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Commentary

Preventing Autonomic Dysreflexia with the Appropriate Type and Level of Care

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DESCRIPTION

Autonomic dysreflexia is a potentially lethal syndrome that arises in patients with Spinal Cord Injury (SCI), and involves a sudden, reflexive increase in blood pressure [1]. This blood pressure enhancement occurs when there is an uncoordinated sympathetic response to a noxious stimulus below the level of injury, most often from the bladder [2]. Specifically, urinary tract infections or procedures on the urinary tract, obstructed foley catheters, distended bladder, rectal impaction, skin pressure ulcers, and childbirth, can all lead to autonomic dysreflexia [3-5]. Because the SCI has rendered the body incapable of proper central inhibition in the face of stimulation, there is an outsized outflow that leads to hypertension [6]. When stretch receptors in the carotid sinus and aortic body perceive this heightened pressure, their activation leads to bradycardia and vasodilation through afferent nerve signaling that stimulates the sinoatrial node.

Because of the life-threatening nature of autonomic dysreflexia, and the inability to cure SCI patients from being prone to the syndrome, proper care is critical to ensuring the well-being of the relevant patient population. Here, we review details of autonomic dysreflexia and its complications, as well as the best ways to manage at-risk patients.

Autonomic dysreflexia is common following certain spinal cord injuries

Between 5 and 7 out of 10 SCI patients whose injury occurs above T6 develop autonomic dysreflexia [2]. Though much less common, autonomic dysreflexia can also occur in lower-level SCIs and in non-traumatic SCIs. In addition to the level, the completeness of SCI is relevant for predicting the severity and frequency of episodes of autonomic dysreflexia. Those with complete SCI have a 91% likelihood of experiencing autonomic dysreflexia, compared to only 27% of those with incomplete SCI.

Most SCI patients who develop autonomic dysreflexia will develop it within a year post-injury [7]. However, the syndrome normally does not develop until reflexes have recovered, with the earliest reported case being 4 days following the injury.

While patients with autonomic dysreflexia can be asymptomatic, severe headache, excessive sweating, bradycardia, and facial flushing tend to accompany the syndrome [8,9]. Respiratory insufficiency is often an overlooked sign of autonomic dysreflexia [10]. Other symptoms that occur with autonomic dysreflexia include nausea, vomiting, anxiety, visual disturbances, and nasal congestion [2].

Complications from autonomic dysreflexia can be life-threatening

An autonomic dysreflexia episode involves a systolic blood pressure increase of at least 25 mmHg above baseline [2]. When a systolic pressure increase reaches 40 mmHg above baseline or at least 150 mmHg, it is considered a significant episode. There are several complications of autonomic dysreflexia, including seizures, left ventricular dysfunction, pulmonary edema, intracranial hemorrhage, reversible encephalopathy syndrome, and retinal detachment [11,12].

When cerebral vasodilation, which often occurs with autonomic dysreflexia, is combined with dangerously high blood pressure, patients are at significant risk for hemorrhagic stroke. Compared to those without autonomic dysreflexia, those with the syndrome have a 300% to 400% higher chance of suffering a stroke [2].

The heart is also at risk during autonomic dysreflexia, and the higher the level of injury, the more severe the cardiovascular dysfunction. While bradycardia that occurs with autonomic

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dysreflexia may be minor, in those with coronary artery disease, autonomic dysreflexia episodes can cause heart attacks.

Treatments for autonomic dysreflexia do not address the underlying pathophysiology

Pharmacological treatments aimed at alleviating autonomic dysreflexia include anti-hypertensive nitrates, adrenergic blockers, and nifedipine, with 10 milligrams (mg) of nifedipine often used as the initial treatment [2,13]. In emergency cases of severe hypertension, nitroglycerine 2% paste is recommended. Botox injections to the bladder are often employed to address autonomic dysreflexia.

Unfortunately, there are several downsides to the currently available treatments for autonomic dysreflexia. In addition to the side effects, which include nausea, vomiting, tachycardia, cardiac arrhythmia, rebound hypotension, flushing, and drowsiness, in many cases, treatment does not reverse the syndrome [2,13]. Further, these pharmacological interventions do not address the underlying cause of sympathetic dysregulation and instead provide only short-term effects in certain organs [14].

While suppressing sympathetic hyperactivity is the aim of several treatment interventions, it is generally agreed that prevention is the best way to address autonomic dysreflexia [15-17].Given that autonomic dysreflexia is commonly caused by urinary bladder distension and recto-sigmoid fecal impaction, care programs that ensure frequent voiding and bowel care are critical [13,17-19]. Proper skin care practices are also essential for the well-being of patients prone to autonomic dysreflexia [20]. Removing the stimulus that is the cause of autonomic dysreflexia as soon as autonomic dysreflexia occurs is necessary before the condition turns into a medical emergency.

Type and level of care are critical for the well-being of those with autonomic dysreflexia

For those prone to autonomic dysreflexia, the syndrome is a constant threat that can occur daily [21]. Further, proper and immediate response is critical in the face of autonomic dysreflexia [22]. It is therefore vital that patients have the right type and level of care for identifying autonomic dysreflexia and administering potentially life-saving interventions. Certain patient populations, including pregnant women, are particularly vulnerable and need specialized attention and monitoring [23].

Medical staff who are not familiar with autonomic dysreflexia often fail to identify it [1]. Further, knowledge of autonomic dysreflexia, including its causes and consequences, is much lower among healthcare professionals with lower levels of training [24,25]. Thus, care from a home health aide is not adequate for optimizing health outcomes for those prone to autonomic dysreflexia.

Successful management of SCI patients at heightened risk for autonomic dysreflexia requires knowledge of guidelines that help in the avoidance of dangerous complications. This management often involves a multidisciplinary approach that includes specialists across spinal rehabilitation, urology, gastroenterology, and other relevant clinical areas [2,12].

Nurses are often the first healthcare providers to identify the syndrome and are key for monitoring patients with autonomic dysreflexia [2]. As such, nursing-driven protocols for treating the syndrome have been shown to be highly successful and associated with low incidences of adverse events [26]. RN, LVN, or LPN level of care is therefore required to protect the health of those who experience autonomic dysreflexia and to prevent untimely death.

CONCLUSION

Patients prone to autonomic dysreflexia are at a constant risk for life-threatening complications. Their well-being relies on their access to consistent and reliable care from those who are knowledgeable about the syndrome and know how to identify and rapidly intervene when the syndrome occurs. Unfortunately, many healthcare professionals do not have the relevant training to ensure the safety of this patient population. Careful consideration must therefore be taken to develop treatment plans for each patient at risk for autonomic dysreflexia.

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