Autonomic Dysreflexia and Foot and Ankle Surgery

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Autonomic dysreflexia is a syndrome of massive imbalance of reflex sympathetic discharge occurring in patients with spinal cord injury with a lesion above the splanchnic outflow (Thoracic 6). Autonomic dysreflexia is characterized by a sudden and severe rise in blood pressure and is potentially life threatening. Because the onset of this entity is rapid and the potential morbidity is severe, it is important for those caring for spinal cord injury patients to be aware of this syndrome. The paper presents a review of the literature, and familiarizes one with the diagnosis, pathophysiology, and treatment. Two illustrative case reports are also presented. (The Journal of Foot & Ankle Surgery 40(3):172–177, 2001)

Key words: autonomic dysreflexia, spinal cord injury, sympathetic hyperactivity

The incidence of spinal cord injury (SCI) in the United States is estimated at 10,000 new cases per year (1). Spinal cord injuries are classified according to the level of neurologic impairment. The level of impairment is determined by a comprehensive sensory and motor exam as endorsed by the *International Standards for Neurological and Functional Classification of Spinal Cord Injury Patients* (2). The sensory level refers to the most caudal segment dermatome of the spinal cord with normal sensation to light touch and pin prick. Motor level is defined as the most caudal segment of the cord with normal motor function. The ASIA (American Spinal Injury Association) impairment scale is a 5-point scale that helps define the degree of impairment (2). A complete injury or ASIA A is defined as no sensory or motor function in the lowest sacral segments. Preservation of either motor or sensory function below the level of injury that includes the lowest sacral segments defines an incomplete injury. Incomplete injuries are further divided as seen in Table 1.

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Mechanism

Under normal circumstances in non-SCI individuals, any noxious stimulus below the splanchnic outflow will increase the blood pressure and heart rate secondary to central mechanisms that control sympathetic nervous activity. In individuals with SCI above the Thoracic 6 (T6) level, descending central sympathetics are interrupted and cannot provide controlling effects, by blocking the sympathetic surge. Therefore, stimulation below the lesion results in an increased activity of sensory endings leading to increased sympathetic activation due to stimulation of spinal reflexes. This then results in regional vasoconstriction, increasing peripheral vascular resistance and cardiac output, thus causing a marked rise in arterial pressure, due to a release of norepinephrine (NE) with alpha adrenergic activation of vascular smooth muscle (10).

Autonomic dysreflexia (AD) is a syndrome of massive imbalance of reflex sympathetic discharge occurring in patients with spinal cord injury with a lesion above the splanchnic outflow (T6). Autonomic dysreflexia is characterized by a sudden and severe rise in blood pressure and is potentially life threatening. It is one of the most common and severe medical complications that can affect spinal cord injured individuals. It results from various noxious stimuli below the level of the splanchnic outflow (T6), which in turn triggers sympathetic hyperactivity. The most common terms for this syndrome are autonomic hyperreflexia and autonomic dysreflexia. Other commonly used terms for this include paroxysmal hypertension (3), autonomic spasticity (4),

TABLE 1 ASIA impairment scale

A	**Complete:** No motor or sensory function is preserved in the sacral segments S4–S5.
B	**Incomplete:** Sensory but not motor function is preserved below the neurological level and includes the sacral segments S4–S5.
C	**Incomplete:** Motor function is preserved below the neurological level, and more than half of key muscles below the neurological level have muscle a grade less than 3.
D	**Incomplete:** Motor function is preserved below the neurological level, and at least half of key muscles below the neurological level have a muscle grade of 3 or more.
E	**Normal:** Motor and sensory function is normal.

From *International Standard for Neurological and Functional Classification of Spinal Cord Injury, Revised 1996.* Reprinted with permission by the American Spinal Injury Association (ASIA).

paroxysmal neurogenic hypertension (5), and spastic hyperreflexia (6). The incidence of AD varies in the literature with between 48% and 85% of individuals with SCI above the T6 level developing this syndrome. The higher the level and the more severe the injury, the greater the risk for developing AD. It is rarely detected before 2 months and presents in most patients by 6 months after the original injury. Injuries with lesions as low as T8 have been reported to develop this as well (7, 8). Most (90%) patients who will have an episode of dysreflexia will develop the clinical signs by 1 year postinjury (9). This condition must be properly assessed and managed quickly and efficiently as soon as clinical signs or symptoms present to prevent a potentially life-threatening crisis. Potential complications include: cerebral hemorrhage, myocardial infarction, seizures, and death. When the cause is identified and treated, patients generally suffer no long-term sequelae.

Intact sensory nerves below the level of the spinal cord injury transmit pain and proprioception information from the body to the spinal cord. The messages ascend in the spinothalamic and posterior columns of the spinal cord. These ascending messages stimulate sympathetic neurons in the intermediolateral gray matter columns. Because of the spinal cord lesion, the patient is unaware of these painful sensations. The severe vasoconstriction causes an elevation of blood pressure, which can be dramatic and sudden. Baroreceptors in the carotids and aortic arch send the information via the hypoglossal and vagus cranial nerves to the vasomotor areas in the brainstem and hypothalamus. Under normal situations, two vasocontrol reflexes take place. The vasomotor center attempts to compensate for the increased blood pressure by increasing parasympathetic stimulation to the heart via the vagus nerve, causing bradycardia and reducing stroke volume. This alone cannot compensate for the severe vasoconstriction. The second reflex is from the vasomotor center

TABLE 2 Most common causes of autonomic dysreflexia

Bladder obstruction
Bowel impaction
Pressure sores
Ingrown toenail
Orgasm
Labor and delivery
Abdominal emergencies
Fractures
Body positioning

TABLE 3 Most common symptoms of autonomic dysreflexia

Hypertension
Headache
Sweating — above level of lesion
Piloerection
Flushing — above level of lesion
Eye symptoms — lid retraction, mydriasis, conjunctival injection
Nasal congestion
Anxiety
Muscle spasticity
Silent autonomic dysreflexia — no symptoms

to attempt to shut off excessive spinal cord sympathetic outflow by stimulating inhibitory tracts to the splanchnic outflow to the cord, causing passive peripheral vasodilatation. Because these pathways are blocked by the SCI, this vasodilatation cannot occur and the blood pressure continues to elevate.

The most common causes of AD are listed in Table 2. While bladder issues are the most common cause of AD, ingrown toenails and procedures on the lower extremities are an important stimulus of AD. The most common symptoms of AD are listed in Table 3. The symptom of most concern is the increased blood pressure, and this may be manifested to the patient by a severe pounding headache. It is important to recognize that AD may be occurring and treatment techniques at this point should be initiated as the blood pressure is being monitored.

Preoperative Evaluation

Autonomic dysreflexia has many potential causes that may originate from the lower extremity. It is important that the initiating cause be identified and treated in order to resolve an episode of AD. Some specific lower extremity insults that may result in an episode include: any painful or irritating stimuli below the level of injury, deep venous thrombosis, pressure ulcers, ingrown toenails, burns or sunburn, insect bites, or contact with hard or sharp objects (11). Constrictive clothing, shoes, or appliances such as wheelchairs or splints can precipitate an attack. Any fracture or traumatic incident along with

surgical or diagnostic procedures can lead to AD. Surgery is a particularly potent stimulus to the development of AD, and even patients with no previous history of this response are at risk during operative procedures (12).

Some measures that can be taken to prevent an episode of AD include, most importantly, a thorough history concerning AD in predisposed patients. Predisposed patients should be free of urinary drainage and clear of bowel impaction. Premedication with anxiolytics and/or analgesics for patients experiencing pain can help prevent AD. Patients not receiving general anesthesia may take 10 mg of nifedipine orally, 30 minutes before the procedure (13).

Perioperative Management

The most critical step in preventing AD during surgery appears to be the selection of the appropriate form of anesthesia. Sufficient anesthetic depth provided by general, regional, or adequate local anesthesia can prevent AD, whereas topical anesthesia, sedation, or lack of anesthesia has led to hypertension in these patients (12). Spinal (intrathecal) anesthesia is recommended for procedures below the umbilicus, especially for incomplete lesions (13). For patients with complete lesions, intravenous sedation and local infiltrations are safe, provided causes of AD are ruled out.

In a study of AD by Eltorai et al. (13), 88.8% of SCI patients not receiving anesthesia developed a rise in systolic blood pressure during preparation for surgery. Further results revealed that AD occurred most commonly during the induction phase, irrespective of the anesthesia technique. The study also concluded that age, sex, and duration of SCI have no distinctive role in the occurrence of AD in surgery. The level of the injury was the most important factor. AD is most commonly seen with cervical injuries followed by upper thoracic and lower thoracic injuries. Patients who had no anesthesia at all had a higher incidence of developing AD. Therefore, it is imperative that an appropriate form of anesthesia be administered despite the fact that many of these patients have sensory deficits. A good rule to follow is if the procedure will cause pain to a patient without a SCI, it will likely cause autonomic dysreflexia in a person with a SCI above T6, and therefore, require anesthesia.

It is essential to position the patient with extreme care. The prone position should be avoided as much as possible. An elevated head is recommended if this patient is a tetraplegic to help decrease an elevation of blood pressure. Room temperature must be pleasant to the patient. It is recommended that the table padding should be at least 4 inches thick. Tight straps, especially around the patient's abdomen, and pressure caused by leaning on the patient, should be avoided.

Treatment Protocol

The treatment of AD involves first removing the stimulus, treating the afferent limb of the response (i.e., provide adequate analgesia), and treating the efferent limb of the reflex with medications. If prior intervention is unsuccessful, nifedipine and nitrates (orally and transdermally) are the most commonly used agents for lowering blood pressure (11). They should be used cautiously in patients who have coronary artery disease. They should not be administered if a patient has been on sildenafil citrate for the past 24 hours (14). Drugs that lower blood pressure by a central action such as clonidine are at times used to lower blood pressure although not as a first-line agent. Treatment for AD has been outlined in recent guidelines developed by the Consortium for Spinal Cord Medicine (11). Key components are listed in Table 4. The most important aspect is to recognize the possibility that AD may occur during the procedures that one may perform. Because of this, a baseline blood pressure should always be taken. It is important to remember that most young SCI patients with cervical lesions have a low resting blood pressure. The importance of this is that if a patient has a blood pressure of 130/80, this may in fact be extremely high and the individual may be having an episode of dysreflexia, if their initial borderline blood pressure is 90/60. Key management points are listed in Table 5.

If the patient complains of a headache or develops any of the other symptoms listed, the blood pressure should be monitored. If the patient is dysreflexic, one should stop the inciting event or the procedure that acted as the stimulus. All tight clothing or wraps should be loosened, as tight clothing alone may be the etiology. The patient should

TABLE 4 Treatment of autonomic dysreflexia

Recognize the symptoms
Remove precipitating stimuli
Elevate head of bed
Pharmacological management: Acute episodes
NTG I/150 g sublingual/or topical
Nifedipine 10 mg (oral not sublingual)
Clonidine 0.3–0.4 mg – alpha agonist

NTG, nitroglycerin.

TABLE 5 Treatment recommendations

SCI patients should be asked about their history of AD.
Document the individual signs and symptoms of AD.
Patients with a SCI level above T6 should be considered at risk.
Have AD treatment protocols available at all times.
Medications should be available as needed.
Discuss specifics with a SCI specialist.

SCI, spinal cord injury; AD, autonomic dysreflexia; T6, sixth thoracic vertebra.

be brought to the upright position as soon as possible to allow the blood pressure to fall from a gravity effect. The blood pressure should be monitored every 5 minutes or more frequently if needed. Usually these interventions will be enough to resolve the AD. If not, then the other common areas should be dealt with. If the patient has a catheter in place, then the catheter should be irrigated and inspected to make sure that there are no kinks in the tubing. If the patient manages his or her bladder via intermittent catheterization, he or she should be catheterized, after applying lidocaine jelly. If the symptoms persist for more than 15 minutes, with the blood pressure recorded as >150/90, then pharmacological intervention may be needed.

The authors hereby present two case reports of AD that were related to the management of foot and ankle pathology.

Case Report 1

A 22-year-old male presented with a painful swollen right great toe after an injury sustained during a wheelchair transfer. He was an incomplete C5 Asia B tetraplegic secondary to a biking injury. The past medical history was significant for asthma, and bowel and bladder incontinence. He had allergies to sulfa medications.

The toe became edematous and he began having increased spasms to his lower extremities along with flushed skin to his face and neck. He also complained of a headache. The patient's blood pressure remained normal. X-rays revealed an old intra-articular fracture to the medial head of the proximal phalanx of the right great toe (Fig. 1). This occurred prior to his spinal cord injury while playing soccer. When the patient re-injured his toe, the fragment became more pronounced against his skin (Fig. 2). Clinically, there was pain along the medial aspect of the interphalangeal joint of the right great toe. Dorsiflexion and plantarflexion was decreased and guarded at the interphalangeal joint (IPJ) level. The patient had palpable pedal pulses, and good sharp/dull discrimination to his lower extremities. There were significant spasms noted to his lower extremities.

The patient was diagnosed with autonomic dysreflexia after his toe injury. All other causes of dysreflexia were ruled out, including bladder and bowel obstruction. The causative factor was concluded to be his injury to the right great toe.

The patient was placed on anti-inflammatory medication, and his spinal cord specialist prescribed a benzodiazepine, lioresal, and ultimately tizanidine for his spasms. His medication was increased until his spasms became more tolerable.

Over the next several weeks, the patient's spasms decreased, but there was residual discomfort to the great toe. He was still having pain to the toe. Surgery was discussed and agreed upon. Preoperative testing, including CBC, PT, PTT, and ECG, was unremarkable. The patient underwent excision of the intra-articular fracture to the right great toe. This was performed under local with IV sedation. The patient had immediate relief of his headache and spasms after the surgery. He healed uneventfully with no recurrence of dysreflexia.

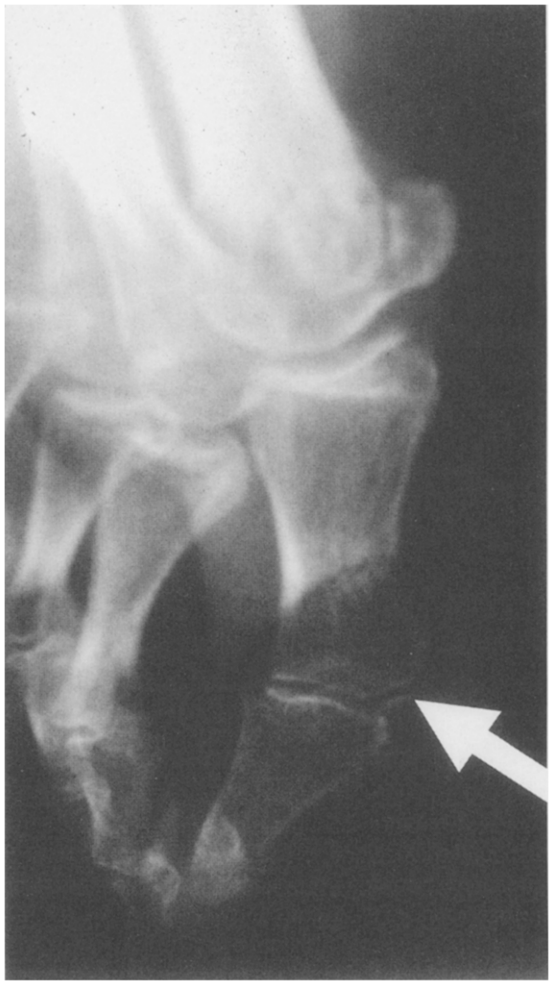

FIGURE 1 Intra-articular fracture of medial head of proximal phalanx right hallux.

Case Report 2

A 32-year-old C7 tetraplegic ASIA A male presented for treatment of an ulcer on the lateral aspect of his left leg. The ulcer originated during a transfer from a wheelchair 2 years ago. Local wound care had consisted of dry sterile dressing changes with antibiotic cream daily. The wound was in constant flux, varying in size and shape but never closed. The wound base was granular with interspersed fibrotic tissue. There was no sign of muscle belly,

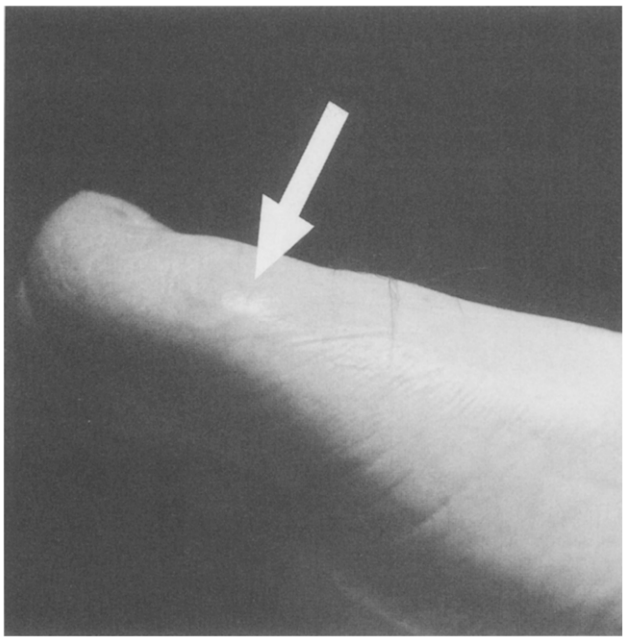

FIGURE 2 Preoperative appearance of intra-articular fragment of bone pressing against skin.

tendon sheath, or exposed bone. There was no sign of sinus tracts or abscess formation. Drainage was serosanguinous. X-rays were negative for bone involvement. Due to the longevity of the ulcer, it was decided to attempt surgical closure. Local wound care measures consisting of sharp and enzymatic debridement prepared the wound bed (Fig. 3). It was decided that primary closure with the use of an external skin-stretching device would be attempted.

The patient's medical status was stable. His past medical history was significant for a neurogenic bowel and bladder, recurrent urinary tract infections, and spasms controlled by medication. Sensory and motor exam was normal through C7 with loss below. The patient had muscle power in the flexors of the fingers of 1/5. Preoperative studies including CBC, basic metabolic profile, ECG, and chest x-rays were within normal limits.

The patient was taken to the operating room where, under local anesthesia and intravenous sedation, the ulcer was excised with two semielliptical incisions. The wound was then slightly undermined. Utilizing 2.0 nylon suture, the proximal and distal wound edges were approximated when an external skin-stretching device was applied to the central aspect of the wound. An immediate increase in blood pressure from 105/70 to 180/90 was observed. The device was removed resulting in immediate normalization of the blood pressure. The wound was completely closed primarily with retention bolsters in the central portion of the wound without the use of the external skin-stretching device.

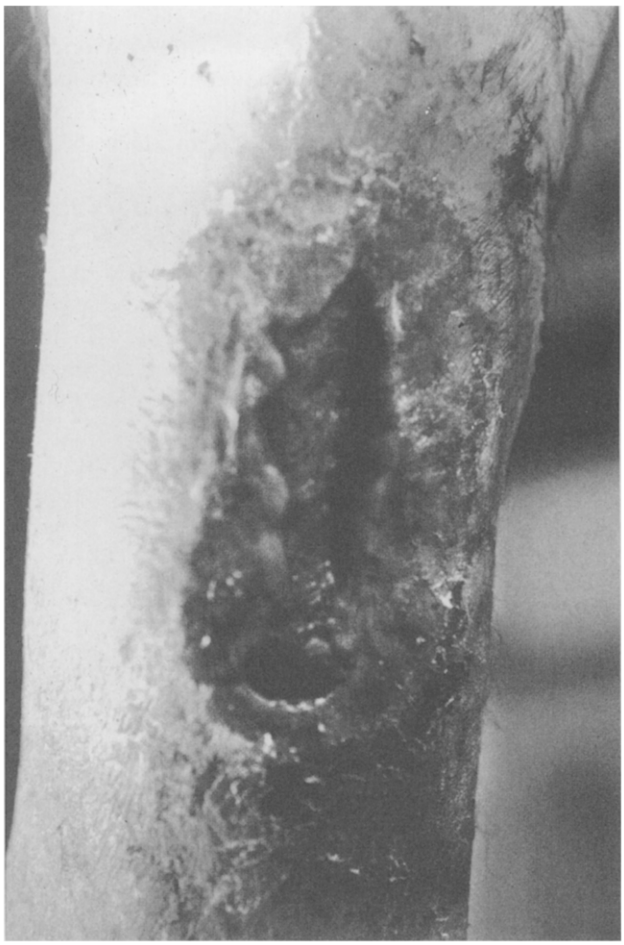

FIGURE 3 Preoperative appearance of lateral leg ulcer.

Discussion

The noxious stimuli required to precipitate an autonomic dysreflexic response can be difficult to isolate. These case presentations demonstrate the importance of being knowledgeable about this syndrome. Case 1 demonstrates how something as innocuous as a chip fracture can cause this response. The isolation of this condition as the primary cause of the patient's dysreflexia was through a systematic review of each organ system. It was not until all other processes were eliminated that the chip fracture was determined to be the etiology. The importance here is not to underestimate local and isolated foot and ankle conditions as the precipitating factors. Case 2 demonstrates the importance of the entire operating room team working in unison. The blood pressure increased during the procedure when the noxious stimulus from the skin-stretching device was applied. This occurred even with the presence of local and intravenous sedation. The increase in blood pressure in another situation may not have received the same intense scrutiny if the team was not aware of the possibility of dysreflexia occurring under these circumstances.

Autonomic dysreflexia can be life threatening in spinal cord injury patients above the T6 level. If left untreated, an intracranial bleed can be precipitated. Treatment must identify the source of the stimulus. In a normal sensate individual, this is easily identifiable but in the insensate individual, it is a systematic process of elimination. The insensate status of the spinal cord patient does not preclude the use of local anesthesia as in other insensate states (i.e., diabetic neuropathy and peripheral nerve injuries). The simple removal of an ingrown nail or granuloma without anesthesia could result in a dysreflexic response. It is thereby incumbent upon the foot and ankle specialist dealing with spinal cord injury patients to be aware of this syndrome, the ramifications of not taking the proper precautions, and the proper perioperative management.

Conclusion

Many factors are related to the prevention of autonomic dysreflexia in SCI patients. These include proper preparation of subjects, avoiding noxious stimuli, proper positioning, prophylactic measures, monitoring, and appropriate anesthesia selection. A team approach is paramount in dealing with SCI patients. The surgical team, anesthesiologists, and physiatrists must know how to recognize the signs and symptoms of AD, how to prevent its occurrence, and how to manage it aggressively. Practitioners treating spinal cord-injured patients need to have a thorough understanding of this condition and be prepared to manage all of its parameters.

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