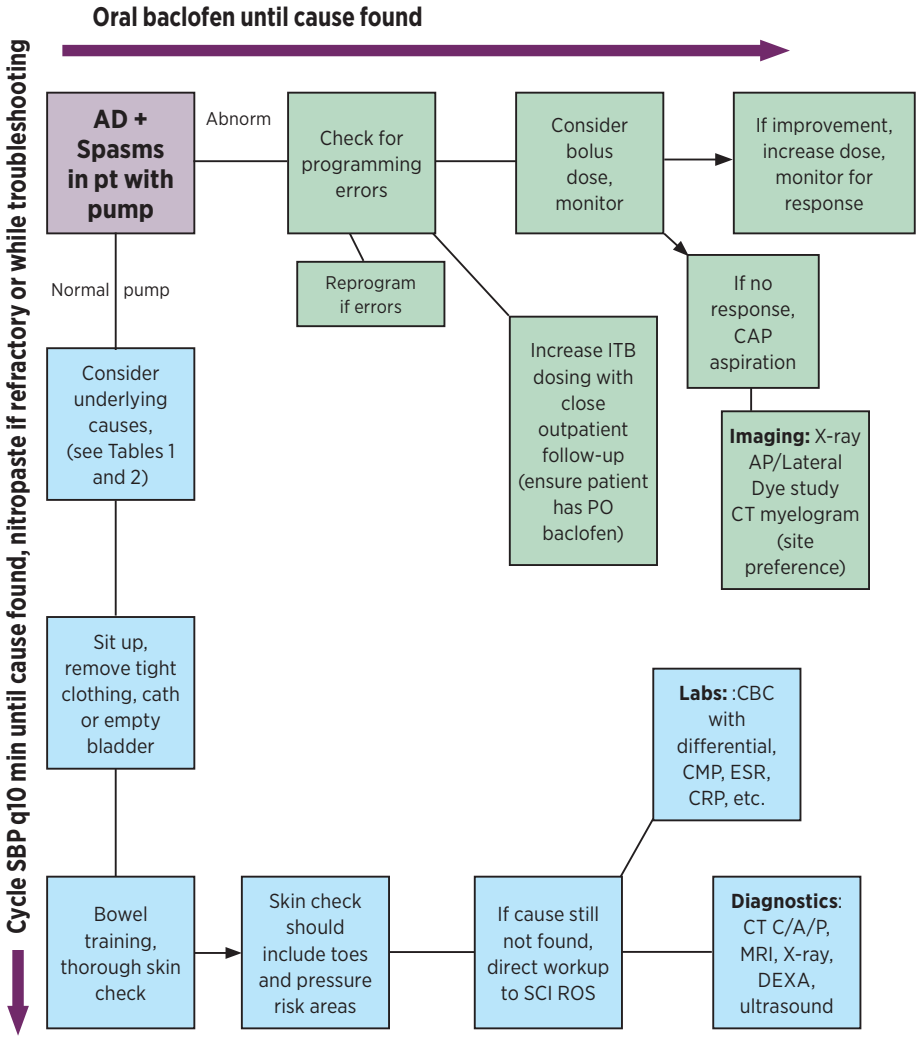
Intrathecal Baclofen Pump Interrogation in the Setting of AD

ITB can be indicated for severe spasticity refractory to oral medications in patients with SCI .[21] If a patient has been on stable long-term dosing, it is prudent to work through a complete differential to identify if another clinical problem is more likely to cause the AD though pump malfunction can still be on the differential.

Clinically, patients may report that their spasticity is suddenly worse during episodes of AD.[22] While pump complications are possible and may present as autonomic dysreflexia and/or worsened spasticity, it is recommended to thoroughly evaluate for underlying causes such as noxious stimuli below the level of SCI that could be contributing or confounding the increase in spasms. There is at least one helpful algorithm already created specifically regarding ITB pump malfunctioning.[23] A potential algorithm for clinicians working up unresolved AD in patients who have an ITB, inspired by Saulino et. al., is shown in Figure 1.[23]

As our case addresses, worsened spasms can coincide with AD episodes. While ITB pump malfunction should be on the differential, ITB pump malfunction incidence

Figure 1. Proposed optional framework for differential diagnosis in AD/uncontrolled spasticity:



A proposed algorithm for someone with AD episodes and spasticity with trigger not yet found. Blue boxes represent AD workflow while green boxes represent potentially concurrent ITB troubleshooting if clinically warranted. PO: oral; AP: anterior/posterior; C/A/P: CT Chest/abdomen/pelvis depending on SCI level. CAP: catheter access port. Site resources and medical contraindications shape imaging.

in available studies ranges about 7–8% of cases. Pump malfunctions can include mechanical failure and battery issues. If malfunctions occur, catheter-related complications are more likely to be the culprit. Programming errors are possible, and this should be checked and reprogrammed during the interrogation phase.[23-25]

Clinical Impact of AD on Morbidity and Mortality in SCI

The three leading causes of death in the first year of a traumatic SCI are respiratory diseases, other heart diseases (often unexplained heart attacks that usually do not represent a true underlying cause of death), and infectious diseases including urosepsis or pressure injury infections.[26]

In those who survive beyond the first year after injury, respiratory diseases are the leading cause of death, followed by infectious diseases, cancer, hypertensive and ischemic heart disease, and other heart diseases, respectively. Together hypertensive/ischemic heart disease and other heart disease encompasses about 19% of deaths in people with SCI .[1] There are many contributory factors, including level of and age at injury, indicating why people with SCI have significantly lower life expectancies than non-injured people. After potential chronic vascular remodeling, it is difficult to not consider AD's role in cardiovascular causes of death in this population.[27-28]

New Directions

An objective hallmark of AD is the rise in systolic blood pressure, which typically is recorded by an automatic blood pressure machine. There is some promising evidence demonstrated in rodents that artificial intelligence (AI) may play a positive role in detecting subtle physiologic triggers of AD. People with SCI could one day use wearable technology with non-invasive sensors to monitor changes in their sympathetic activity leading to improved time to diagnosis and treatment of AD.[29]

Clinical Vignette Outcome

Given the patient's presenting symptoms of sudden worsened spasticity and frequent uncontrolled AD, there was concern of ITB pump involvement. Prior pump troubleshooting/refills demonstrated no errors in logs and matching residuals. There was concern for pump malfunction or catheter malposition due to sluggish return at the catheter access port. N.U. was referred for a catheter dye study and imaging that demonstrated adequate flow through the catheter without evidence of disruption. The patient required hospitalization and consultation from the UPMC SCI consult team to provide further work-up for these symptoms.

The patient required several emergency department visits and inpatient acute care admissions for recurrent AD. He was treated for urinary tract infections and had close communication and follow up with his outpatient SCI physician. Ultimately, while experiencing AD with documented unstable vital signs, it was observed that his previously considered well-healing pressure injury had increasing drainage. While the area/size externally was thought to be improving according to the caregiver, computer tomography (CT) abdomen/pelvis showed a gluteal abscess adjacent to bone that was communicating with the wound.

After conservative measures including abscess drainage in the acute care bedside were unsuccessful, the patient underwent a left ischial wound debridement, bone debridement, and myocutaneous flap closure with plastic surgery. He was recommended for a 30-day bedrest period followed by a progressive seating protocol during his inpatient rehabilitation admission to optimize healing and maintain the integrity of the flap. During the inpatient rehabilitation stay, his ITB pump was monitored to ensure dosing was commensurate to his usual level of spasticity. N.U. was discharged home with his family and continued to follow-up in the outpatient SCI clinic with rarer occurrences of AD than previous to his flap procedure.

Takeaways

- Autonomic dysreflexia is life-threatening and a careful differential diagnosis can determine a patient's underlying cause
- It is important to remember that anything below the level of injury can ultimately be the cause of AD. This may require a wide differential and broad diagnostic workup
- Pressure injuries may appear to be improving on the surface because they're getting smaller yet still carry risk for deep infection potentially tracking to bone
- A patient having worsened spasticity in the setting of previously established and unchanged ITB dosing warrants further workup beyond intrathecal pump interrogation
- Interdisciplinary collaboration is crucial for diagnosis and treatment of AD in people with SCI

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