

Neurogenic Shock

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Abstract: Neurogenic shock is a type of distributive shock that describes the sudden loss of autonomic tone due to spinal cord injury often characterized by hypotension and relative bradycardia. Loss of sympathetic tone occurs with injuries above T6 and results in decreased systemic vascular resistance. Peripheral vasoconstrictors, chronotropes, and inotropes may be needed in cases of neurogenic shock. Autonomic instability may develop and often persists several weeks after the injury. Aggressive management is imperative in the initial phases of neurogenic shock to avoid further secondary ischemic injury to the cord.

Keywords: Spinal cord injury, shock, autonomic dysreflexia.

INTRODUCTION

Neurogenic shock describes the sudden loss of autonomic tone due to spinal cord injury (SCI). Disruption of the descending sympathetic pathways results in unopposed vagal tone in the vascular smooth muscle, causing decreased systemic vascular resistance and vasodilation. The hypotension that results from neurogenic shock places patients at increased risk of secondary spinal cord ischemia due to impairment of autoregulation [1-3]. Though the terms are sometimes used interchangeably, neurogenic shock describes the hemodynamic changes following SCI, whereas spinal shock is characterized by a reversible reduction of sensory, motor, or reflex function of the spinal cord below the level of injury [4].

Our search strategy involved a PubMed search from 1986 to 2012 of articles using the search term "neurogenic shock" in the English language. Many articles were excluded due to irrelevance to the topic and scope of this discussion. Also references from these articles were scanned and relevant articles included. Articles involving both adults and children were utilized, and select animal studies were included.

CLINICAL MANIFESTATIONS

Neurogenic shock is a type of distributive shock, but should be a diagnosis of exclusion in the early phases of traumatic resuscitation after hemorrhagic shock is ruled out. There is no definitive diagnostic test, but classically patients exhibit hypotension and relative bradycardia. The bradycardia is often exacerbated by suctioning, defecation, turning, and hypoxia [5]. The skin is often warm and flushed initially. Hypothermia may develop because of profound vasodilation and heat loss. Often the central venous pressure is low due to decreased systemic vascular resistance. An

animal model of complete cervical SCI demonstrated bradycardia and hypotension, increased cardiac output due to increased stroke volume, and an increase in serum vasopressin [6]. There is some evidence to suggest that hypertension occurs within the first few minutes of SCI, often in the field or emergency department, and hypotension may follow later [7-9]. Thus, patients with SCI must be monitored closely for the development of neurogenic shock even if it is not present on presentation. The joint committee of the American Spinal Injury Association and the International Spinal Cord Society proposed a set of definitions of general autonomic nervous system dysfunction (neurogenic shock, orthostatic hypotension, autonomic dysreflexia, temperature dysregulation, sweating disturbances) following adult SCI that should be assessed by clinicians [10].

EPIDEMIOLOGY

Pediatric SCI occurs in 1.99 per 100,000 children in the United States, and new cases account for approximately 1,500 annual hospital admissions [11]. The most common (41-56%) cause of traumatic pediatric SCI is motor vehicle crashes, and 67% of these patients are not properly restrained [11-13]. Other causes of SCI include spinal anesthesia, Guillain-Barre syndrome, other neuropathies, and autonomic nervous system toxins. Causes of SCI unique to the pediatric population include birth-related injuries, lap-belt injuries, transverse myelitis, and child abuse. Also, cervical subluxation may result in cervical SCI in children with Trisomy 21, juvenile idiopathic arthritis, skeletal dysplasias, and tonsillopharyngitis [14-16]. Cervical SCI is more common in children than in adults, presumably due to the anatomical features distinguishing these populations including larger head size and underdeveloped neck muscles in children. Cervical SCI has an overall mortality of 18-27% in children [12, 13].

Spinal cord injury without radiographic abnormality (SCIWORA) and spinal cord injury without evidence of radiographic trauma (SCIWORET) are terms introduced

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before imaging modalities were sophisticated enough to reveal evidence of trauma that we now are able to see using magnetic resonance imaging (MRI). SCIWORET is a term more commonly used in adult trauma because of pre-existing conditions such as spinal stenosis and disc herniation. A 2001 article reported that SCIWORA occurs in 38% of children with cervical SCI, and is common in sporting injuries and in victims of child abuse [12]. This incidence is likely decreasing as MRI technology advances.

The loss of sympathetic tone, and thus neurogenic shock, is most common when the level of the injury is above T6 [8]. Moreover, neurogenic shock may occur anytime after the onset of injury or illness, ranging from the time of presentation to several weeks after presentation. No human studies document the hemodynamic changes occurring after acute SCI in children, and the incidence of neurogenic shock in children with SCI is unknown. However, reports indicate anywhere from 50-90% of adults with cervical SCI require fluid resuscitation and vasoactive infusions to achieve the adult parameters recommended (MAP >85-90 mm Hg for 7 days) by the Congress of Neurological Surgeons' guidelines for management of SCI [5, 8, 17-20]. Adults with higher SCI (C1-C5) may be more likely to require cardiovascular interventions, such as vasoactive agents or cardiac pacing, than lower (C6-C7) SCI [21].

MANAGEMENT

Decreased systemic vascular resistance results in a relative hypovolemia due to increased venous capacity, and isotonic fluid administration is often necessary. However, hypotension due to neurogenic shock is often refractory to fluid resuscitation. Nevertheless, hypotension in a trauma patient cannot be assumed to be due to neurogenic shock initially, and could be a sign of hemorrhagic shock. Thus, trauma victims with hypotension should be treated initially with crystalloid (0.9% sodium chloride, ringer's lactate) or colloid (albumin, blood products) fluids and evaluated for any ongoing blood loss. Patients should be adequately resuscitated from a hemodynamic perspective before undergoing operative spinal cord decompression.

Hypotension must be treated immediately in order to avoid secondary ischemic SCI. Cervical SCI is often seen in patients who also have traumatic brain injury and hypotension cannot be tolerated in the setting of traumatic brain injury either. Mannitol should be avoided if shock is present in patients with suspected traumatic brain and spinal cord injury, as hypertonic saline is now recommended as first-line osmotherapy agent in pediatric severe traumatic brain injury [22].

If hypotensive patients have normal chronotropy and inotropy, then an α_1 agonist acting as a peripheral vasoconstrictor such as phenylephrine is indicated. Norepinephrine may also be considered, as it has α_1 and β_1 agonistic activity [23]. Epinephrine and vasopressin infusions may be used in refractory cases of hypotension [23]. The evidence for the elevated MAP goal (MAP >85-90 mm Hg for 7 days) in adults published by the Congress of Neurological Surgeons is weak [24]. Of course, the definition of hypotension in children (systolic blood pressure

<70 mm Hg plus twice the age in years) is appropriate to consider when preventing and treating shock in general in children, but in the case of SCI the blood pressure should probably be maintained higher than the minimum acceptable blood pressure; the blood pressure goals in pediatric patients with SCI are unknown [20].

If bradycardia is present, patients may respond to atropine, glycopyrrolate, or vasoactive infusions with chronotropic, vasoconstrictor, and inotropic properties such as dopamine or norepinephrine. Also, isoproterenol may be considered if a strictly chronotropic agent is needed.

Phenylephrine can potentially cause reflex bradycardia, as there is no β agonist activity, and should be used with caution in patients with bradycardia as part of their neurogenic shock presentation. In rare cases, cardiac pacing has been successful however since the cause of the bradycardia is neurochemical rather than electrophysiological, it may be more prudent to use pharmacological treatments. If patients demonstrate particular sensitivity to suctioning or positioning, one may consider giving atropine or glycopyrrolate prior to manipulation. Methylxanthines (theophylline, aminophylline) and propantheline have also been used for refractory bradycardia [25-29]. Sinus bradycardia is most common in patients with severe cervical SCI, but patients may develop other dysrhythmias, including AV block, atrial fibrillation, or even cardiac arrest [5].

PROGNOSIS

Patients with cervical SCI are more likely to develop neurogenic shock [5, 8, 19, 30]. In fact, patients with thoracolumbar SCI do not commonly develop neurogenic shock [5]. Likewise, complete injuries and higher grade injuries according to the American Spinal Injury Association grade often lead to more severe neurogenic shock [5, 19, 24, 30-32]. Presence of neurogenic shock has been shown to lead to delays in operative management, which may potentially worsen outcome as well [30]. Though it is recommended to avoid and aggressively treat hypotension, it is unknown whether hypotension worsens outcome [33].

Neurogenic shock can persist for 1-6 weeks after the injury [3, 5, 24]. Autonomic dysreflexia, low resting blood pressure, and orthostatic hypotension are not uncommon during the chronic phase, often after neurogenic shock has resolved [1, 2, 34, 35]. Autonomic instability is often manifested by episodic hypertension, flushing, diaphoresis, and tachycardia.

In conclusion, spinal cord injury regardless of mechanism may result in neurogenic shock characterized by sudden loss of autonomic tone resulting in hypotension and relative bradycardia. Higher lesions are associated with more severe deficits. Peripheral vasoconstrictors, chronotropes, and inotropes may be needed in cases of neurogenic shock. The hypotension that results from loss of autonomic tone can precipitate further secondary ischemic injury to the spinal cord, and should be managed aggressively. Dysautonomia may develop and often persists several weeks after the injury. Any patient presenting with the possibility of SCI should have their spine immobilized as soon as is practical to prevent any further injury to or compression on the spinal cord.

CONFLICT OF INTEREST

The authors confirm that this article content has no conflicts of interest.

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